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In this issue

Psychotherapy of the Medically Ill
By Toksoz B. Karasu

Special Section:
The Psychobiology of Depression—
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Patients on long-term therapy, especially high doses, should be evaluated periodically for possible adjustment or discontinuance of drug therapy.

Adverse Reactions: Drowsiness, cholestatic jaundice, agranulocytosis, eosinophilia, leukopenia, hemolytic anemia, thrombocytopenic purpura and pancytopenia; postural hypotension, tachycardia, fainting, dizziness and, occasionally, a shock-like condition; reversal of epinephrine effects; EKG changes have been reported, but relationship to myocardial damage is not confirmed; neuromuscular (extrapyramidal) reactions; pseudoparkinsonism, motor restlessness, dystonias, persistent tardive dyskinesia, hyperreflexia in the newborn; psychotic symptoms, catatonic-like states, cerebral edema; convulsive seizures; abnormality of the cerebrospinal fluid proteins; urticarial reactions and photosensitivity, exfoliative dermatitis, contact dermatitis; lactation and breast engorgement (in females on large doses), false positive pregnancy tests, amenorrhea, gynecomastia; hyperglycemia, hypoglycemia, glycosuria; dry mouth, nasal congestion, constipation, adynamic ileus, urinary retention, miosis, mydriasis; after prolonged substantial doses, skin pigmentation, epithelial keratopathy, lenticular and corneal deposits and pigmentary retinopathy, visual impairment; mild fever (after large I.M. dosage); hyperpyrexia; increased appetite and weight; a systemic lupus erythematosus-like syndrome; peripheral edema.

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This section contains very brief articles (maximum of 1,000 words, 10 references, 1 table, and *no figures*) reporting new research findings, including preliminary data from pilot studies, and case reports that 1) describe new syndromes, 2) cast a new light on established ones, 3) indicate a new therapeutic procedure of potential value, or 4) describe adverse effects or previously unreported complications of drugs or therapeutic interventions. Because of the stringent criteria for this section, authors can expect more rapid publication than is possible in other sections. Submitted papers that substantially exceed the stated maximum length or contain figures will be returned to the author unreviewed.

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1. Berne E: Principles of Group Treatment. New York, Oxford University Press, 1966, p 26

2. Blackwell B, Marley E, Price J, et al: Hypertensive interactions between monoamine oxidase inhibitors and foodstuffs. Br J Psychiatry 113:349-365, 1967
3. Gold PW, Goodwin FK: Urinary free cortisol in depression and mania. Presented at the 130th annual meeting of the American Psychiatric Association, Toronto, Ont, Canada, May 2-6, 1977
4. Brosin H: Communication systems of the consultation process, in The Psychiatric Consultation. Edited by Mendel W, Solomon P. New York, Grune & Stratton, 1968

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Warnings. *Usage in Pregnancy*—Safe use of Navane during pregnancy has not been established. Therefore, this drug should be given to pregnant patients only when, in the judgment of the physician, the expected benefits from the treatment exceed the possible risks to mother and fetus. Animal reproduction studies and clinical experience to date have not demonstrated any teratogenic effects.

In the animal reproduction studies with Navane, there was some decrease in conception rate and litter size, and an increase in resorption rate in rats and rabbits, changes which have been similarly reported with other psychotropic agents. After repeated oral administration to rats (5 to 15 mg/kg/day), rabbits (3 to 50 mg/kg/day), and monkeys (1 to 3 mg/kg/day) before and during gestation, no teratogenic effects were seen. (See Precautions.)

Usage in Children—The use of Navane in children under 12 years of age is not recommended because safety and efficacy in the pediatric age group have not been established.

As is true with many CNS drugs, Navane may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery, especially during the first few days of therapy. Therefore, the patient should be cautioned accordingly.

As in the case of other CNS-acting drugs, patients receiving Navane should be cautioned about the possible additive effects (which may include hypotension) with CNS depressants and with alcohol.

Precautions. An antiemetic effect was observed in animal studies with Navane; since this effect may also occur in man, it is possible that Navane may mask signs of overdosage of toxic drugs and may obscure conditions such as intestinal obstruction and brain tumor.

In consideration of the known capability of Navane and certain other psychotropic drugs to precipitate convulsions, extreme caution should be used in patients with a history of convulsive disorders or those in a state of alcohol withdrawal since it may lower the convulsive threshold. Although Navane potentiates the actions of the barbiturates, the dosage of the anticonvulsant therapy should not be reduced when Navane is administered concurrently.

Caution as well as careful adjustment of the dosage is indicated when Navane is used in conjunction with other CNS depressants other than anticonvulsant drugs.

Though exhibiting rather weak anticholinergic properties, Navane should be used with caution in patients who are known or suspected to have glaucoma, or who might be exposed to extreme heat, or who are receiving atropine or related drugs.

Use with caution in patients with cardiovascular disease.

Also, careful observation should be made for pigmentary retinopathy, and lenticular pigmentation (fine lenticular pigmentation has been noted in a small number of patients treated with Navane for prolonged periods). Blood dyscrasias (agranulocytosis, pancytopenia, thrombocytopenic purpura), and liver damage (jaundice, biliary stasis) have been reported with related drugs.

Undue exposure to sunlight should be avoided. Photosensitive reactions have been reported in patients on Navane.

Intramuscular Administration—As with all intramuscular preparations, Navane Intramuscular should be injected well within the body of a relatively large muscle. The preferred sites are the upper outer quadrant of the buttock (i.e., gluteus maximus) and the mid-lateral thigh.

The deltoid area should be used only if well developed, such as in certain adults and older children, and then only with caution to avoid radial nerve injury. Intramuscular injections should not be made into the lower and mid-thirds of the upper arm. As with all intramuscular injections, aspiration is necessary to help avoid inadvertent injection into a blood vessel.

Adverse Reactions. *Note:* Not all of the following adverse reactions have been reported with Navane (thiothixene). However, since Navane has certain chemical and pharmacologic similarities to the phenothiazines, all of the known side effects and toxicity associated with phenothiazine therapy should be borne in mind when Navane is used.

Cardiovascular effects: Tachycardia, hypotension, lightheadedness, and syncope. In the event hypotension occurs, epinephrine should not be used as a pressor agent since a paradoxical further lowering of blood pressure may result. Nonspecific EKG changes have been observed in some patients receiving Navane. These changes are usually reversible and frequently disappear on continued Navane therapy. The incidence of these changes is lower than that observed with some phenothiazines. The clinical significance of these changes is not known.

CNS effects: Drowsiness, usually mild, may occur although it usually subsides with continuation of Navane therapy. The incidence of sedation appears similar to that of the piperazine group of phenothiazines, but less than that of certain aliphatic phenothiazines. Restlessness, agitation and insomnia have been noted with Navane (thiothixene). Seizures and paradoxical exacerbation of psychotic symptoms have occurred with Navane infrequently.

Hyperreflexia has been reported in infants delivered from mothers having received structurally related drugs.

In addition, phenothiazine derivatives have been associated with cerebral edema and cerebrospinal fluid abnormalities.

Extrapyramidal symptoms, such as pseudo-parkinsonism, akathisia, and dystonia have been reported. Management of these extrapyramidal symptoms depends upon the type and severity. Rapid relief of acute symptoms may require the use of an injectable antiparkinson agent. More slowly emerging symptoms may be managed by reducing the dosage of Navane and/or administering an oral antiparkinson agent.

Persistent Tardive Dyskinesia: As with all antipsychotic agents tardive dyskinesia may appear in some patients on long term therapy or may occur after drug therapy has been discontinued. The risk seems to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities.

There is no known effective treatment for tardive dyskinesia: antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear.

Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked.

It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time, the syndrome may not develop.

Hepatic effects: Elevations of serum transaminase and alkaline phosphatase, usually transient, have been infrequently observed in some patients. No clinically confirmed cases of jaundice attributable to Navane have been reported.

Hematologic effects: As is true with certain other psychotropic drugs, leukopenia and leukocytosis, which are usually transient, can occur occasionally with Navane. Other antipsychotic drugs have been associated with agranulocytosis, eosinophilia, hemolytic anemia, thrombocytopenia and pancytopenia.

Allergic reactions: Rash, pruritus, urticaria, photosensitivity and rare cases of anaphylaxis have been reported with Navane. Undue exposure to sunlight should be avoided. Although not experienced with Navane, exfoliative dermatitis and contact dermatitis (in nursing personnel) have been reported with certain phenothiazines.

Endocrine disorders: Lactation, moderate breast enlargement and amenorrhea have occurred in a small percentage of females receiving Navane. If persistent, this may necessitate a reduction in dosage or the discontinuation of therapy. Phenothiazines have been associated with false positive pregnancy tests, gynecomastia, hypoglycemia, hyperglycemia, and glycosuria.

Autonomic effects: Dry mouth, blurred vision, nasal congestion, constipation, increased sweating, increased salivation, and impotence have occurred infrequently with Navane therapy. Phenothiazines have been associated with miosis, mydriasis, and adynamic ileus.

Other adverse reactions: Hyperpyrexia, anorexia, nausea, vomiting, diarrhea, increase in appetite and weight, weakness or fatigue, polydipsia and peripheral edema.

Although not reported with Navane, evidence indicates there is a relationship between phenothiazine therapy and the occurrence of a systemic lupus erythematosus-like syndrome.

NOTE: Sudden deaths have occasionally been reported in patients who have received certain phenothiazine derivatives. In some cases the cause of death was apparently cardiac arrest or asphyxia due to failure of the cough reflex. In others, the cause could not be determined nor could it be established that death was due to phenothiazine administration.

Dosage and Administration. Dosage of Navane should be individually adjusted depending on the chronicity and severity of the condition. In general, small doses should be used initially and gradually increased to the optimal effective level, based on patient response.

Some patients have been successfully maintained on once-a-day Navane therapy.

Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

Navane Intramuscular Solution—For Intramuscular Use Only. Where more rapid control and treatment of acute behavior is desirable, the intramuscular form of Navane may be indicated. It is also of benefit where the very nature of the patient's symptomatology, whether acute or chronic, renders oral administration impractical or even impossible.

For treatment of acute symptomatology or in patients unable or unwilling to take oral medication, the usual dose is 4 mg of Navane Intramuscular administered 2 to 4 times daily. Dosage may be increased or decreased depending on response. Most patients are controlled on a total daily dosage of 16 to 20 mg. The maximum recommended dosage is 30 mg/day. An oral form should supplant the injectable form as soon as possible. It may be necessary to adjust the dosage when changing from the intramuscular to oral dosage forms. Dosage recommendations for Navane Capsules and Concentrate appear in the following paragraphs.

Navane Capsules; Navane Concentrate—In milder conditions, an initial dose of 2 mg three times daily. If indicated, a subsequent increase to 15 mg/day total daily dose is often effective.

In more severe conditions, an initial dose of 5 mg twice daily.

The usual optimal dose is 20 to 30 mg daily. If indicated, an increase to 60 mg/day total daily dose is often effective. Exceeding a total daily dose of 60 mg rarely increases the beneficial response.

Overdosage. Manifestations include muscular twitching, drowsiness, and dizziness. Symptoms of gross overdosage may include CNS depression, rigidity, weakness, torticollis, tremor, salivation, dysphagia, hypotension, disturbances of gait, or coma.

Treatment: Essentially symptomatic and supportive. For Navane oral, early gastric lavage is helpful. For Navane oral and Intramuscular, keep patient under careful observation and maintain an open airway, since involvement of the extrapyramidal system may produce dysphagia and respiratory difficulty in severe overdosage. If hypotension occurs, the standard measures for managing circulatory shock should be used (I.V. fluids and/or vasoconstrictors).

If a vasoconstrictor is needed, levarterenol and phenylephrine are the most suitable drugs. Other pressor agents, including epinephrine, are not recommended, since phenothiazine derivatives may reverse the usual pressor action of these agents and cause further lowering of blood pressure.

If CNS depression is present, recommended stimulants include amphetamine, dextroamphetamine, or caffeine and sodium benzoate. Picrotoxin or pentylenetetrazol should be avoided. Extrapyramidal symptoms may be treated with antiparkinson drugs.

There are no data on the use of peritoneal or hemodialysis, but they are known to be of little value in phenothiazine intoxication.

How Supplied. Navane (thiothixene) is available as capsules containing 1 mg, 2 mg, 5 mg, and 10 mg of thiothixene in bottles of 100 and 1,000. Navane is also available as capsules containing 20 mg of thiothixene in bottles of 100 and 500.

Navane (thiothixene hydrochloride) Concentrate is available in 120 ml (4 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, 5 mg, 6 mg, 8 mg, and 10 mg, and in 30 ml (1 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, and 5 mg. Each ml contains thiothixene hydrochloride equivalent to 5 mg of thiothixene. Contains alcohol, U.S.P. 7.0% v/v (small loss unavoidable).

Navane (thiothixene hydrochloride) Intramuscular solution is available in a 2 ml amber glass vial in packages of 10. Each ml contains thiothixene hydrochloride equivalent to 2 mg of thiothixene, dextrose 5% w/v, benzyl alcohol 0.9% w/v, and propyl gallate 0.02% w/v.

References. 1. Stotsky BA: Relative efficacy of parenteral haloperidol and thiothixene for the emergency treatment of acutely excited and agitated patients. *Dis Nerv Syst* 38:967-973, 1977. 2. Bressler B, Friedel RO: A comparison between chlorpromazine and thiothixene in a Veterans Administration hospital population. *Psychosomatics* 12:275-277, 1971. 3. Denber HCB, Turns D: Double blind comparison of thiothixene and trifluoperazine in acute schizophrenia. *Psychosomatics* 13:100-104, 1972. 4. Sterlin C, Ban TA, Lehman HE, Saxena BM: The place of thiothixene in the treatment of schizophrenic patients. *Can Psychiatr Assoc J* 15:3-4, 1970. 5. Brauzer B, Goldstein BJ: Comparative effects of intramuscular thiothixene and trifluoperazine in psychotic patients. *J Clin Pharmacol* 8:400-403, 1968. 6. Engelhardt DM, Rudorfer L: The chronic schizophrenic outpatient in an urban community: Social and vocational adjustment. Presented as a Scientific Exhibit at the 130th Annual Meeting, American Psychiatric Association, Toronto, Canada, May 2-6, 1977. 7. Itil TM, Unverdi C, Wohlrabe J, Larsen V, Levitt J: Drug therapy of psychosis associated with organic brain syndrome, presented as a scientific exhibit at the American Public Health Association Centennial, Atlantic City, New Jersey, November 12-16, 1972. 8. Dillenkoffer RL, et al: Electrocardiographic evaluations of schizophrenic patients. Presented as a Scientific Exhibit at the 125th Annual Meeting of the American Psychiatric Association, Dallas, Texas, May 1-4, 1972. 9. Goldstein B, Weiner D, Banas F: Clinical evaluation of thiothixene in chronic ambulatory schizophrenic patients, in Lehmann HE, Ban TA (eds): *Modern Problems in Pharmacopsychiatry*. Basel, S Karger, 1969, vol 2, pp 45-52.

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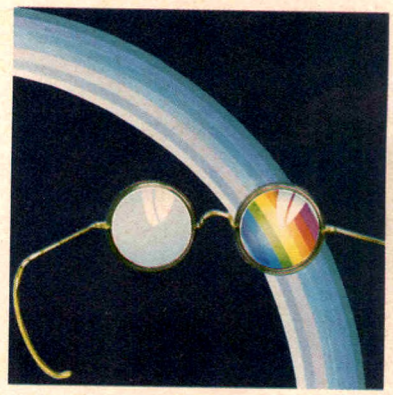
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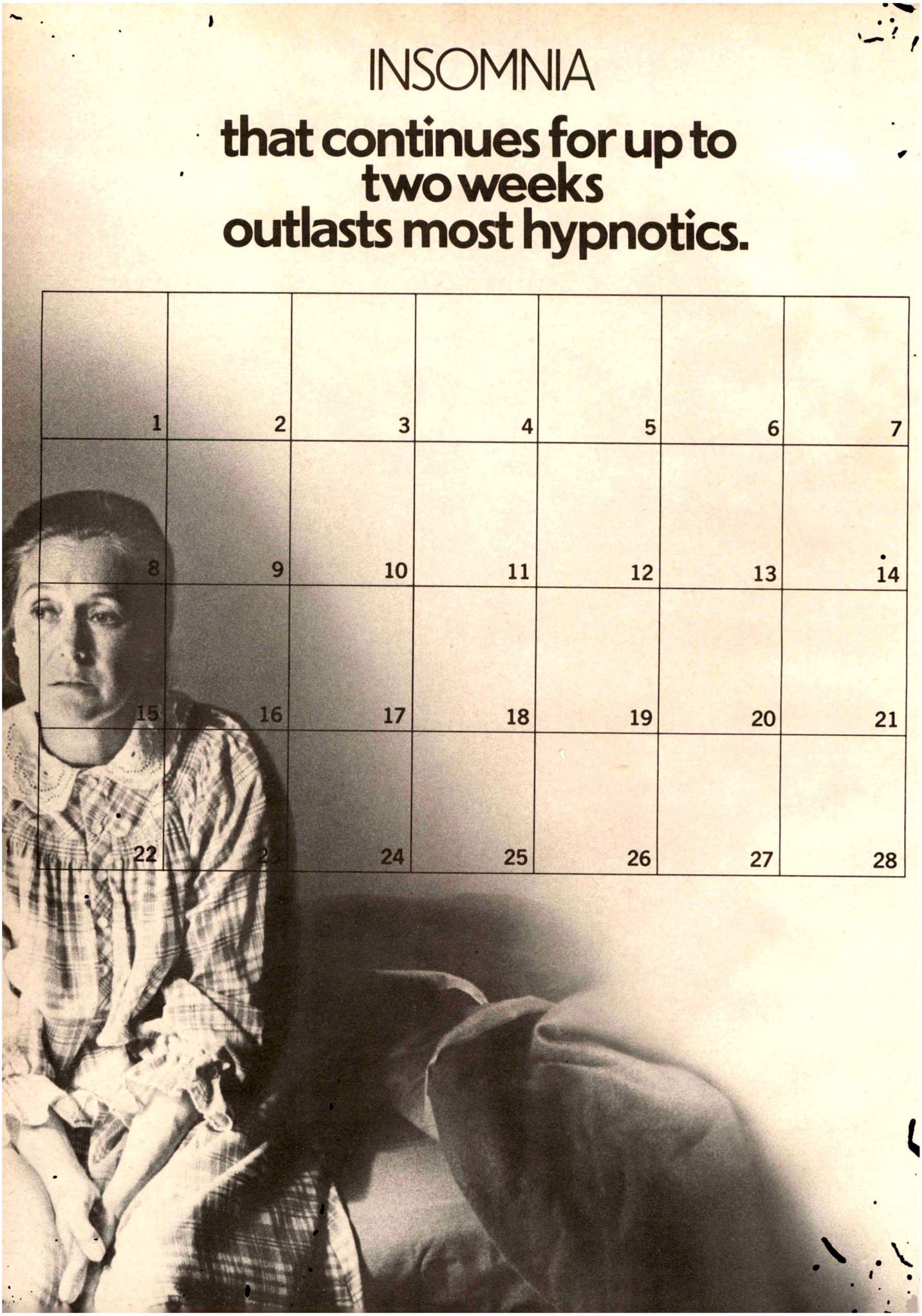


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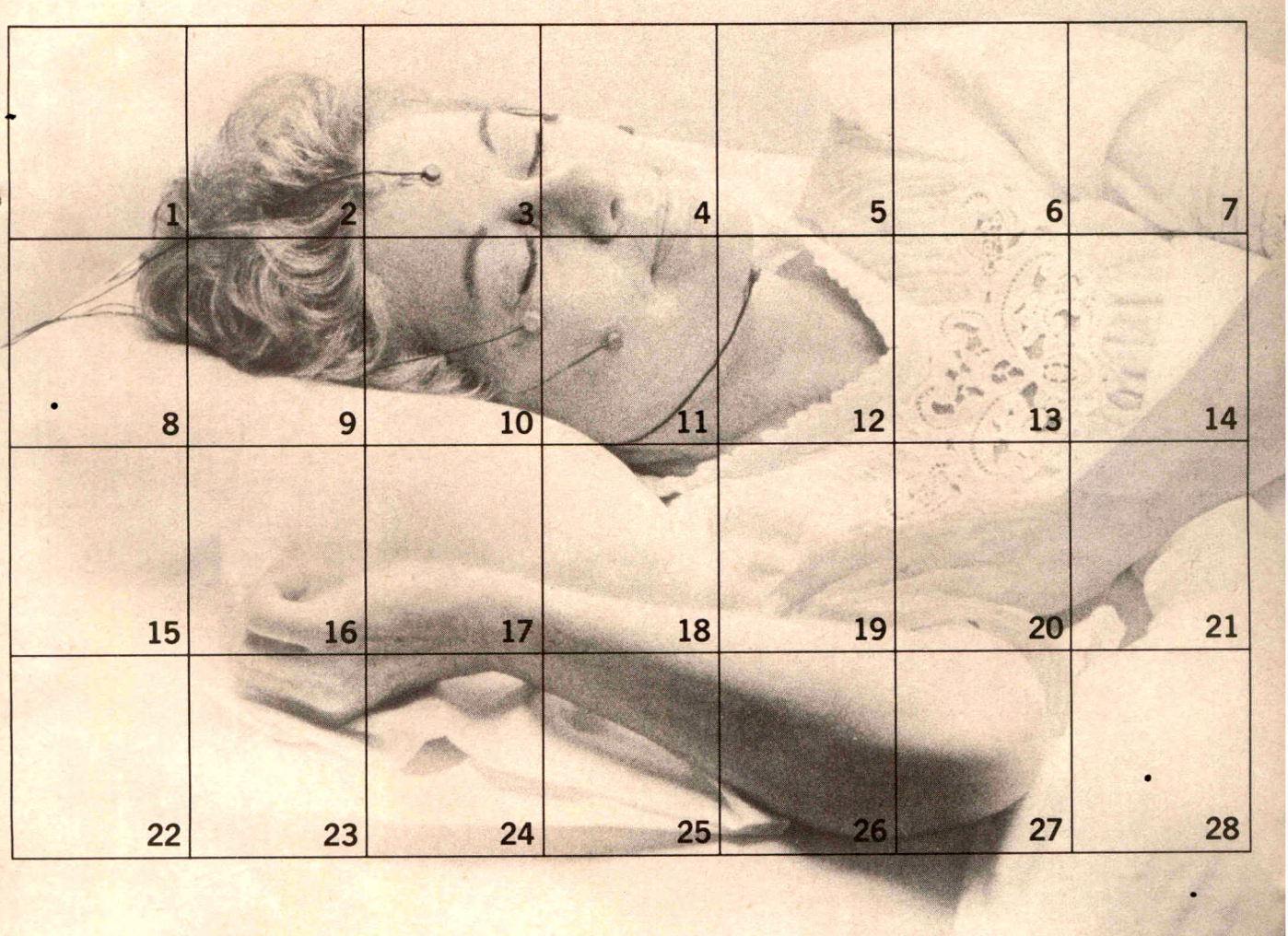
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*Another recently published study showed that ethchlorvynol (500 mg) did not result in a statistically significant reduction of sleep latency, minutes of wake time after sleep onset, number of nighttime awakenings and amount of wakefulness at medication nights 1-3 or at medication nights 12-14.[5]

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Before prescribing Dalmane (flurazepam HCl), please consult complete product information, a summary of which follows:
Indications: Effective in all types of insomnia characterized by difficulty in falling asleep, frequent nocturnal awakenings and/or early morning awakening; in patients with recurring insomnia or poor sleeping habits; in acute or chronic medical situations requiring restful sleep. Objective sleep laboratory data have shown effectiveness for at least 28 consecutive nights of administration. Since insomnia is often transient and intermittent, prolonged administration is generally not necessary or recommended.
Contraindications: Known hypersensitivity to flurazepam HCl.
Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).

Usage in Pregnancy: **Several studies of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) suggest increased risk of congenital malformations during the first trimester of pregnancy. Dalmane, a benzodiazepine, has not been studied adequately to determine whether it may be associated with such an increased risk. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage.
Precautions: In elderly and debilitated, limit dosage to 15 mg to reduce risk of oversedation, dizziness, confusion and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in patients who are severely depressed, or with latent depression or suicidal tendencies, or with impaired renal or hepatic function. Periodic blood counts and liver and kidney function tests are advised during repeated therapy.
Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity, and elevated SGOT, SGPT, total and direct bilirubins and alkaline phosphatase.
Dosage: Individualize for maximum beneficial effect.
Adults: 30 mg usual dosage; 15 mg may suffice in some patients.
Elderly or debilitated patients: 15 mg initially until response is determined.
Supplied: Capsules containing 15 mg or 30 mg flurazepam HCl.

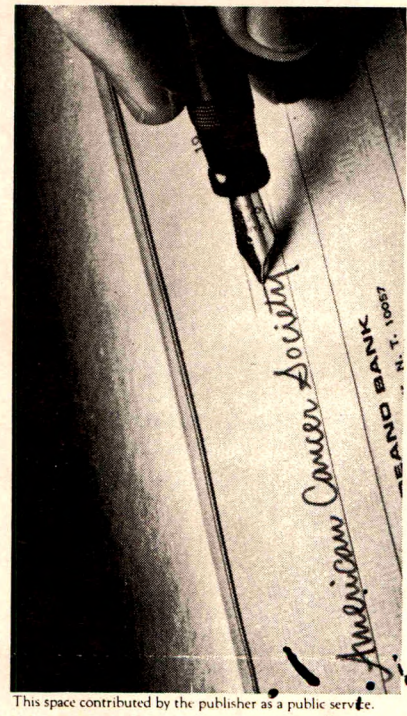
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5. Kripke DF, Lavie P, Hernandez J: *Psychopharmacology* 56:221-223, 1978
6. *AMA Drug Evaluations*, Ed 3. Littleton Mass, Publishing Sciences Group, Inc., 1977, p. XXXIII

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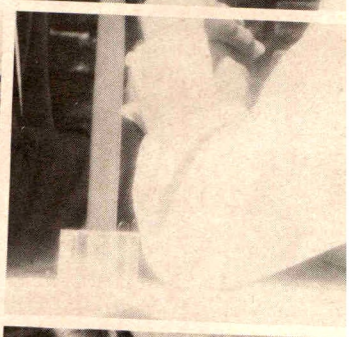
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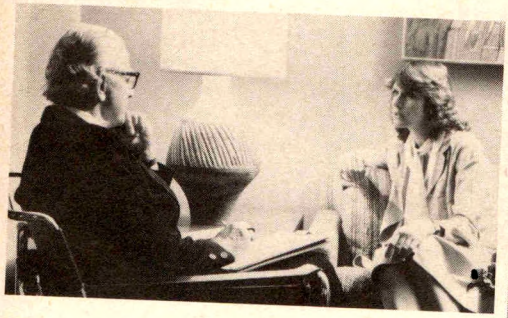
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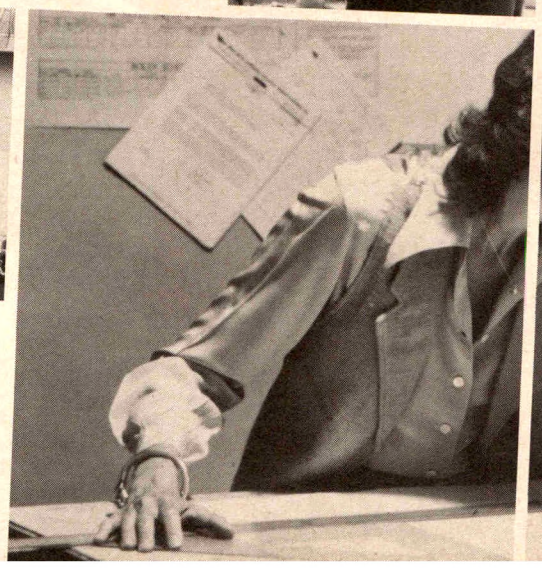


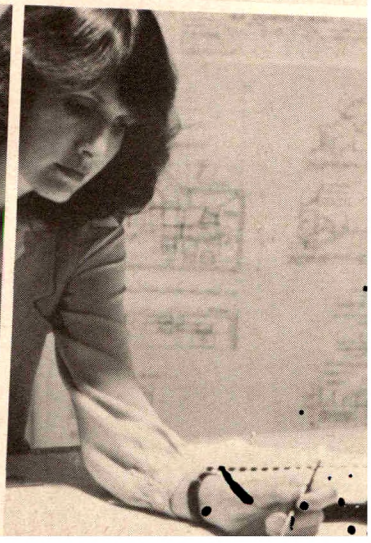












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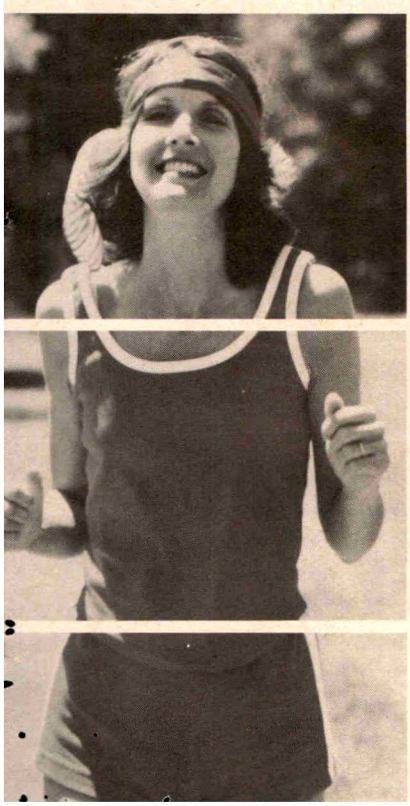
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CONTRAINDICATIONS: Central nervous system depression from drugs (barbiturates, alcohol, narcotics, analgesics, antihistamines); evidence of bone marrow depression; known hypersensitivity to phenothiazines or amitriptyline. Should not be given concomitantly with a monoamine oxidase inhibitor since hyperpyretic crises, severe convulsions, and deaths have occurred from such combinations. When used to replace a monoamine oxidase inhibitor, allow a minimum of 14 days to elapse before initiating therapy with TRIAVIL. Therapy should then be initiated cautiously with gradual increase in dosage until optimum response is achieved. Not recommended for use during acute recovery phase following myocardial infarction.

WARNINGS: TRIAVIL should not be given concomitantly with guanethidine or similarly acting compounds since TRIAVIL may block the antihypertensive action of such compounds. Use cautiously in patients with history of urinary retention, angle-closure glaucoma, increased intraocular pressure, or convulsive disorders. Dosage of anticonvulsive agents may have to be increased. In patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely. Tricyclic antidepressants, including amitriptyline HCl, have been reported to produce arrhythmias, sinus tachycardia, and prolongation of conduction time, particularly in high doses. Myocardial infarction and stroke have been reported with tricyclic antidepressant drugs. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. May impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle. In patients who use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage. Not recommended in children or during pregnancy.

PRECAUTIONS: Suicide is a possibility in depressed patients and may remain until significant remission occurs. Such patients should not have access to large quantities of this drug.

Perphenazine: Should not be used indiscriminately. Use with caution in patients who have previously exhibited severe adverse reactions to other phenothiazines. Likelihood of some untoward actions is greater with high doses. Closely supervise with any dosage. The antiemetic effect of perphenazine may obscure signs of toxicity due to overdosage of other drugs or make more difficult the diagnosis of disorders such as brain tumor or intestinal obstruction. A significant, not otherwise explained, rise in body temperature may suggest individual intolerance to perphenazine, in which case discontinue.

If hypotension develops, epinephrine should not be employed, as its action is blocked and partially reversed by perphenazine. Phenothiazines may potentiate the action of central nervous system depressants (opiates, analgesics, antihistamines, barbiturates, alcohol) and atropine. In concurrent therapy with any of these, TRIAVIL should be given in reduced dosage. May also potentiate the action of heat and phosphorous insecticides. There is sufficient experimental evidence to conclude that chronic administration of antipsychotic drugs which increase prolactin secretion has the potential to induce mammary neoplasms in rodents under the appropriate conditions. There are recognized differences in the physiological role of prolactin between rodents and humans. Since there are, at present, no adequate epidemiological studies, the relevance to human mammary cancer risk from prolonged exposure to perphenazine and other antipsychotic drugs is not known.

Amitriptyline: In manic-depressive psychosis, depressed patients may experience a shift toward the manic phase if they are treated with an antidepressant. Patients with paranoid symptomatology may have an exaggeration of such symptoms. The tranquilizing effect of TRIAVIL seems to reduce the likelihood of this effect. When amitriptyline HCl is given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required. Paralytic ileus may occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs.

Caution is advised if patients receive large doses of ethchlorvynol concurrently. Transient delirium has been reported in patients who were treated with 1 g of ethchlorvynol and 75-150 mg of amitriptyline HCl.

Amitriptyline HCl may enhance the response to alcohol and the effects of barbiturates and other CNS depressants.

Concurrent administration of amitriptyline HCl and electroshock therapy may increase the hazards associated with such therapy. Such treatment should be limited to patients for whom it is essential. Discontinue several days before elective surgery if possible. Elevation and lowering of blood sugar levels have both been reported. Use with caution in patients with impaired liver function.

ADVERSE REACTIONS: Similar to those reported with either constituent alone.
Perphenazine: Extrapyramidal symptoms (opisthotonus, oculogyric crisis, hyperreflexia, dystonia, akathisia, acute dyskinesia, ataxia, parkinsonism) have been reported and can usually be controlled by the concomitant use of effective antiparkinsonian drugs and/or by reduction in dosage, but sometimes persist after discontinuation of the phenothiazine.

Tardive dyskinesia may appear in some patients on long-term therapy or may occur after drug therapy with phenothiazines and related agents has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. Symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw. Involuntary movements of the extremities sometimes occur. There is no known treatment for tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms. It is advised that all antipsychotic agents be discontinued if the above symptoms appear. If treatment is reinstituted, or dosage of the particular drug increased, or another drug substituted, the syndrome may be masked. Fine vermicular movements of the tongue may be an early sign of the syndrome. The full-blown syndrome may not develop if medication is stopped when lingual vermiculation appears.

Other side effects are skin disorders (photosensitivity, itching, erythema, urticaria, eczema, up to exfoliative dermatitis); other allergic reactions (asthma, laryngeal edema, angioneurotic edema, anaphylactoid reactions); peripheral edema; reversed epinephrine effect; hyperglycemia; endocrine disturbances (lactation, galactorrhea, gynecomastia, disturbances of menstrual cycle); altered cerebrospinal fluid proteins; paradoxical excitement; hypertension, hypotension, tachycardia, and ECG abnormalities (quinidine-like effect); reactivation of psychotic processes; catatonic-like states; autonomic reactions, such as dry mouth or salivation, headache, anorexia, nausea, vomiting, constipation, obstipation, urinary frequency or incontinence, blurred vision, nasal congestion, and a change in pulse rate; other adverse reactions reported with various phenothiazine compounds, but not with perphenazine, include grand mal convulsions, cerebral edema, polyphagia, pigmentary retinopathy, photophobia, skin pigmentation, and failure of ejaculation.

The phenothiazine compounds have produced blood dyscrasias (pancytopenia, thrombocytopenic purpura, leukopenia, agranulocytosis, eosinophilia); and liver damage (jaundice, biliary stasis).

Pigmentation of the cornea and lens has been reported to occur after long-term administration of some phenothiazines. Although it has not been reported in patients receiving TRIAVIL, the possibility that it might occur should be considered.

Hypnotic effects, lassitude, muscle weakness, and mild insomnia have also been reported.

Amitriptyline: Note: Listing includes a few reactions not reported for this drug, but which have occurred with other pharmacologically similar tricyclic antidepressant drugs and must be considered when amitriptyline is administered. *Cardiovascular:* Hypotension; hypertension; tachycardia; palpitation; myocardial infarction; arrhythmias; heart block; stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth; blurred vision; disturbance of accommodation; increased intraocular pressure; constipation; paralytic ileus; urinary retention; dilatation of urinary tract. *Allergic:* Skin rash; urticaria; photosensitization; edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis; leukopenia; eosinophilia; purpura; thrombocytopenia. *Gastrointestinal:* Nausea; epigastric distress; vomiting; anorexia; stomatitis; peculiar taste; diarrhea; parotid swelling; black tongue. Rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido; elevated or lowered blood sugar levels. *Other:* Dizziness, weakness; fatigue; headache; weight gain or loss; increased perspiration; urinary frequency; mydriasis; drowsiness; alopecia. *Withdrawal Symptoms:* Abrupt cessation after prolonged administration may produce nausea, headache, and malaise. These are not indicative of addiction.

OVERDOSAGE: All patients suspected of having taken an overdosage should be admitted to a hospital as soon as possible. Treatment is symptomatic and supportive. However, the intravenous administration of 1–3 mg of physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage of physostigmine should be repeated as required particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine. On this basis, in severe overdosage with perphenazine-amitriptyline combinations, symptomatic treatment of central anticholinergic effects with physostigmine salicylate should be considered.

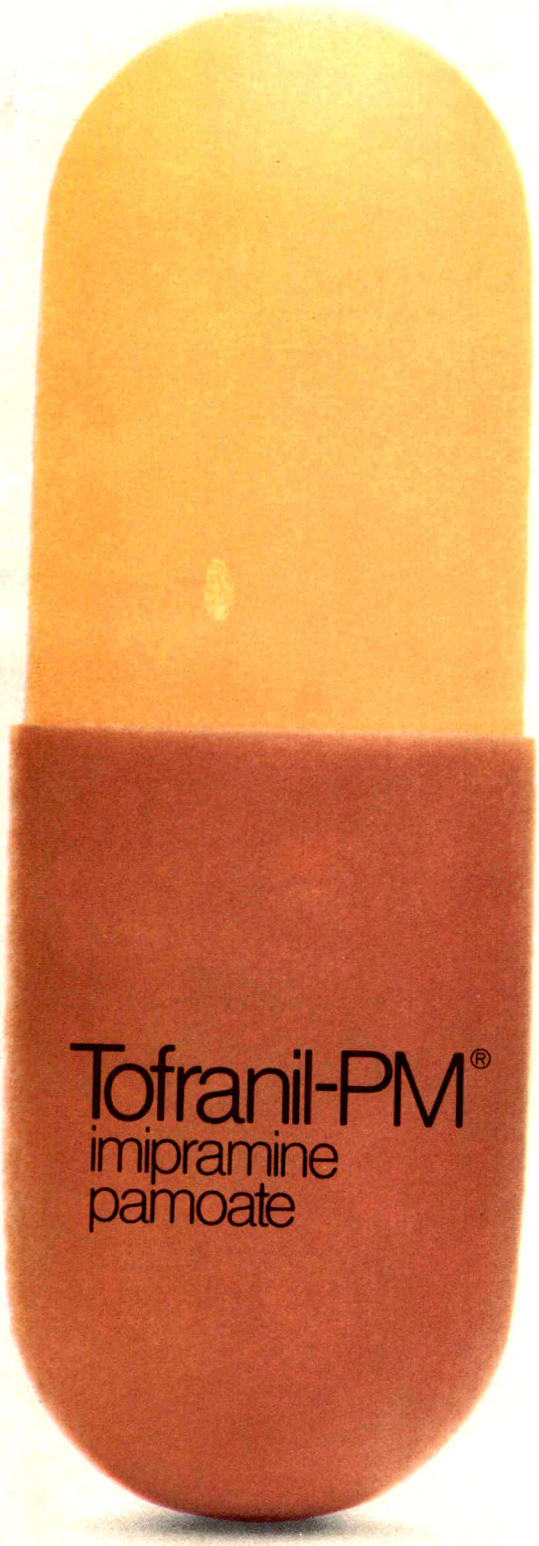
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For more detailed information, consult your MSD Representative or see full Prescribing Information. Merck Sharp & Dohme, Division of Merck & Co., INC., West Point, Pa. 19486.

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for many patients

THE RIGHT ANTIDEPRESSANT



No tricyclic antidepressant works more effectively to brighten the mood, relieve sleep disturbances, and ameliorate mild anxiety.[1,2] And none appears to be better balanced[3] in terms of the biogenic amine theory of depression, which relates depression to functional deficiencies of the catecholamines.

For Tofranil-PM is almost equally effective in inhibiting the postsynaptic uptake of both serotonin and norepinephrine, the two neurotransmitters most often implicated in depression, while other tricyclic antidepressants tested are heavily skewed towards one or the other.*

*Nortriptyline, a chemical derivative of amitriptyline, also exhibits balanced action, but does not appear as effective in treating serotonin-mediated depression as imipramine.

Please see last page for a brief summary of the prescribing information.

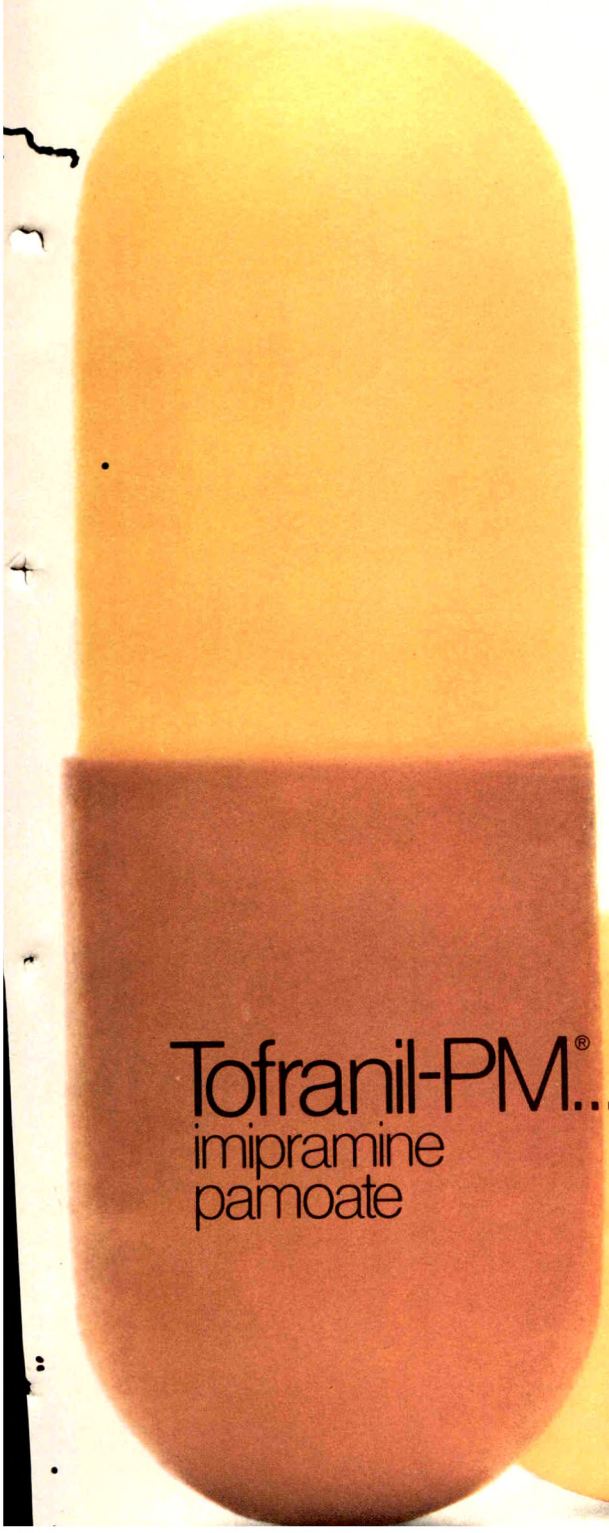
Geigy

for many patients
with mixed
anxiety-depression

THE RIGHT

Geigy

COMBINATION...



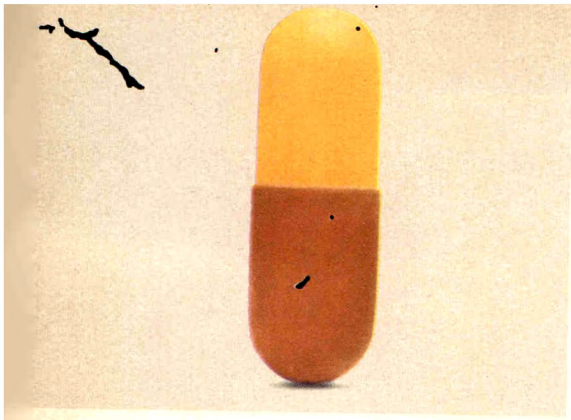
While Tofranil-PM alone exerts a sedative effect in depression, some patients with mixed anxiety-depression require the added control of a minor tranquilizer. For such patients, you can confidently prescribe diazepam (or virtually any other antianxiety agent) adjunctively with Tofranil-PM —unsurpassed among tricyclic antidepressants for its mood-brightening activity.[1, 2]

Tofranil-PM is effective when used alone or in combination regimens. Virtually any combination of psychotropics is possible—a flexibility difficult to achieve with fixed-combination antidepressant regimens.

The decision of which drugs to combine, and at what dosages, remains entirely in your hands . . . where it belongs.

Please see last page for a brief summary of the prescribing information.

Tofranil-PM®...plus **Valium** or any antianxiety agent
imipramine pamoate
(diazepam)



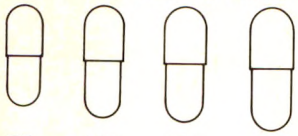
for many patients, the right antidepressant alone... or the basis of the right combination

Tofranil-PM® imipramine pamoate ...unsurpassed antidepressant action

For the majority of patients with neurotic depression, Tofranil-PM may be the only drug needed to produce a satisfactory brightening of mood.[1, 2] A recent report[4] notes that the two leading single-entity tricyclic antidepressants—imipramine pamoate (Tofranil-PM) and amitriptyline—provide equally effective antidepressant activity.

Better quality sleep—usually without morning "hangover"

Recent evaluation confirms that "...patients treated with imipramine pamoate tended ($P < .09$) to have a better quality of sleep than those treated with amitriptyline."[4]



75 mg 100 mg 125 mg 150 mg

Each capsule contains imipramine pamoate equivalent to 75, 100, 125 or 150 mg of imipramine hydrochloride.

Many patients show good results on Tofranil-PM 75 mg h.s.; however, the most effective daily dose may be the 150-mg capsule.

Tofranil-PM®
imipramine pamoate

References

1. Lippman S: Depression—A good approach for the non-psychiatrist. II—Treatment of choice Res and Staff Phys *24* (7):100-104, 1978.
2. Data on file, Geigy Pharmaceuticals, Ardsley, New York 10502.
3. Maas JW: Biogenic amines and depression. Arch Gen Psychiatry *32*:1357-1361, 1975.
4. Goldberg HL, Finnerty RJ: Which tricyclic for depressed outpatients, imipramine pamoate or amitriptyline? Dis Nerv Syst *38*:785-9, 1977.

Tofranil-PM®
brand of imipramine pamoate

Capsules of 75 mg.
Capsules of 100 mg.
Capsules of 125 mg.
Capsules of 150 mg.
For oral administration

Each 75-mg. capsule contains imipramine pamoate equivalent to 75 mg. of imipramine hydrochloride.
Each 100-mg. capsule contains imipramine pamoate equivalent to 100 mg. of imipramine hydrochloride.
Each 125-mg. capsule contains imipramine pamoate equivalent to 125 mg. of imipramine hydrochloride.
Each 150-mg. capsule contains imipramine pamoate equivalent to 150 mg. of imipramine hydrochloride.

Brief Summary of Prescribing Information

Indications For the relief of symptoms of depression. Endogenous depression is more likely to be alleviated than other depressive states.

Contraindications The concomitant use of monoamine oxidase inhibiting compounds is contraindicated. Hyperpyretic crises or severe convulsive seizures may occur in patients receiving such combinations. The potentiation of adverse effects can be serious, or even fatal. When it is desired to substitute Tofranil-PM, brand of imipramine pamoate, in patients receiving a monoamine oxidase inhibitor, as long an interval should elapse as the clinical situation will allow, with a minimum of 14 days. Initial dosage should be low and increases should be gradual and cautiously prescribed.

The drug is contraindicated during the acute recovery period after a myocardial infarction. Patients with a known hypersensitivity to this compound should not be given the drug. The possibility of cross-sensitivity to other dibenzazepine compounds should be kept in mind.

Warnings *Usage in Pregnancy:* Safe use of imipramine during pregnancy and lactation has not been established; therefore, in administering the drug to pregnant patients, nursing mothers, or women of childbearing potential, the potential benefits must be weighed against the possible hazards. Animal reproduction studies have yielded inconclusive results. There have been clinical reports of congenital malformation associated with the use of imipramine, but a causal relationship has not been confirmed.

Extreme caution should be used when this drug is given to:

— patients with cardiovascular disease because of the possibility of conduction defects, arrhythmias, myocardial infarction, strokes and tachycardia;
— patients with increased intraocular pressure, history of urinary retention, or history of narrow-angle glaucoma because of the drug's anticholinergic properties;
— hyperthyroid patients or those on thyroid medication because of the possibility of cardiovascular toxicity;
— patients with a history of seizure disorder because this drug has been shown to lower the seizure threshold;
— patients receiving guanethidine or similar agents since Tofranil-PM, brand of imipramine pamoate, may block the pharmacologic effects of these drugs.

Since imipramine may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks, such as operating an automobile or machinery, the patient should be cautioned accordingly.

Usage in Children: Tofranil-PM, brand of imipramine pamoate, should not be used in children of any age because of the increased potential for acute overdosage due to the high unit potency (75 mg., 100 mg., 125 mg. and 150 mg.). Each capsule contains imipramine pamoate equivalent to 75 mg., 100 mg., 125 mg. or 150 mg. imipramine hydrochloride.

Precautions It should be kept in mind that the possibility of suicide in seriously depressed patients is inherent in the illness and may persist until significant remission occurs. Such patients should be carefully supervised during the early phase of treatment with Tofranil-PM, brand of imipramine pamoate, and may require hospitalization. Prescriptions should be written for the smallest amount feasible.

Hypomanic or manic episodes may occur, particularly in patients with cyclic disorders. Such reactions may necessitate discontinuation of the drug. If needed, Tofranil-PM, brand of imipramine pamoate, may be resumed in lower dosage when these episodes are relieved. Administration of a tranquilizer may be useful in controlling such episodes.

Prior to elective surgery, imipramine should be discontinued for as long as the clinical situation will allow.

An activation of the psychosis may occasionally be observed in schizophrenic patients and may require reduction of dosage and the addition of a phenothiazine.

In occasional susceptible patients or in those receiving anticholinergic drugs (including antiparkinsonism agents) in addition, the atropine-like effects may become more pronounced (e.g., paralytic ileus). Close supervision and careful adjustment of dosage is required when this drug is administered concomitantly with anticholinergic or sympathomimetic drugs.

Avoid the use of preparations, such as decongestants and local anesthetics, which contain any sympathomimetic amine (e.g., adrenalin, noradrenalin), since it has been reported that tricyclic antidepressants can potentiate the effects of catecholamines.

Patients should be warned that the concomitant use of alcoholic beverages may be associated with exaggerated effects.

Both elevation and lowering of blood sugar levels have been reported.

Concurrent administration of imipramine with electroshock therapy may increase the hazards; such treatment should be limited to those patients for whom it is essential, since there is limited clinical experience.

Adverse Reactions Note: Although the listing which follows includes a few adverse reactions which have not been reported with this specific drug, the pharmacological similarities among the tricyclic antidepressant drugs require that each of the reactions be considered when imipramine is administered.

Cardiovascular: Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke, falls.

Psychiatric: Confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia and nightmares; hypomania; exacerbation of psychosis.

Neurological: Numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures, alterations in EEG patterns; tinnitus.

Anticholinergic: Dry mouth, and, rarely, associated sublingual adenitis; blurred vision, disturbances of accommodation, mydriasis; constipation, paralytic ileus; urinary retention, delayed micturition, dilation of the urinary tract.

Allergic: Skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight); edema (general or of face and tongue); drug fever; cross-sensitivity with desipramine.

Hematologic: Bone marrow depression including agranulocytosis; eosinophilia; purpura; thrombocytopenia. Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during therapy; the drug should be discontinued if there is evidence of pathological neutrophil depression.

Gastrointestinal: Nausea and vomiting, anorexia, epigastric distress, diarrhea; peculiar taste, stomatitis, abdominal cramps, black tongue.

Endocrine: Gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido, impotence; testicular swelling; elevation or depression of blood sugar levels.

Other: Jaundice (simulating obstructive); altered liver function; weight gain or loss; perspiration; flushing; urinary frequency; drowsiness, dizziness, weakness and fatigue; headache; parotid swelling; alopecia.

Withdrawal Symptoms: Though not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache and malaise.

How Supplied Tofranil-PM, brand of imipramine pamoate, is available as follows:

75-mg., coral-colored capsules equivalent to 75 mg. imipramine hydrochloride in bottles of 30, 100 and 1,000 capsules.

100-mg., dark yellow/coral-colored capsules equivalent to 100 mg. imipramine hydrochloride in bottles of 30, 100 and 1,000 capsules.

125-mg., light yellow/coral-colored capsules equivalent to 125 mg. imipramine hydrochloride in bottles 30, 100 and 1,000 capsules.

150-mg., coral-colored capsules equivalent to 150 mg. imipramine hydrochloride in bottles of 30, 100 and 1,000 capsules.

667120 (9/75) 98-146-840-A

For complete details, including description, actions, dosage and administration, and overdosage, please see full prescribing information.

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Providing complete psychiatric care in a general hospital setting with special emphasis on disorders of adolescence.

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Announces Two Courses

Clinical Neurology For Psychiatrists:

Part 1 and Part 2

Under the Direction of

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Department of Neurology
Montefiore Hospital and Medical Center

Part 1. Friday, Saturday, and Sunday; March 23, 24 and 25. This course, designed for psychiatrists preparing for the written part of the **American Board of Psychiatry and Neurology**, will consist of a series of lectures, videotaped demonstrations, and a practice written examination. **30 hours of Category 1 credit will be awarded.**

Part 2. Saturday and Sunday; October 6 and 7. the continuation, a course for candidates for the oral section of the Board, will review major neurological disorders by presenting audiovisual demonstrations and practice examinations. **20 hours of Category 1 credit will be awarded.**

FOR FURTHER INFORMATION CONTACT:

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The Brown Schools: Specialists in Residential Treatment

Residential treatment has become highly specialized in the field of mental health. It is a specific treatment modality for those who need a totally planned and structured environment.

The Brown Schools has developed a wide range of professional services that can be utilized to implement an individually planned residential treatment program. The degree of structure and protection, the intensity of therapy, the methods of education and training are controlled and modified with the resident's changing needs.

Professionals in the areas of psychiatry, psychology, nursing, social work, education, prevocational training, speech pathology, and recreation have developed expertise in the specific area of residential treatment. Each service area is designed as a component of an integrated therapeutic milieu.

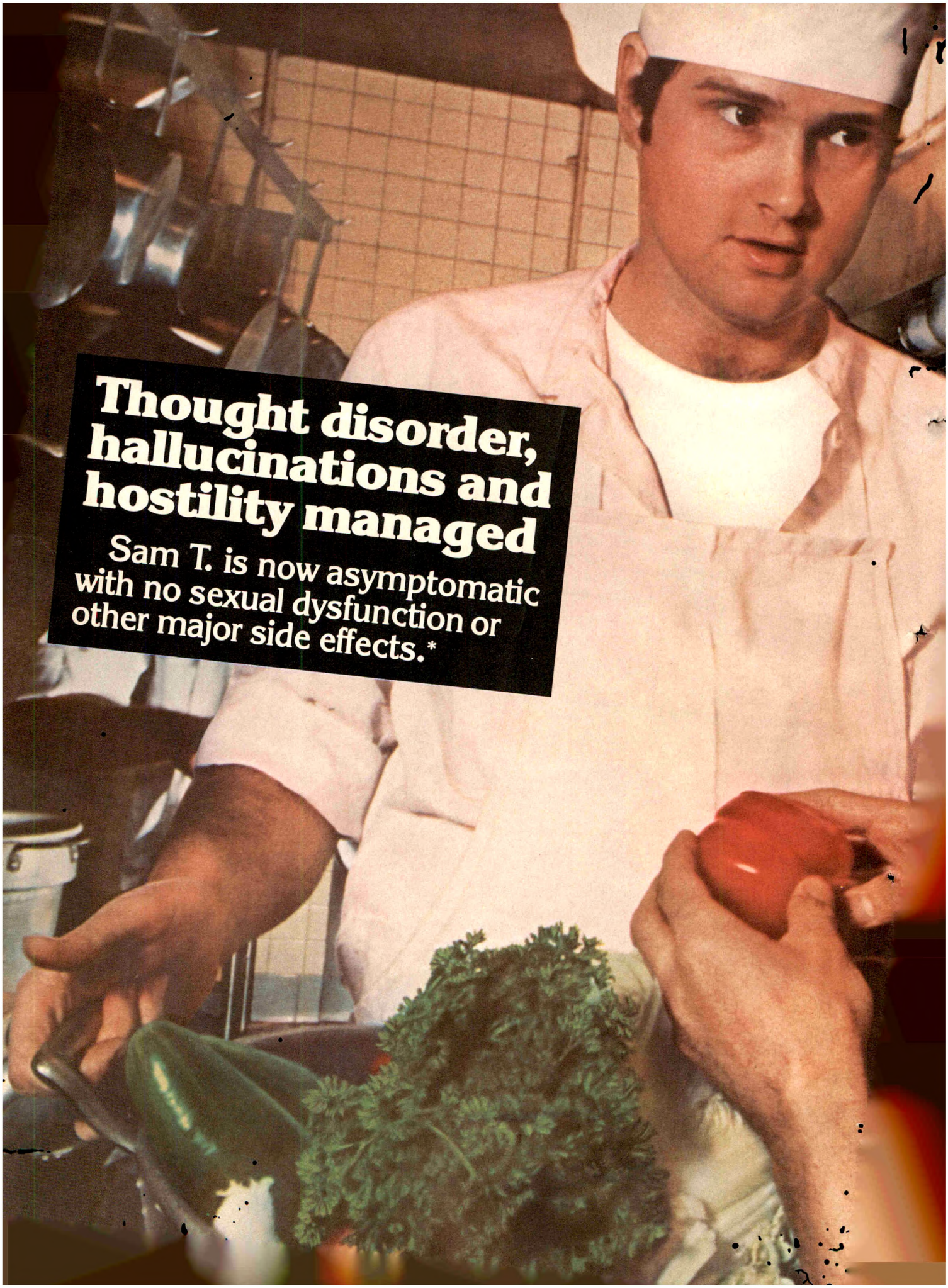
The three residential treatment centers of The Brown Schools provide complete programming for those in need of twenty-four hour care. Services are available for children, adolescents, and adults with emotional disturbance, mental retardation, and neurological impairment. Two small group homes in Austin provide for reintegration into the community and complement the services offered in the other centers. For information, write:

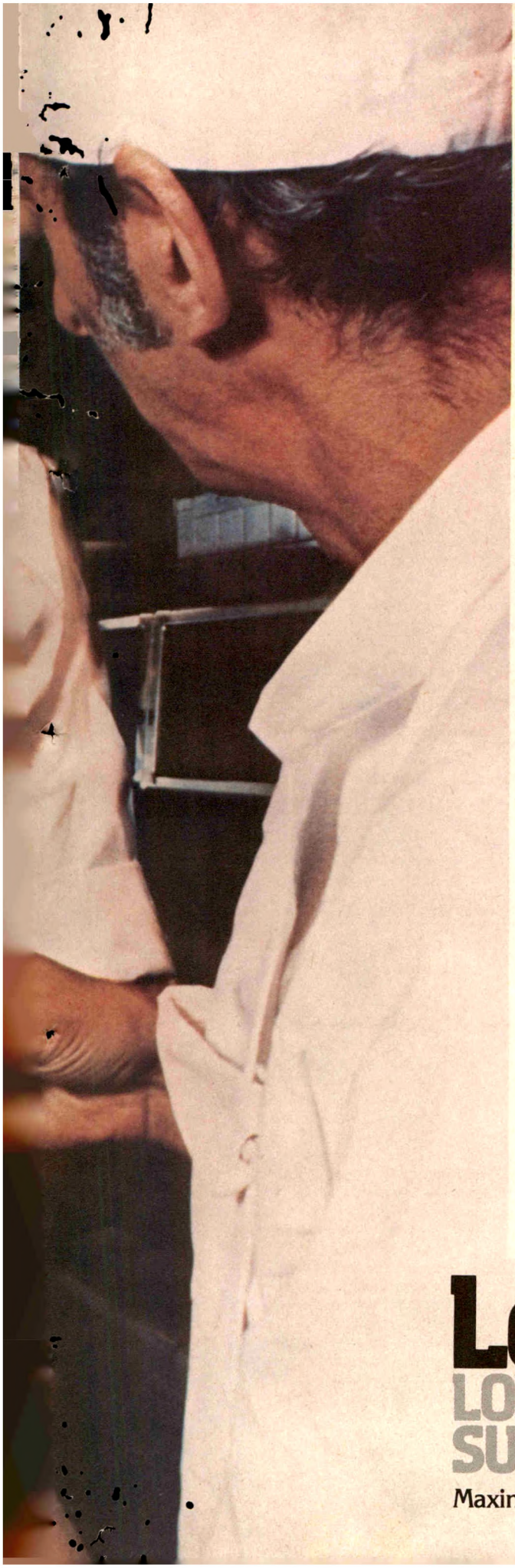
Director of Admissions/
Department C-O
The Brown Schools
P.O. Box 4008,
Austin, Texas 78765.
Toll Call: (512) 478-6662
Out of State Free: (800) 531-5305
From Texas Free: (800) 252-5404

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Thought disorder, hallucinations and hostility managed
Sam T. is now asymptomatic with no sexual dysfunction or other major side effects.*



LOXITANE® Loxapine Succinate

Permits rapid management of Sam's hostility and excited behavior

Thirty-six year old salad maker, claiming to be the "Wild Man of the West" was brought to the hospital by his kitchen supervisor. History revealed prior psychiatric hospitalizations and therapies including psychotropic drugs. Hypertension was noted as a complication of earlier treatment with anti-psychotic drugs. Diagnosis: chronic schizophrenia, acute exacerbation. He responded promptly to LOXITANE® C Loxapine HCl Concentrate 50 mg stat, followed by LOXITANE® Loxapine Succinate capsules 30 mg t.i.d.

Permits aggressive titration for rapid establishment of optimal management of Sam's thought disorder and hallucinations

During first week, LOXITANE dosage increased to 50 mg q.i.d. Significant improvement in thought disorder noted by third week. Auditory hallucinations and delusions much less frequent. Patient calmer and more cooperative.

Permits sustained symptom management with minimal risk of serious adverse effects, such as sexual dysfunction

Patient revealed that while on outpatient status, he failed to take his previously prescribed antipsychotic for a ten-month period after he met and married his girlfriend. He stated the drug "didn't let me be a man." No sexual dysfunction reported with LOXITANE.

Side effects are usually mild, transitory and easily managed

Patient felt anxious because his "heart was beating so fast," and he "could not sit still." Increased pulse rate and restlessness subsided following decrease of LOXITANE to 20 mg q.i.d.

Consistence in schizophrenic symptom management and tolerance promotes patient compliance

All admitting symptomatology under control by the fourth week. By the sixth week, he was anxious to return home. Stated "this medicine doesn't make me feel drowsy like the other did." Discharged by the eighth week. He returned to work fully managed on LOXITANE 20 mg t.i.d., and free from recurrence of previously reported side effects.

*Painful ejaculation and urine containing sperm have not been reported. Endocrine disturbances such as galactorrhea and menstrual irregularities have only been rarely reported. Other sexual side effects infrequently reported. Although not reported to date, possibility of hepatic, renal, ocular, or phototoxicity cannot be ruled out at this time. Transient liver enzyme changes not definitely related to LOXITANE have been reported. Not an actual patient, but a simulation to illustrate the action of LOXITANE as reported in clinical studies.

See LOXITANE prescribing information on following page for indications, warnings and precautions and for more detailed information concerning side effects.

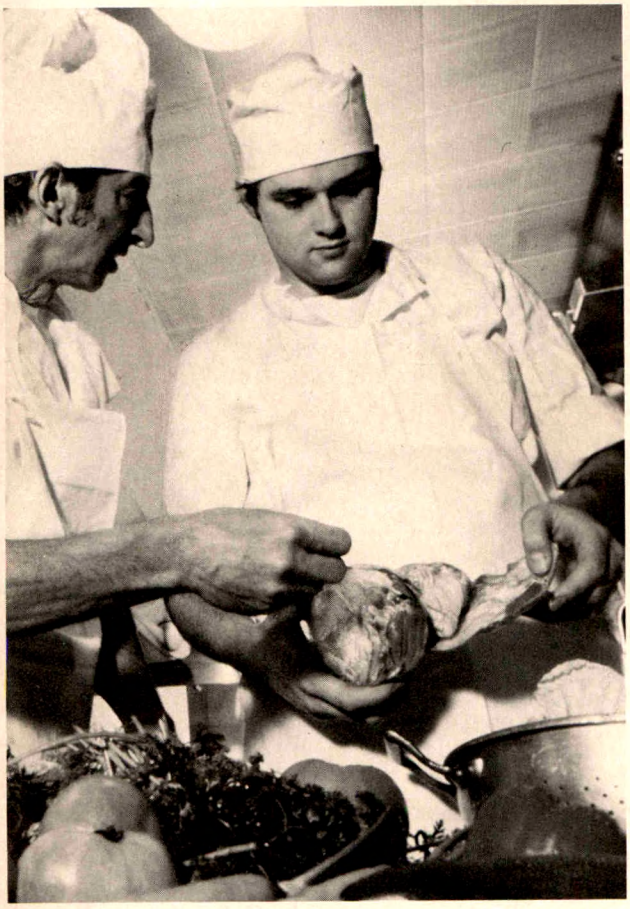
Loxitane®

LOXAPINE SUCCINATE Lederle

Capsules:
5 mg, 10 mg,
25 mg, 50 mg

Maximum management... minimum risk

Maximum management... minimum risk



Loxitane®
LOXAPINE Lederle SUCCINATE

Recommended Daily Dosage

Initial Dosage	MILD	MODERATE	SEVERE
	10 mg b.i.d.	10 mg t.i.d or q.i.d.	25 mg b.i.d.
First 7 to 10 days	Increase dosage until psychotic symptoms are controlled. Dosage should not exceed 250 mg/day. Usual dosage during titration: 50 to 150 mg/day.		
Maintenance Dosage	Adjust to lowest effective level. Usual maintenance dosage: 60 to 100 mg/day. Many patients are controlled with dosages as low as 20 to 60 mg/day.		

Brief Summary
LOXITANE® Loxapine Succinate Capsules
LOXITANE® C Loxapine Hydrochloride Oral Concentrate
INDICATIONS: Manifestations of schizophrenia.
CONTRAINDICATIONS: Comatose or severely depressed states; hypersensitivity to the drug.
WARNINGS: Safe use during pregnancy or lactation has not been established; weigh potential benefits to possible hazards. Not recommended for use in children under 16. May impair mental and/or physical abilities especially during early therapy; warn ambulatory patients about activities requiring alertness and concomitant use of alcohol or other CNS depressants. Not recommended for management of behavioral complications in mentally retarded patients.
PRECAUTIONS: Use with extreme caution in patients with a history of convulsive disorders; use with caution in patients with cardiovascular disease or in those with glaucoma or a tendency to urinary retention particularly when on concomitant anticholinergic medication. Loxapine has an antiemetic effect in animals which might occur in man masking signs of overdosage of toxic drugs and obscuring intestinal obstruction or brain tumor. Since possible ocular toxicity cannot be excluded, observe carefully for pigmentary retinopathy and lenticular pigmentation.
ADVERSE REACTIONS: CNS effects, other than extrapyramidal, infrequent. Mild drowsiness may occur at beginning of therapy or upon dosage increase, usually subsides with continued therapy. Sedation, dizziness, faintness, staggering gait, muscle twitching, weakness and confusional states have been reported. Extrapyramidal reactions often occur early in treatment manifested by Parkinson-like symptoms (tremor, rigidity, excessive salivation, masked facies, akathisia) controllable by dosage reduction or antiparkinson drugs at usual dosages. Dystonic and dyskinetic reactions, while less frequently occurring, may be more severe, requiring dosage reduction or temporary withdrawal plus appropriate counteractive drugs. Persistent Tardive Dyskinesia may appear during prolonged therapy or following discontinuance, the risk greater in the elderly, especially females, on high dosage. Symptoms, persistent and in some patients apparently irreversible, are characterized by rhythmical involuntary movement of the tongue, face, mouth and jaw sometimes accompanied by involuntary movement of extremities. Since there is no known effective treatment, discontinue all antipsychotic drugs if symptoms appear. Reinstitution of treatment, increased dosage, or switching to another agent may mask syndrome. The syndrome may not develop if medication is stopped when fine vermicular movements of the tongue first appear. Cardiovascular Effects: Tachycardia, hypotension, hypertension, lightheadedness and syncope. ECG changes, not known to be related to loxapine use, have been reported. Skin: Dermatitis, edema of face, pruritus, seborrhea. Possible photosensitivity and/or phototoxicity; skin rashes of unknown etiology seen in a few patients in hot summer months. Anticholinergic: Dry mouth, nasal congestion, constipation, blurred vision (more likely to occur with concomitant use of antiparkinson agents). Other: Nausea, vomiting, weight gain or loss, dyspnea, ptosis, hyperpyrexia, flushed facies, headache, paresthesia, polydipsia. Rarely, galactorrhea and menstrual irregularity of unknown etiology.

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Chlorpromazine
Haloperidol
Thioridazine
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Trifluoperazine
Fluphenazine .2HCl
Fluphenazine decanoate
Butaperazine
Acetophenazine
Piperacetazine
Perphenazine

Antianxiety

*Diazepam
Oxazepam
Chlordiazepoxide

LITHIUM
Plasma Lithium
RBC Lithium

MHPG
3-methoxy-4 hydroxy-phenethyleneglycol

Others

*The secondary amines automatically assayed with no extra charge.

Reaching therapeutic dosages

QUICK HELPFUL INFORMATION FOR PHYSICIANS

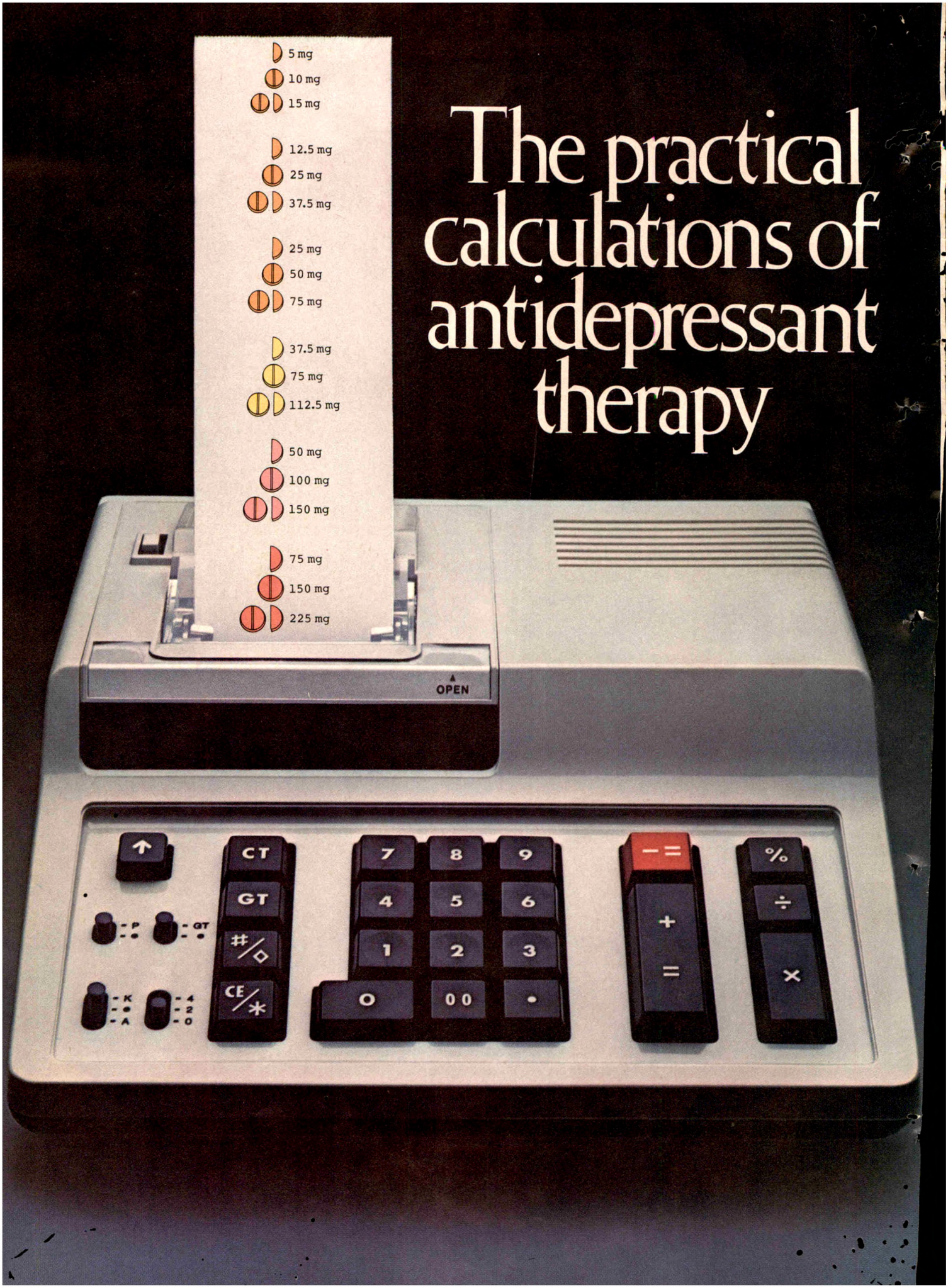
Reduction of unwanted side effects

Maintaining good remissions

Increasing the % of patients who will respond to drug therapy

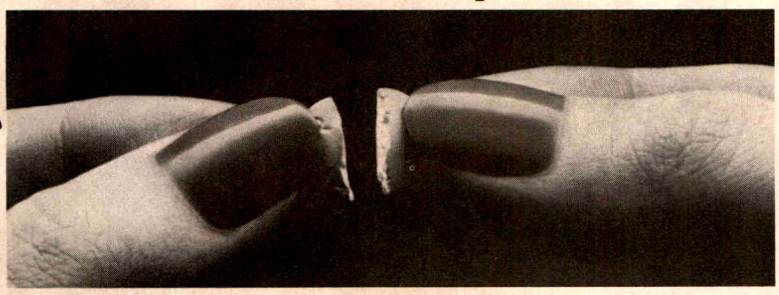
Drug compliance

FOR THE ABOVE ASSAYS, GAS CHROMATOGRAPHY WITH A NITROGEN DETECTOR IS USED. THIS METHODOLOGY GIVES VALUES CONSISTENT WITH THOSE REPORTED IN CLINICAL AND RESEARCH LITERATURE.


5 mg
10 mg
15 mg
12.5 mg
25 mg
37.5 mg
25 mg
50 mg
75 mg
37.5 mg
75 mg
112.5 mg
50 mg
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225 mg
The practical calculations of antidepressant therapy
OPEN
CT
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Divide scored Endep tablets



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Add flexibility to dosage titration

Long term treatment of depression with amitriptyline requires precise calculation of dosage to meet the individual needs of each patient. Because all six strengths of Endep are scored, you can easily increase or decrease dosage in half-tablet steps, without the bother and expense of a new prescription.

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Subtract waste and cost

Scored Endep tablets permit you to adjust the regimen without wasting unused tablets or burdening the patient with the cost of a new prescription.

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Multiply therapeutic options

The pharmacokinetic properties of Endep are such that its clinical effect is the same whether prescribed *t.i.d.* or as a single daily dose. Thus, once you have established the proper dosage, you can prescribe the entire dose *h.s.*, minimizing anticholinergic and sedative side effects, and helping to assure patient compliance with this simple regimen.

By prescribing the higher strength tablets *h.s.*—secure in the knowledge that the dosage can be adjusted without resorting to a new prescription—your patient will economize further on a per milligram basis.

Endep®

amitriptyline HCl/Roche

The only antidepressant scored for easy division

Endep
amitriptyline HCl/Roche

Before prescribing, please consult complete product information, a summary of which follows:

Contraindications: Known hypersensitivity. Do not use with monoamine oxidase (MAO) inhibitors or within at least 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use; then initiate cautiously, gradually increasing dosage until optimal response is achieved. Use not recommended during acute recovery phase after myocardial infarction.

Warnings: May block action of guanethidine or similar antihypertensives. Use with caution in patients with history of seizures, urinary retention, angle-closure glaucoma, increased intraocular pressure. Closely supervise cardiovascular patients, hyperthyroid patients and those receiving thyroid medications. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, including amitriptyline HCl, especially in high doses. Myocardial infarction and stroke reported with use of this class of drugs.) May impair alertness; warn against hazardous occupations or driving a motor vehicle during therapy. Weigh possible benefits against hazards during pregnancy, the nursing period and in women of childbearing potential. Not recommended in children under 12.

Precautions: May exaggerate symptoms in schizophrenic and paranoid patients, or shift manic-depressives to manic stage; reduce dose or administer major tranquilizer concomitantly. Close supervision and careful dose adjustments required when given with anticholinergic or sympathomimetic agents. Exercise care in patients receiving large doses of ethchlorvynol; transient delirium reported with concomitant administration. May enhance effects of alcohol, barbiturates and other CNS depressants. Because of the possibility of suicide in depressed patients, do not permit easy access to large drug quantities in these patients. Because it may increase hazards of electroshock therapy, limit concomitant use to essential treatment. If possible, discontinue drug several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported.

Adverse Reactions: Note: This list includes a few adverse reactions not reported with this specific drug but requiring consideration because of similarities of tricyclic antidepressants. *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, constipation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, black tongue. *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, jaundice, alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache and malaise. These are not indicative of addiction.

Supplied: *Scored Tablets:* 10, 25, 50, 75, 100, 150 mg.

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PROLIXI

The Prolixin® (Fluphenazine) system of schizophrenia management—

spanning virtually every clinical situation

There is scarcely a patient-type presenting with schizophrenia that cannot be successfully managed with one of the numerous forms of Prolixin (Fluphenazine).

For the newly admitted, agitated patient, a first choice would be short-acting Prolixin Injection (Fluphenazine Hydrochloride Injection USP).

For long-term management of more "dependable" patients, you have the titration flexibility of four potencies of Prolixin Tablets (Fluphenazine Hydrochloride Tablets USP).

For patients who "cheek" or otherwise resist tablet medication, you can utilize pleasant tasting, orange-flavored Prolixin Elixir (Fluphenazine Hydrochloride Elixir USP).

For long-term maintenance of less compliant patients, you can employ unique, long-acting Prolixin Decanoate (Fluphenazine Decanoate Injection) and achieve effective neuroleptization for up to four weeks and longer with a single injection.

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25 mg in 1 ml

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Hydrochloride
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10 ml multiple dose vial
2.5 mg per ml

5 ml
multiple dose vial
25 mg per ml

Prolixin® Elixir
Fluphenazine
Hydrochloride
Elixir USP

0.5 mg per ml

Prolixin® Tablets
Fluphenazine
Hydrochloride
Tablets USP

10 mg | 5 mg | 2.5 mg | 1 mg

See next page for brief summaries.

PROLIXIN® (Fluphenazine Hydrochloride)
TABLETS/ELIXIR/INJECTION

Prolixin Tablets (Fluphenazine Hydrochloride Tablets USP) provide 1, 2.5, 5, or 10 mg fluphenazine hydrochloride per tablet. Prolixin Elixir (Fluphenazine Hydrochloride Elixir USP) provides 0.5 mg fluphenazine hydrochloride per ml (2.5 mg per 5 ml teaspoonful) with 14% alcohol by volume. Prolixin Injection (Fluphenazine Hydrochloride Injection USP) provides 2.5 mg fluphenazine hydrochloride per ml; it contains 0.1% methylparaben and 0.01% propylparaben as preservatives.

CONTRAINDICATIONS: In presence of suspected or established subcortical brain damage. In patients who have a blood dyscrasia or liver damage, or who are receiving large doses of hypnotics, or who are comatose or severely depressed. In patients who have shown hypersensitivity to fluphenazine; cross-sensitivity to phenothiazine derivatives may occur.

WARNINGS: Mental and physical abilities required for driving a car or operating heavy machinery may be impaired by use of this drug. Potentiation of effects of alcohol may occur. Safety and efficacy in children have not been established because of inadequate experience in use in children.

Usage in Pregnancy: Safety for use during pregnancy has not been established; weigh possible hazards against potential benefits if administering this drug to pregnant patients.

PRECAUTIONS: Caution must be exercised if another phenothiazine compound caused cholestatic jaundice, dermatoses or other allergic reactions because of the possibility of cross-sensitivity. When psychotic patients on large doses of a phenothiazine drug are to undergo surgery, hypotensive phenomena should be watched for; less anesthetics or central nervous system depressants may be required. Because of added anticholinergic effects, fluphenazine may potentiate the effects of atropine.

Use fluphenazine cautiously in patients exposed to extreme heat or phosphorus insecticides; in patients with a history of convulsive disorders since grand mal convulsions have occurred; and in patients with special medical disorders such as mitral insufficiency or other cardiovascular diseases, and pheochromocytoma. Bear in mind that with prolonged therapy there is the possibility of liver damage, pigmentary retinopathy, lenticular and corneal deposits, and development of irreversible dyskinesia.

There is sufficient experimental evidence to conclude that chronic administration of antipsychotic drugs which increase prolactin secretion has the potential to induce mammary neoplasms in rodents under the appropriate conditions. There are recognized differences in the physiological role of prolactin between rodents and humans. Since there are, at present, no adequate epidemiological studies, the relevance to human mammary cancer risk from prolonged exposure to fluphenazine hydrochloride and other antipsychotic drugs is not known.

Periodic checking of hepatic and renal functions and blood picture should be done. Monitor renal function of patients on long-term therapy; if BUN becomes abnormal, discontinue fluphenazine. "Silent pneumonias" are possible.

Abrupt Withdrawal: In general, phenothiazines do not produce psychic dependence. However, gastritis, nausea and vomiting, dizziness, and tremulousness have been reported following abrupt cessation of high dose therapy; reports suggest that these symptoms can be reduced if concomitant antiparkinsonian agents are continued for several weeks after the phenothiazine is withdrawn.

ADVERSE REACTIONS: *Central Nervous System*—Extrapyramidal symptoms are most frequently reported. Most often these symptoms are reversible, but they may be persistent. They include pseudoparkinsonism, dystonia, dyskinesia, akathisia, oculogyric crises, opisthotonos, hyperreflexia. The incidence and severity of such reactions will depend more on individual patient sensitivity, but dosage level and patient age are also determinants. As these reactions may be alarming, the patient should be forewarned and reassured. These reactions can usually be controlled by administration of an anti-parkinsonian drug such as benztropine mesylate and by subsequent reduction in dosage.

Persistent Tardive Dyskinesia: As with all antipsychotic agents, persistent and sometimes irreversible tardive dyskinesia may appear in some patients on long-term therapy or may occur after discontinuation of drug. The risk seems greater in elderly patients, especially females, on high dosages. The syndrome is characterized by rhythmical involuntary movements of tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and may be accompanied by involuntary movements of extremities. There is no known effective therapy for tardive dyskinesia; usually the symptoms are not alleviated by antiparkinsonism agents. If the symptoms appear, discontinuation of all antipsychotic agents is suggested. The syndrome may be masked if treatment is reinstituted, or drug dosage increased, or a different antipsychotic agent used. Reports are that fine vermicular movements of the tongue may be an early sign of the syndrome which may not develop if medication is stopped at that time.

Phenothiazine derivatives have been known to cause restlessness, excitement, or bizarre dreams; reactivation or aggravation of psychotic processes may be encountered. If drowsiness or lethargy occur, the dosage may need to be reduced. Dosages, far in excess of the recommended amounts, may induce a catatonic-like state.

Autonomic Nervous System—Hypertension and fluctuations in blood pressure have been reported. Although hypotension is rarely a problem, patients with pheochromocytoma, cerebral vascular or renal insufficiency or severe cardiac reserve deficiency such as mitral insufficiency appear to be particularly prone to this reaction and should be observed carefully. Supportive measures including intravenous vasopressor drugs should be instituted immediately should severe hypotension occur; Levarterenol Bitartrate Injection is the most suitable drug; *epinephrine should not be used* since phenothiazine derivatives have been found to reverse its action. Nausea, loss of appetite, salivation, polyuria, perspiration, dry mouth, headache and constipation may occur. Reducing or temporarily discontinuing the dosage will usually control these effects. Blurred vision, glaucoma, bladder paralysis, fecal impaction, paralytic ileus, tachycardia, or nasal congestion have occurred in some patients on phenothiazine derivatives.

Metabolic and Endocrine—Weight change, peripheral edema, abnormal lactation, gynecomastia, menstrual irregularities, false results on pregnancy tests, impotency in men and increased libido in women have occurred in some patients on phenothiazine therapy.

Allergic Reactions—Itching, erythema, urticaria, seborrhea, photosensitivity, eczema and exfoliative dermatitis have been reported with phenothiazines. The possibility of anaphylactoid reactions should be borne in mind.

Hematologic—Blood dyscrasias including leukopenia, agranulocytosis, thrombocytopenic or nonthrombocytopenic purpura, eosinophilia, and pancytopenia have been observed with phenothiazines. If soreness of the mouth, gums or throat or any symptoms of upper respiratory infection occur and confirmatory leukocyte count indicates cellular depression, therapy should be discontinued and other appropriate measures instituted immediately.

Hepatic—Liver damage manifested by cholestatic jaundice, particularly during the first months of therapy, may occur; treatment should be discontinued. A cephalin flocculation increase, sometimes accompanied by alterations in other liver function tests, has been reported in patients who have had no clinical evidence of liver damage.

Others—Sudden deaths have been reported in hospitalized patients on phenothiazines. Previous brain damage or seizures may be predisposing factors. High doses should be avoided in known seizure patients. Shortly before death, several patients showed flare-ups of psychotic behavior patterns. Autopsy findings have usually revealed acute fulminating pneumonia or pneumonitis, aspiration of gastric contents, or intramyocardial lesions. Although not a general feature of fluphenazine, potentiation of central nervous system depressants such as opiates, analgesics, antihistamines, barbiturates, and alcohol may occur.

Systemic lupus erythematosus-like syndrome, hypotension severe enough to cause fatal cardiac arrest, altered electrocardiographic and electroencephalographic tracings, altered cerebrospinal fluid proteins, cerebral edema, asthma, laryngeal edema, and angioneurotic edema; with long-term use, skin pigmentation and lenticular and corneal opacities have occurred with phenothiazines. Local tissue reactions occur only rarely with injections of fluphenazine decanoate.

For full prescribing information, consult package inserts.

HOW SUPPLIED: *Tablets*—1 mg in bottles of 50 and 500; 2.5 mg and 5 mg in bottles of 50 and 500 and in Unimatic® single-dose cartons of 100; 10 mg in bottles of 50 and 500. *Elixir*—in bottles of 473 ml (1 pint) and in 60 ml dropper-assembly bottles with dropper calibrated at 0.5 ml (0.25 mg), 1 ml (0.5 mg), 1.5 ml (0.75 mg), and 2 ml (1 mg). *Injection*—in multiple-dose vials of 10 ml.

PROLIXIN DECANOATE®
Fluphenazine Decanoate Injection

Prolixin Decanoate (Fluphenazine Decanoate Injection) provides 25 mg fluphenazine decanoate per ml in a sesame oil vehicle with 1.2% (w/v) benzyl alcohol as a preservative.

CONTRAINDICATIONS: In presence of suspected or established subcortical brain damage. In patients who have a blood dyscrasia or liver damage, or who are receiving large doses of hypnotics, or who are comatose or severely depressed. In patients who have shown hypersensitivity to fluphenazine; cross-sensitivity to phenothiazine derivatives may occur.

Not intended for use in children under 12.

WARNINGS: Mental and physical abilities required for driving a car or operating heavy machinery may be impaired by use of this drug. Physicians should be alert to the possibility that severe adverse reactions may occur which require immediate medical attention. Potentiation of effects of alcohol may occur. Safety and efficacy in children have not been established because of inadequate experience in use in children.

Usage in Pregnancy: Safety for use during pregnancy has not been established; weigh possible hazards against potential benefits if administering this drug to pregnant patients.

PRECAUTIONS: Caution must be exercised if another phenothiazine compound caused cholestatic jaundice, dermatoses or other allergic reactions because of the possibility of cross-sensitivity. When psychotic patients on large doses of a phenothiazine drug are to undergo surgery, hypotensive phenomena should be watched for; less anesthetics or central nervous system depressants may be required. Because of added anticholinergic effects, fluphenazine may potentiate the effects of atropine.

Use fluphenazine decanoate cautiously in patients exposed to extreme heat or phosphorus insecticides; in patients with a history of convulsive disorders since grand mal convulsions have occurred; and in patients with special medical disorders such as mitral insufficiency or other cardiovascular diseases, and pheochromocytoma. Bear in mind that with prolonged therapy there is the possibility of liver damage, pigmentary retinopathy, lenticular and corneal deposits, and development of irreversible dyskinesia.

Fluphenazine decanoate should be administered under the direction of a physician experienced in the clinical use of psychotropic drugs. Periodic checking of hepatic and renal functions and blood picture should be done. Renal function of patients on long-term therapy should be monitored; if BUN becomes abnormal, treatment should be discontinued. "Silent pneumonias" are possible.

There is sufficient experimental evidence to conclude that chronic administration of antipsychotic drugs which increase prolactin secretion has the potential to induce mammary neoplasms in rodents under the appropriate conditions. There are recognized differences in the physiological role of prolactin between rodents and humans. Since there are, at present, no adequate epidemiological studies, the relevance to human mammary cancer risk from prolonged exposure to fluphenazine decanoate and other antipsychotic drugs is not known.

ADVERSE REACTIONS: *Central Nervous System*—Extrapyramidal symptoms are most frequently reported. Most often these symptoms are reversible, but they may be persistent. They include pseudoparkinsonism, dystonia, dyskinesia, akathisia, oculogyric crises, opisthotonos, hyperreflexia. Muscle rigidity sometimes accompanied by hyperthermia has been reported following use of fluphenazine decanoate. One can expect a higher incidence of such reactions with fluphenazine decanoate than with less potent piperazine derivatives or straight-chain phenothiazines. The incidence and severity will depend more on individual patient sensitivity, but dosage level and patient age are also determinants. As these reactions may be alarming, the patient should be forewarned and reassured. These reactions can usually be controlled by administration of an antiparkinsonian drug such as benztropine mesylate and by subsequent reduction in dosage.

Persistent Tardive Dyskinesia: As with all antipsychotic agents, persistent and sometimes irreversible tardive dyskinesia may appear in some patients on long-term therapy or may occur after discontinuation of drug. The risk seems greater in elderly patients, especially females, on high dosages. The syndrome is characterized by rhythmical involuntary movements of tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and may be accompanied by involuntary movements of extremities. There is no known effective therapy for tardive dyskinesia; usually the symptoms are not alleviated by antiparkinsonism agents. If the symptoms appear, discontinuation of all antipsychotic agents is suggested. The syndrome may be masked if treatment is reinstituted, or drug dosage increased, or a different antipsychotic agent used. Reports are that fine vermicular movements of the tongue may be an early sign of the syndrome which may not develop if medication is stopped at that time.

Phenothiazine derivatives have been known to cause restlessness, excitement, or bizarre dreams; reactivation or aggravation of psychotic processes may be encountered. If drowsiness or lethargy occur, the dosage may need to be reduced. Dosages, far in excess of the recommended amounts, may induce a catatonic-like state.

Autonomic Nervous System—Hypertension and fluctuations in blood pressure have been reported. Although hypotension is rarely a problem, patients with pheochromocytoma, cerebral vascular or renal insufficiency or severe cardiac reserve deficiency such as mitral insufficiency appear to be particularly prone to this reaction and should be observed carefully. Supportive measures including intravenous vasopressor drugs should be instituted immediately should severe hypotension occur; Levarterenol Bitartrate Injection is the most suitable drug; *epinephrine should not be used* since phenothiazine derivatives have been found to reverse its action. Nausea, loss of appetite, salivation, polyuria, perspiration, dry mouth, headache and constipation may occur. Reducing or temporarily discontinuing the dosage will usually control these effects. Blurred vision, glaucoma, bladder paralysis, fecal impaction, paralytic ileus, tachycardia, or nasal congestion have occurred in some patients on phenothiazine derivatives.

Metabolic and Endocrine—Weight change, peripheral edema, abnormal lactation, gynecomastia, menstrual irregularities, false results on pregnancy tests, impotency in men and increased libido in women have occurred in some patients on phenothiazine therapy.

Allergic Reactions—Itching, erythema, urticaria, seborrhea, photosensitivity, eczema and exfoliative dermatitis have been reported with phenothiazines. The possibility of anaphylactoid reactions should be borne in mind.

Hematologic—Blood dyscrasias including leukopenia, agranulocytosis, thrombocytopenic or nonthrombocytopenic purpura, eosinophilia, and pancytopenia have been observed with phenothiazines. If soreness of the mouth, gums or throat or any symptoms of upper respiratory infection occur and confirmatory leukocyte count indicates cellular depression, therapy should be discontinued and other appropriate measures instituted immediately.

Hepatic—Liver damage manifested by cholestatic jaundice, particularly during the first months of therapy, may occur; treatment should be discontinued. A cephalin flocculation increase, sometimes accompanied by alterations in other liver function tests, has been reported in patients who have had no clinical evidence of liver damage.

Others—Sudden deaths have been reported in hospitalized patients on phenothiazines. Previous brain damage or seizures may be predisposing factors. High doses should be avoided in known seizure patients. Shortly before death, several patients showed flare-ups of psychotic behavior patterns. Autopsy findings have usually revealed acute fulminating pneumonia or pneumonitis, aspiration of gastric contents, or intramyocardial lesions. Although not a general feature of fluphenazine, potentiation of central nervous system depressants such as opiates, analgesics, antihistamines, barbiturates, and alcohol may occur.

Systemic lupus erythematosus-like syndrome, hypotension severe enough to cause fatal cardiac arrest, altered electrocardiographic and electroencephalographic tracings, altered cerebrospinal fluid proteins, cerebral edema, asthma, laryngeal edema, and angioneurotic edema; with long-term use, skin pigmentation and lenticular and corneal opacities have occurred with phenothiazines. Local tissue reactions occur only rarely with injections of fluphenazine decanoate.

For full prescribing information, consult package insert.

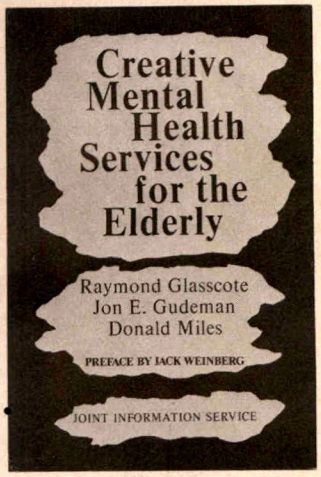
HOW SUPPLIED: 1 ml Unimatic® single-dose preassembled syringes and cartridge-needle units and 5 ml vials.

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SQUIBB® "The Priceless Ingredient of every product is the honor and integrity of its maker."™

The Joint Information Service of the American Psychiatric Association and the Mental Health Association releases Two New Volumes That

Focus on the Elderly



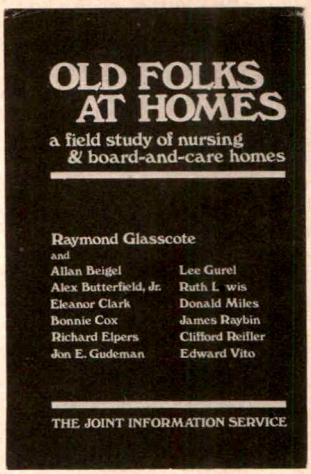
This carefully documented book reports a field study of ten programs, four in America and six in England and Scandinavia, which serve as practical demonstrations of creative mental health services for the elderly. They include:

- a novel "Neighborhood Family" providing vigorous support to elderly residents of several trailer parks
- a high school for the elderly which sends its students abroad for study trips
- a "Human Development Project" that focuses on responding to the psychological needs of the elderly
- a carefully coordinated system of "respite hospitalization," which promotes the health of the elderly while allowing maximum use of hospital beds and family resources
- a "Lucy Booth," patterned after the Peanuts comic strip, which provides easy access to many kinds of services
- and many other innovative, successful approaches.

Each program is subjected to intensive scrutiny. The resulting document becomes a handbook, and, as well, a forum through which some of the world's most experienced practitioners of "the psychiatry of old age" present their views.

Dr. Wilma Donohue, director of the International Center for Social Gerontology, characterizes this volume as "a masterful job of putting everything in relief . . . the guidelines for action are right here."

190 pages. Casebound. Price $8.50.



Concerned about the very bad reputation that nursing homes have received from many quarters in recent years, the Joint Information Service set up a field study to visit a systematically chosen sample of nursing homes, and board-and-care homes as well, to see to what extent the care of and the quality of life for the patients differed from the geriatrics units of state hospitals. Altogether, sixty nursing homes and 31 board-and-care homes in ten locales were visited, in each case by the senior author accompanied by two mental health professionals.

The teams were surpised to learn that care and quality of life in many of the nursing and board-and-care homes they visited were better—sometimes dramatically better—than that in the mental health facilities and often at lower cost.

This unique publication provides an overview of all facilities visited plus vignettes of approximately half. Federal and state rules and regulations are also expertly synthesized into brief and readable style.

148 pages. Casebound. Price $6.50

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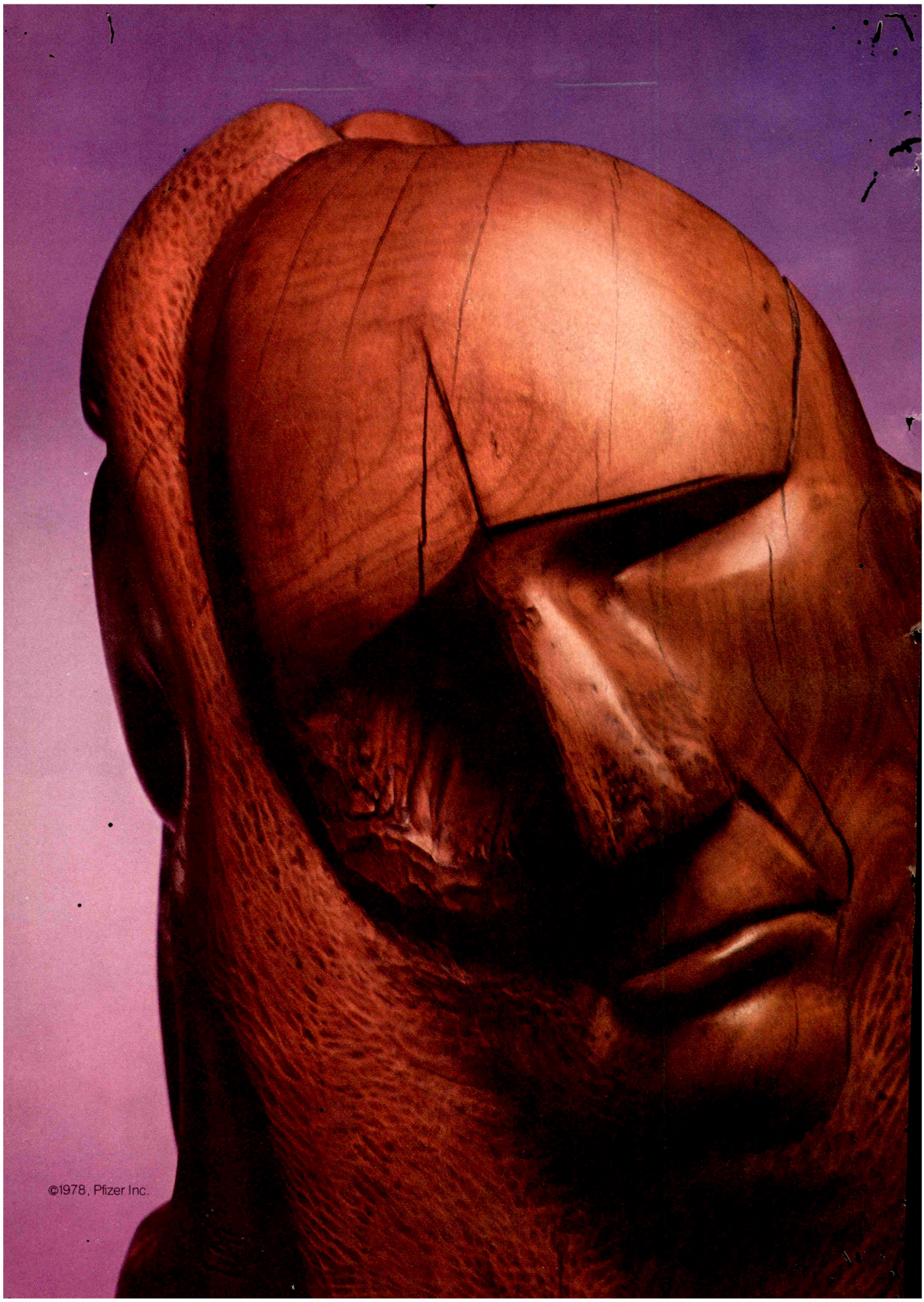
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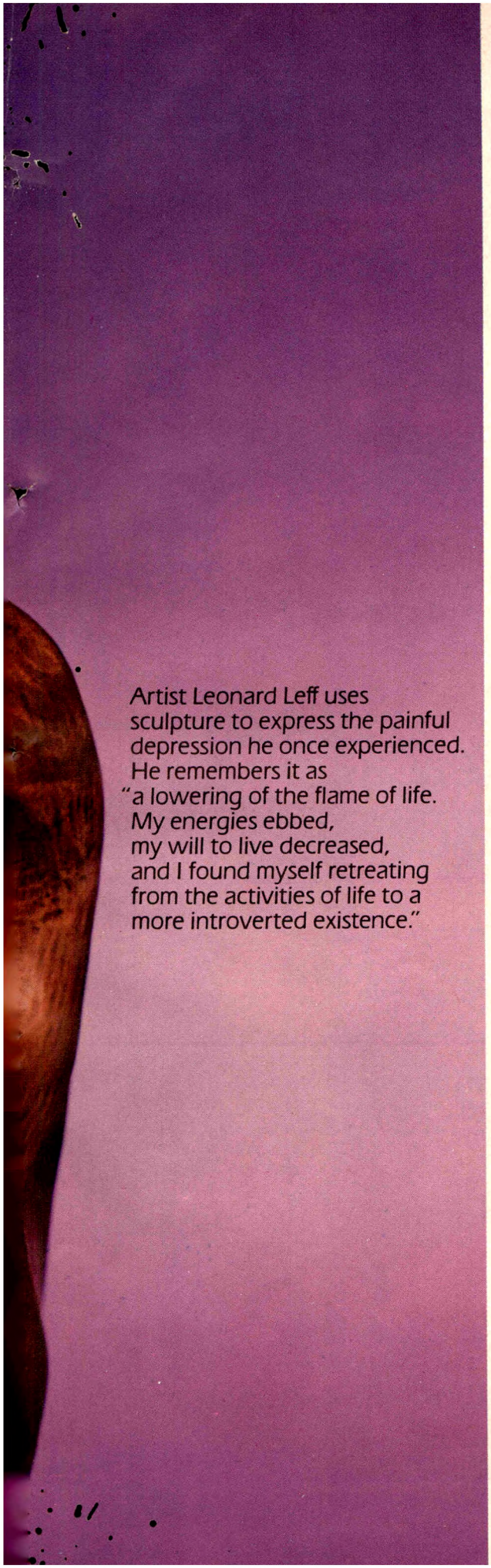
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AJP


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Artist Leonard Leff uses sculpture to express the painful depression he once experienced. He remembers it as "a lowering of the flame of life. My energies ebbed, my will to live decreased, and I found myself retreating from the activities of life to a more introverted existence."

WHEN DEPRESSION EXPRESSES ITSELF

SINEQUAN®
(DOXEPIN HCl)

ANTIDEPRESSANT EFFECTIVENESS with convenient once-a-day *h.s.* dosage*

150-MG CAPSULE†

Also available in:
100-mg, 75-mg, 50-mg, 25-mg, 10-mg CAPSULES and ORAL CONCENTRATE, 10 mg/ml, in 120-ml (4-oz) bottles

*The total daily dosage of Sinequan, up to 150 mg, may be administered on a once-a-day schedule without loss of effectiveness.

† The 150-mg capsule strength is intended for maintenance therapy only and is not recommended for initiation of treatment.

See Brief Summary on next page for information on contraindications, warnings, precautions and adverse reactions.

ANTIDEPRESSANT EFFECTIVENESS

SINEQUAN®
(DOXEPIN HCl)

150-mg CAPSULE*

CONVENIENT ONCE-A-DAY *h.s.* DOSAGE

which may improve patient compliance. The total daily dosage, up to 150 mg per day, may be given on a once-a-day schedule without loss of effectiveness. Sinequan may also be given on a divided dosage schedule, up to 300 mg per day.

PROMINENT SEDATIVE EFFECT

which may help to relieve the difficulty in falling and staying asleep, and the early-morning awakening often associated with depression.

ESTABLISHED ANTIANXIETY ACTIVITY

to help alleviate the anxiety which often accompanies clinical depression.

USUALLY WELL TOLERATED

At doses up to 150 mg per day, Sinequan does not generally affect the antihypertensive activity of guanethidine and related compounds. Tachycardia and hypotension have been reported occasionally. Drowsiness is the most commonly observed side effect. Dry mouth, blurred vision, constipation and urinary retention have been reported.

EXTENDED RANGE OF DOSAGE STRENGTHS

for flexibility in individualizing therapy.

*The 150-mg capsule strength is intended for maintenance therapy only and is not recommended for initiation of treatment.

BRIEF SUMMARY
SINEQUAN® (doxepin HCl) Capsules/Oral Concentrate

Contraindications. Contraindicated in individuals who have shown hypersensitivity to the drug, and in patients with glaucoma or a tendency to urinary retention. These disorders should be ruled out, particularly in older patients. Possibility of cross sensitivity with other dibenzoxepines should be kept in mind.

Warnings. The once-a-day dosage regimen of SINEQUAN (doxepin HCl) in patients with intercurrent illness or patients taking other medications should be carefully adjusted. This is especially important in patients receiving other medications with anticholinergic effects.

Usage in Geriatrics: The use of SINEQUAN on a once-a-day dosage regimen in geriatric patients should be adjusted carefully based on the patient's condition.

Usage in Pregnancy: Reproduction studies performed in animals have shown no evidence of harm to the animal fetus. Since there is no experience in pregnant women receiving this drug, safety in pregnancy has not been established. There are no data with respect to the secretion of the drug in human milk and its effect on the nursing infant.

Usage in Children: Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

MAO Inhibitors: Serious side effects and even death have been reported following the concomitant use of certain drugs with MAO inhibitors. Therefore, MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with this drug. The exact length of time may vary and is dependent upon the particular MAO inhibitor being used, the length of time it has been administered and the dosage involved.

Usage with Alcohol: It should be borne in mind that alcohol ingestion may increase the danger inherent in any intentional or unintentional SINEQUAN overdosage. This is especially important in patients who may use alcohol excessively.

Precautions. Since drowsiness may occur with the use of this drug, patients should be warned of that possibility and cautioned against driving a car or operating dangerous machinery while taking this drug.

Patients should also be cautioned that their response to alcohol may be potentiated.

Since suicide is an inherent risk in any depressed patient, and may remain so until significant improvement has occurred, patients should be closely supervised during the early course of therapy. Prescriptions should be written for the smallest feasible amount.

Should increased symptoms of psychosis or shift to manic symptomatology occur, it may be necessary to reduce dosage or add a major tranquilizer to the dosage regimen.

Adverse Reactions. NOTE: Some of the adverse reactions noted below have not been specifically reported with SINEQUAN use. However, due to the close pharmacological similarities among the tricyclics, the reactions should be considered when prescribing SINEQUAN.

Anticholinergic Effects: Dry mouth, blurred vision, constipation, and urinary retention have been reported. If they do not subside with continued therapy, or become severe, it may be necessary to reduce the dosage.

Central Nervous System Effects: Drowsiness is the most commonly noticed side effect. This tends to disappear as therapy is continued. Other infrequently reported CNS side effects are confusion, disorientation, hallucinations, numbness, paresthesias, ataxia, and extrapyramidal symptoms and seizures.

Cardiovascular: Cardiovascular effects including hypotension and tachycardia have been reported occasionally.

Allergic: Skin rash, edema, photosensitization, and pruritus have occasionally occurred.

Hematologic: Eosinophilia has been reported in a few patients. There have been occasional reports of bone marrow depression manifesting as agranulocytosis, leukopenia, thrombocytopenia, and purpura.

Gastrointestinal: Nausea, vomiting, indigestion, taste disturbances, diarrhea, anorexia, and aphthous stomatitis have been reported. (See anticholinergic effects.)

Endocrine: Raised or lowered libido, testicular swelling, gynecomastia in males, enlargement of breasts and galactorrhea in the female, raising or lowering of blood sugar levels have been reported with tricyclic administration.

Other: Dizziness, tinnitus, weight gain, sweating, chills, fatigue, weakness, flushing, jaundice, alopecia, and headache have been occasionally observed as adverse effects.

Dosage and Administration. For most patients with illness of mild to moderate severity, a starting daily dose of 75 mg is recommended. Dosage may subsequently be increased or decreased at appropriate intervals and according to individual response. The usual optimum dose range is 75 mg/day to 150 mg/day.

In more severely ill patients higher doses may be required with subsequent gradual increase to 300 mg/day if necessary. Additional therapeutic effect is rarely to be obtained by exceeding a dose of 300 mg/day.

In patients with very mild symptomatology or emotional symptoms accompanying organic disease, lower doses may suffice. Some of these patients have been controlled on doses as low as 25-50 mg/day.

The total daily dosage of SINEQUAN (doxepin HCl) may be given on a divided or once-a-day dosage schedule. If the once-a-day schedule is employed the maximum recommended dose is 150 mg/day. This dose may be given at bedtime. **The 150 mg capsule strength is intended for maintenance therapy only and is not recommended for initiation of treatment.**

Antianxiety effect is apparent before the antidepressant effect. Optimal antidepressant effect may not be evident for two to three weeks.

Overdosage.

A. Signs and Symptoms

1. Mild: Drowsiness, stupor, blurred vision, excessive dryness of mouth.
2. Severe: Respiratory depression, hypotension, coma, convulsions, cardiac arrhythmias and tachycardias.

Also: urinary retention (bladder atony), decreased gastrointestinal motility (paralytic ileus), hyperthermia (or hypothermia), hypertension, dilated pupils, hyperactive reflexes.

B. Management and Treatment

1. Mild: Observation and supportive therapy is all that is usually necessary.
2. Severe: Medical management of severe SINEQUAN overdosage consists of aggressive supportive therapy. If the patient is conscious, gastric lavage, with appropriate precautions to prevent pulmonary aspiration, should be performed even though SINEQUAN is rapidly absorbed. The use of activated charcoal has been recommended, as has been continuous gastric lavage with saline for 24 hours or more. An adequate airway should be established in comatose patients and assisted ventilation used if necessary. EKG monitoring may be required for several days, since relapse after apparent recovery has been reported. Arrhythmias should be treated with the appropriate antiarrhythmic agent. It has been reported that many of the cardiovascular and CNS symptoms of tricyclic antidepressant poisoning in adults may be reversed by the slow intravenous administration of 1 mg to 3 mg of physostigmine salicylate. Because physostigmine is rapidly metabolized, the dosage should be repeated as required. Convulsions may respond to standard anticonvulsant therapy; however, barbiturates may potentiate any respiratory depression. Dialysis and forced diuresis generally are not of value in the management of overdosage due to high tissue and protein binding of SINEQUAN.

Supply. SINEQUAN is available as capsules containing doxepin HCl equivalent to: 10 mg, 75 mg, and 100 mg doxepin: bottles of 100, 1000, and unit-dose packages of 100 (10 x 10's). 25 mg and 50 mg doxepin: bottles of 100, 1000, 5000, and unit-dose packages of 100 (10 x 10's). 150 mg doxepin: bottles of 50, 500, and unit-dose packages of 100 (10 x 10's). SINEQUAN Oral Concentrate (10 mg/ml) is available in 120 ml bottles with an accompanying dropper calibrated at 5 mg, 10 mg, 15 mg, 20 mg, and 25 mg. Each ml contains doxepin HCl equivalent to 10 mg doxepin. Just prior to administration, SINEQUAN Oral Concentrate should be diluted with approximately 120 ml of water, whole or skimmed milk, or orange, grapefruit, tomato, prune or pineapple juice. SINEQUAN Oral Concentrate is not physically compatible with a number of carbonated beverages. For those patients requiring antidepressant therapy who are on methadone maintenance, SINEQUAN Oral Concentrate and methadone syrup can be mixed together with Gatorade®, lemonade, orange juice, sugar water, Tang®, or water; but not with grape juice. Preparation and storage of bulk dilutions is not recommended.

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Psychotherapy of the Medically Ill

BY TOKSOZ B. KARASU, M.D.

The author assesses the current status of psychotherapy with medically ill patients. He reviews the special problems of psychological treatment for this population; the respective rationales for the utilization of various psychotherapeutic approaches including psychoanalysis, dynamic psychotherapy, and family and group therapy; and the results of case reports and research studies. The author identifies specific areas of future investigation and research, elucidates some specific implications for clinical practice, and recommends more critical exploration of the role of psychotherapy in the treatment of the medically ill.

EXTENSIVE STUDIES of psychodynamic and psychological responses to life stress formulations continue to contribute to an increasing theoretical literature with important implications for psychological intervention; yet comparable strides have not been made in the efficacy of psychotherapy as a treatment modality for medical illness. It is generally believed by medical and psychiatric clinicians that psychosomatic patients are especially poor candidates for psychotherapeutic interventions, especially psychoanalysis (1–3). Wolff (1) considered three-quarters of his psychosomatic patients unsuitable for intensive, interpretive psychotherapy; Sifneos (2) observed that such patients actually get worse from the psychodynamic process. Sperling (3) has contended that whether one uses supportive therapy or psychoanalysis in the treatment of psychosomatic disorders, either approach is uneconomic: supportive therapy because the patient's dependency is never resolved and psychoanalysis because of the specialized skill required and the scarcity of qualified analysts who are properly prepared to treat such cases. These researchers and others have therefore suggested modifications of classical psychoanalytic techniques that may be applied when more orthodox methods have met with excessive resistance or failure (1–7).

Studies of the effects of psychotherapy for the medically ill have been relatively minimal and inconsistently controlled, and when globally evaluated for therapeutic results (physical and/or psychological), they have been equivocal in overall outcome (8–10). However, in one historical review of the effects of psychotherapy (11), the highly variable and null results were seen as embedded in the "outcome problem" of psychotherapeutic research design, that is, the typical misleading tendency to include assessments of psychological treatment at termination only. In reexamination of controlled follow-up studies of six months or more across a full spectrum of psychotherapeutic modalities (psychoanalysis, individual dynamic psychotherapy, and behavioral and group approaches) used with three psychosomatic conditions (ulcerative colitis, peptic ulcer, and asthma), *all* gave positive results. Strong evidence indicated that whether a psychodynamic approach per se is necessary and specific, it can be effective. Moreover, the almost bizarre conclusion was reached that dynamic therapies were validated as effective for psychosomatic conditions—and for no other diagnoses whatsoever, including the psychoneuroses. This review is intended to bring up to date the various findings that have been reported in this area and to critically assess the current state of the art of psychotherapeutic intervention for the medically ill.

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THE SPECIFIC NATURE OF THE PROBLEM

Obstacles to psychiatric treatment include the complex nature of psychosomatic forms of illness that re-

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side on an intricate continuum of psyche. They include somatic influences and manifestations (12); the presumed personality characteristics, attitudes, and defense mechanisms of the patients that have direct implications for motivation for psychotherapy and conduct during treatment, e.g., profound resistance, denial of psychic conflicts (1–4, 6, 13–19); and, all too often, countertransference reactions of internists and psychiatrists themselves (1, 19, 20)—all of which converge on the treatment situation to make the practice of psychotherapy for psychosomatic illnesses a formidable, if not impossible, undertaking.

Major issues for psychological intervention include whether the patient with a psychosomatic disorder can sustain sufficient motivation for undertaking and maintaining treatment, that is, establishing a productive doctor-patient relationship, and, once in treatment, whether he or she can comprehend and withstand attending to the psychological side of the illness. In these regards it has been noted that persons with psychosomatic disorders characteristically set up the most rigid and archaic defense mechanisms of denial and reaction formation against the experience of affect (3) and are unable to experience and express negative feelings, particularly aggression (6). The irony of these mechanisms for psychological intervention per se is represented in the observation that the psychosomatic patient needs to use and attend to his or her physical symptoms for the very purpose of warding off psychological insights (16); indeed, a psychosomatic illness is said to develop when a patient is unable to acknowledge and tolerate an emotional conflict and its accompanying instinctual impulses (1). Moreover, while psychosomatic events typically declare themselves in connection with situations of stress arising from within the individual or his or her immediate environment, frequently the psychosomatic sufferer is not aware of such connections or of being under any particular stress (18).

In accordance with these observations, a psychosomatic character pattern has been elucidated. It encompasses the following: unusual object relationships, especially denoted by a lack of libidinal affect; an impoverished use of language in which operational thinking (i.e., extremely detached and pragmatic) predominates; an inability to use regression or dream-type functioning; and a marked lack of neurotic symptoms and neurotic character adaptations, with facial movements, bodily gestures, sensory-motor expressions, and physical pain manifested when one might otherwise expect neurotic behaviors (14).

Directly related to motivation for treatment have been the observations that many psychosomatic patients consider psychological conflict as a sign of weak character, emotional problems as synonymous with malingering; that revealing one's dependency is typically considered shameful (5); and that the ulcer patient would finally agree to psychiatric treatment not because of a recognition of his or her own needs, but only when he or she was convinced that he or she was doing it for someone else, i.e., helping the doctor (21). Others have confirmed that the patients with the most severe and long-standing illnesses were those most unable to tolerate having their denial shaken and were most prone to terminate treatment prematurely (7, 16). That is, when these patients begin to see that the physical symptoms of others are related to life difficulties and when they are forced to realize that their *own* physical problem may have the same basis, they do not return to therapy.

The manifestations of these features during the psychotherapy process have included the observations that preliminary interviews with psychosomatic patients were pervaded by a type of inertia that threatened to bring discussion to an end; that the investigator had to make vigorous efforts to stimulate associative material concerning the patients' relationships, life experiences, and illness; that significant or painful events were overlooked unless directly solicited and were recounted with minimal emotional expression (14); and that even when the patients' symptoms inflict physical pain, they fail to receive much attention during the therapeutic encounter (18). This was characterized both by a physiological hardiness or attitude of blithe disregard for one's own physical welfare as though the body were a decathected object and a psychological hardiness or refusal to give in to psychic pain, anguish, or depression.

Psychosomatic patients generate an impression of superhuman emotional control, behind which appears to reside the necessity to refuse to reveal one's needs, dependency, disappointment, anger, despair, or any incapacity or failure. Weisman (22) attributed this to the ego ideal of these patients, which is "inaccessibly high"; and Castelnuovo-Tedesco (23) noted, especially in ulcerative colitis patients, a fundamental "compliance-versus-rebellion" conflict that is resistive to the treatment situation. In the analytic situation aloofness, detachment, testiness, irritability, and contentious demandingness regularly came to the fore in situations that signify being humiliated, coerced, or otherwise used or violated. More globally, it is said that most psychosomatic patients in psychological treatment possess "a negative attitudinal set" (6).

Mushatt (13) found that ulcerative colitis patients were unable to free associate and could not tolerate therapist silences. Sifneos (2) observed that psychosomatic patients had little to talk about and were unable to explain to the therapist why they found the transference situation so discomforting. Stevens (24) found that the transference and interpretation in psychotherapy, especially in psychoanalysis, mobilized enormous anxiety in these patients, which they found intolerable. Struck by psychosomatic patients' marked unamenability to classical dynamic psychotherapy, Sifneos significantly validated the prevalence among these patients of a group of characteristics that would resist and even be antithetical to the analytic process. They include poverty of fantasy life, constriction of emotional functioning, inability to find appropriate

words to verbalize feelings, and absence of an ability and motivation for self-examination. Sifneos found that 25 psychosomatic patients had twice as many of these so-called "alexithymic" characteristics as did 25 control subjects (17).

The fragile egos of these patients, as well as the particular nature of their defensive structure, has another significant implication for psychological treatment. Just as the removal of the latent psychotic patient's neurotic defenses can produce an acute psychotic episode, the analysis of the psychosomatic patient's strong denial and reaction formations can bring about not an amelioration, but an aggravation, of the patient's somatic condition (3). It is also reportedly feared that analytical psychotherapy of patients with psychosomatic illness is likely to precipitate a psychotic breakdown; however, according to Wolff (1), this has never occurred in his experience.

Aside from the psychosomatic risks and resistances of the patients are the reactions and counterreactions of internists and psychiatrists themselves to the psychological treatment of psychosomatic disorders. Wolff (1) has pointed out the difficulty of overcoming medical practitioners' reluctance to refer the patient to a psychotherapist, especially if the medical regimen has met with some success, and the tendency to regard psychotherapy as the last line of medicine for any disorder that appears to be a physical illness. Even when the medical doctor's resistance has been overcome, psychotherapists themselves may be disenchanted with the prospect of treating persons with apparently organic, often life-threatening, illnesses. Craddock (20) has referred to the limited psychiatric experience with and knowledge of these diseases, which discourage attempts at psychological treatment. Fain and Marty (19) have noted the risk to the analyst, who may feel that such patients constitute a chronic narcissistic blow to his or her interpretive powers and who may react with a countertransferential lack of interest. Moreover, the fear that too early or too direct a confrontation may result in the very early termination of treatment, intense negative transference, the possible precipitation of a psychotic breakdown, or, more likely, an acute exacerbation of the very illness that is being treated, even the risk of death, becomes a matter of particular delicacy and concern for the prospective psychotherapist of the medically ill patient.

PSYCHOANALYSIS AND INDIVIDUAL PSYCHOTHERAPY

Rationale of Treatment

Theories about the etiology of psychosomatic disorders and their manifestations in the patient naturally have implications for what one views as the treatment of choice. Long-term psychotherapy, especially psychoanalysis, as an essential aspect of treatment largely develops out of models of psychosomatic illness that have specific psychogenic factors (e.g., psychological defenses, predispositions, precipitating psychosocial situations, early life experiences, specific affects), and especially psychological conflict (e.g., dependency), as their pivot point in the etiology of disease-specific physiological patterns (3, 25–27). In addition, the possible role of infantile psychophysiological patterns of function has highlighted the need to address crucial developmental phases in psychotherapy (28, 29). The roles of object loss and reactive response to illness (especially the affects of helplessness and hopelessness), separation, symbolic functions, and conversion mechanisms in the impact of illness on body image have reflected the need to deal with reactive issues, depression, and object relations in treatment (30–32).

Clinical Cases and Research Studies

Much of the psychoanalytic groundwork for psychosomatic disorders exists in the form of detailed clinical case studies (3, 23, 33–35). Sperling (3) reported the successful treatment of several children with severe psychosomatic disorders. In the course of child play analysis for a 4-year-old with ulcerative colitis, the child's complete submission to his mother was radically altered. When the first manifest displays of his aggression appeared, they were accompanied by an immediate and striking improvement not only in the illness but in the child's total behavior (3). A 10-year-old asthmatic boy remained asthma-free for the 2 years during his analysis plus 9 years subsequent to it (33). There were comparable results with an 8-year-old boy with asthma after just 7 months of treatment (34). However, along with these highly encouraging outcomes were certain dangers: the development of a very strong transference neurosis, which culminated in a severe asthmatic attack; soiling and nightmares correspondent with improvement; and more frequent asthmatic attacks in a family member, who became more difficult in general. These authors emphasized the importance of early treatment for maximal therapeutic success.

The treatment of adults has been less consistent in outcome. In striking concordance with efforts to make a colitis/gout patient aware of the destructive implications of his marked dependency, his attacks became increasingly frequent, resulting in at least 25 major exacerbations during the 3 years of psychiatric treatment. The patient's colitis had occurred primarily in response to coercion by a significant person, whereas the gout attacks were precipitated by real or threatened separations or when issues of independence were uppermost (35). Stein and associates (36), who studied the changes in hydrochloric acid secretion in a patient with a gastric fistula during 2½ years of intensive psychotherapy, found that 8 months after the psychotherapy was begun, the high normal level of the gastric hydrochloric acid dropped abruptly and remained at a low level for 12 months. This striking change occurred when the patient became aware of and began to express material related to her oral-aggressive drives.

In conclusion, individual case reports of success-

ful psychoanalysis for patients with psychosomatic disorders, especially children, have been clinically impressive but subjectively anecdotal and impressionistic. In addition it is ironic that the focus on the relationship between course of treatment and exacerbation of illness makes it appear that exacerbations are often triggered by the very process of "cure." Finally, however dynamically relevant and therapeutically positive such studies are, they leave unanswered the question of evidence by omission. As Freeman and associates (37) noted, "One must wonder how many failures go unreported" (p. 599). However tantalizing the above findings, the need for data from larger samples is becoming increasingly apparent.

A larger, more systematic study of dynamic/therapeutic relationships was conducted by Weisman (22), who found that six men treated with psychoanalysis had fewer recurrences of symptoms than did untreated individuals. No single specific variable could account for the frequency and duration of exacerbations, or length of symptom-free intervals. However, in one case the exacerbation of ulcer symptoms occurred with the threat of depletion of the therapist's "love"; the controlled release of the patient's restrained anger was considered an important aspect of treatment.

Weinstock (38) reported the results of the first national questionnaire survey of psychoanalytic treatment by experienced analysts in 25 cases of severe ulcerative colitis, in which previously hospitalized patients were treated for long periods (1½ to 5 years). Approximately 80% of them were symptom-free for 3 to 18 years, with a median "cure" period of 9 years. The 20% with poorer results were primarily those with severe, unremitting symptoms for an average period of 9 years before treatment. The two modalities used (psychoanalysis and psychoanalytically oriented psychotherapy) were not significantly different in overall effect, and the analysts concluded that psychiatric treatment per se had been the decisive factor in maintaining the patients' symptom-free status.

Perhaps the most extensive and revealing series of matched controlled studies represent three decades of observation and psychotherapy of ulcerative colitis patients (39–43). In the initial study, 5 completely analyzed patients showed greater psychological improvement than those unanalyzed, suggesting that even a few well-timed and insightful sessions can serve to release impounded anger and lead to better life adjustment (39). In a much more intensive study, 57 patients who had received psychotherapy in addition to the usual medical regimen had more favorable somatic and psychological responses than did a matched control group. Of the total group of patients, 75% improved psychologically and 66% somatically. In most cases there was a simultaneous increase or decrease in both psychological and somatic responses, a concomitance rather than alternation of symptoms, i.e., overt signs of emotional disorder appeared most frequently during exacerbations rather than remissions of the somatic symptoms (16). The length of treatment also had interesting effects on symptoms. When proctoscopic and symptomatic ratings were recorded over an 8-year period, a definite pattern emerged. In the first year of psychotherapy the treated patients rated consistently lower than the control group; in the second and third years there was sustained improvement; and in the fourth to eighth years there was marked improvement (the control group remained essentially the same throughout the 8 years) (40).

Later studies compared 30 colitis patients with controls on pretreatment factors (41) and on a variety of "psychotherapeutic situation" factors (42). The following four pretreatment factors had clear implications for poor psychotherapeutic prognosis: severe psychopathology, significant symbiotic dependency, little ego strength, and a significant drive for secondary gain from the illness. Within the psychotherapeutic situation per se, the most lasting somatic improvement derived from a combination of high patient hope and high therapist interest, empathy, and optimism. In terms of specific technique, interpretation proved most valuable only with active-individuated patients in whom symbiosis was a minor issue; passive-symbiotic types did most poorly with interpretation. Psychoanalysis was precluded during attacks of colitis in any patient. Briefer and less intensive uncovering techniques (44–46) were able to bring about significant somatic improvement in most patients, but the longer therapies were almost twice as effective in promoting and maintaining remissions of the disease (42).

In conclusion, although the difficulties (3, 33, 35) as well as the successes (3, 22, 33, 34, 36, 38–40) of analytic approaches and insight-oriented psychodynamic therapies have been amply reported, ultimately only carefully controlled research studies that take into account such significant variables as length of treatment (40), pretreatment patient factors (41), and therapist qualities and specific techniques (42) will determine their feasibility and effectiveness and the degree to which therapeutic alternatives (e.g., supportive approaches) are needed.

FAMILY THERAPY

Rationale of Treatment

Insofar as the fundamental dynamics of the mother-child relationship have been regarded as crucial to the psychosomatic pattern of response, it is felt that severe psychosomatic disorders may be reversible if it is possible to directly change the relationship (i.e., "mutual symbiosis") existing between mother and child (3, 33). In young children, especially, this goal can be particularly well accomplished in the concomitant treatment of mother and child, "where a modification of the unconscious need of the mother [can] manifest itself in a change in the somatic response of the child" (3, p. 281). More generally, the basic thesis behind most family treatment is that the child with a psycho-

somatic illness is the "symptom bearer" of the family and that the family system must be changed in order to enable the child to alter his or her special role in it (47–53). It is also believed that traditional, one-to-one, dynamically oriented approaches may fail with the chronic patient because they intensify his role as a sick member of the family while neglecting the roles of parents and siblings in perpetuating the illness. In brief, the symptoms are said to play an important role in the defensive structure of the family; they are hypothesized to be the consequence of a conflict-detouring process in which the parents' total concentration on, and infantilization of the child enables them to detour and avoid dealing with their own marital conflicts. Thus the psychosomatic disorder is seen as situated in the feedback processes of child and family, in which the family's feedback to the child's symptoms becomes an autonomous process that maintains the symptom.

Certain types of family organization or characteristics are said to be crucial to the development and maintenance of psychosomatic symptoms, which can be best addressed in family therapy. These characteristics include "enmeshment" (high degree of involvement with other members of the family), "overprotectiveness" (high degree of concern with each other's welfare), and "rigidity" (detouring conflict onto the child such that when the family's low threshhold of conflict is approached, the child becomes ill). The goals of family treatment are to identify and change family patterns that perpetuate the psychosomatic symptoms (47–54). In the conjoint treatment of patients and spouses, the rationales for family treatment include providing a more direct outlet for repressed rage (55) and helping to break the denial of the psychosomatic patient with the presence of another family member (4).

Research Studies

In her pioneering studies Sperling (3, 33) conducted psychoanalytic treatment of asthmatic children with their mothers either concomitantly or intermittently, and of the mother only when the child was the psychosomatic sufferer. In both types of treatment there was a remarkable improvement in the asthma of the child. This was attributed to the mother's having gained increasing insight into the ways in which she signaled her needs for the child to be sick and her increased ability to control them. In a controlled intensive study of 10 families of eczematous and asthmatic children, half of whom had attended a therapeutic nursery school, the eczematous symptoms markedly diminished (54). During the psychotherapy of the family the dynamics of eczematous exacerbations were revealed and changed; the hitherto covert conflict of the parents came to the fore; they began to verbalize quite openly their deep disappointment, frustration, and anger with each other; and the child began to be less infantilized and more assertive. However, it should be noted that asthmatic attacks appeared at this time. (The authors attributed the attacks to the disruption of the previous intrafamilial equilibrium.)

In an elaborate series of studies Liebman, Minuchin, Rosman, and associates (48–53) reported the use of structural family therapy in the treatment of intractable asthma, infantile diabetes, and anorexia nervosa. In the study of asthma (51) 7 patients were treated with weekly family therapy conducted in three phases with successive goals: alleviation of symptoms (through deep-breathing exercises with the father), identification and modification of family patterns that exacerbated and perpetuated severe symptoms, and the lasting disengagement of the patient from the family. Family therapy was considered successful in all cases. Six subjects did not experience acute attacks of asthma requiring hospitalization; all 7 were no longer dependent on chronic steroid treatment; they had more normal lifestyles in school and with their peers; they engaged in more physical activity; and there was a positive change in the interpersonal relationship of siblings and parents.

In the treatment of anorexia nervosa the authors developed a "family lunch session" as the focal point around which the family therapy was pivoted (50, 52). The lunch sessions had the major purpose of enabling the patient to eat in the presence of her parents without a power struggle, and the parents were given specific tasks that served to separate the patient from their conflicts. A research study of the effectiveness of this program showed a significant weight gain in all 8 of the anorectic children studied (52). Aponte and Hoffman (53) found that once the parents of an anorectic girl established a better relationship with each other, they could effect weight gain in their child for the first time. The girl gained 14.5 kg in 5 weeks, and 6 months later continued to be in good physical shape, had a boyfriend, and was leading a more independent life. Barcai (47), in two case reports, noted that each patient showed a continuous weekly weight gain of .9–1.4 kg from the time of the first therapeutic contact until normal weight was attained within 3 to 4 months. One, in fact, responded with disappearance of the symptom after a 2-hour family session. These results are in accord with earlier findings by Minuchin and Barcai (49).

Other studies of family treatment have involved the conjoint family therapy of husband and wife in the case of patients who somatize, especially those with chronic migraine headaches (4, 55). As a result of the conjoint family treatment of migraines, symptom relief was successfully obtained (although the patient's basic fear of aggressiveness remained unresolved) (55). In addition, although the conjoint family method did result in the prompt alleviation of symptoms, the therapist did not attribute the outcome to the use of this modality per se. Rather, he concluded, "this result appeared similar to that obtained by a new drug or new therapist and was probably attributable to providing a new sense of hope and security" (55, p. 63).

With regard to motivation, Raft and associates (4)

studied 20 patients for whom symptoms could be directly related to psychological conflict, but who upon individual interview refused to accept the relationship. Seventeen of the 20 patients, when interviewed individually, did not believe that psychiatric intervention was needed. As a result of a family interview, however, 80% of them accepted psychiatric recommendations and referral.

The above studies may be said to suffer the same pitfalls of other psychotherapy outcome studies (11). However, the application and utilization of family therapy techniques for psychosomatic illnesses have been lent substantial support by the study data and outcomes. The advantages of the family therapy technique have in particular included the prospects of a high acceptance rate (4), the freeing of the child and his or her family from the fear for the child's life, the immediate establishment of areas of child autonomy and parental control, the giving of hope and positive expectations to the family (52), and, ultimately, the reduction of symptoms (3, 33, 47, 50–54).

GROUP THERAPY

Rationale of Treatment

Group psychotherapy, like family therapy, has been advanced in order to address some of the presumed limitations of individual therapy for patients with psychosomatic disorders. Since a major concern is the special difficulty in entering a therapeutic relationship, two inherent changes in the manner in which the transference is manifested in group therapy in contrast to dyadic treatment are claimed to make it an especially suitable modality. The transference is split and its intensity is lessened due to the presence of several persons, including peers, with whom the patient interacts. In addition, the group format inherently offers the following distinct advantages to these patients: increased stimuli for participation and interaction, which can facilitate their involvement in the therapeutic process and the development of relationships; sharing of guilt, which allows their excessively rigid and archaic superego attitudes to be eased; and accessibility to others' intense feelings, which increases awareness of their own. The group format also offers greater interpersonal contact, which provides increased ego support for their typically weak egos and lessens isolation and the threat of separation; and, most critically, offers a greater opportunity to express emotions in an accepting peer atmosphere, which allows the displaced affect of these patients to be channeled verbally and directly, rather than only through somatic and autonomic avenues (56). Others have cited the group as an excellent medium for making psychosomatic patients "affect-conscious," that is, stirring up affects and emotions (6); the educational value of raising and answering critical questions, especially the increased transmission of information patients were unable to approach with their medical doctors (57–59); and the implicit gains of identification with persons having similar somatic experiences and attitudes (56), in particular, long-term survivors of illness (58).

Research Studies

A review of the literature reveals that the most consistent recipients of group psychotherapy include patients with asthma (probably the most generally investigated psychosomatic illness, with many forms of therapy) (52, 60–62), postmyocardial infarction (7, 58, 59, 63, 64), obesity (65–67), and chronic pain (6). Patients with these disorders have primarily been placed in homogeneous groups based on commonality of the illness entity or symptoms. Less typical formats have been the (late 1940s) spontaneous formation of heterogeneous psychosomatic patient groups comprised of members with different classical illnesses (56), and, as Wolff (68) noted, the heterogeneous combination of medical and nonmedical patients.

More than 25 years of clinical experience (1945–1970) with group psychotherapy for psychosomatic disorders have elicited a report of consistently impressive results (56). Sixty-six percent to 100% of the members improved; this included groups of chronic menopausal, ileostomy, and ulcer patients, including those with histories of unsuccessful individual treatment and severe underlying psychiatric disorders. The ileostomy and ulcer groups had the most successful outcomes and had maintained their improvement upon follow-up several years later. In addition, the all-female groups fared better than the groups containing men and women. On the basis of such results, proponents of homogeneous groups view the sharing of similar somatic symptoms and experiences as a decided advantage for positive outcomes (56, 63); however, the inclusion of nonpsychosomatic patients is often desirable in order to deemphasize the somatic issues that tend to be overly stressed in such groups (1).

The following studies have all been of homogeneous groups. In the study of asthma, Groen and Pelser's (60) long-term, controlled, and follow-up investigations of group psychotherapy for 200 patients are perhaps the most exemplary. These researchers used three comparison groups: symptomatic treatment only, ACTH, and group psychotherapy (male and female group therapy of 4- and 2-year durations, respectively). Despite the severity of diagnoses, group therapy was quite successful, both somatically and psychologically. Group therapy patients rated significantly higher than both control groups in all somatic outcome categories, including a significantly lower mortality rate; 73% of them were clinically cured, compared with 17% of the symptomatic treatment group and 29% of the ACTH group. Statistically significant Rorschach changes and informal confirmation by spouses and group leaders validated the positive psychological changes (69). Others have successfully treated not the patient per se, but the family members of asthma patients, with group psychotherapy (61, 62), especially concerning issues of sibling rivalry and family support

of the asthmatic child. While the parents definitely benefited from the group therapy learning process in terms of their awareness and ability to verbalize roles enacted within their own family constellations, there were no outcome assessments of the asthmatic children.

The sometimes subtle vicissitudes of the issue of psychological versus somatic improvement have also emerged in a variety of other studies, including the group treatment of obesity (65–67); patients with chronic pain, especially headaches (6); and the inpatient and outpatient management of myocardial infarction patients (7, 58, 59, 63, 64). With up to 2 years of group psychotherapy in the treatment of obesity, patients improved in their general adaptation, home life, and emotional well-being, but weight losses were at best minor (65–67). In the treatment of 56 pain patients using a combination of psychoanalytically oriented methods and encounter techniques, the group was considered an excellent medium for making the patients "affect-conscious" and in helping them perceive the interaction of their life difficulties, affective experiences, and somatic symptoms. However, neither the frequency of the headaches nor the magnitude of the pain appeared to be materially affected; and an increase in symptoms was observed as the patients became increasingly aware of the nonphysical origins of their problems (6).

Among the studies that have met with the most enthusiasm have been those involving postmyocardial patients (58, 59, 63, 64). The group sessions essentially served the function of an information gathering and sharing experience regarding issues of medication, nutrition, and physical activity, as well as attitudes toward family members, sex, and death. (This finding is at variance with findings from work with asthmatic patients, where it took approximately two years before the patients began to discuss psychogenic issues; they never discussed sex or fear of death [60].) There were no exacerbations of symptoms during the group sessions, and implicit gains included identification with long-term survivors of coronary illness, which naturally increased as the group members progressed in convalescence (63). In controlled studies (7, 64) the widest acclaim was for several short-term nonsymptomatic benefits such as directly answering patients' concerns, high acceptance of and adherence to a physical fitness program, and self-reports of increased self-confidence. However, longer term benefits such as fewer cardiac symptoms, increased return to work, and lower death rates were less conclusive. Ibrahim and associates' long-term, large sample controlled study (64) found no statistically significant changes in psychological factors (e.g., autonomy or understanding) or in physiological factors (e.g., blood pressure or cholesterol levels) during the entire follow-up period of 1½ years. However, there were several physical improvements of short-term duration and evidence of reduced hospital stay and increased survival rates.

As with other psychotherapeutic formats, the actual structural or process factors responsible for group therapy's outcomes have been minimally addressed. These factors include homogeneity of group composition (56), the maintenance of a "tolerant, educative attitude" rather than an interpretive one (60), and issues of short-term versus long-term, and psychological versus somatic, outcome criteria. Other research problems remain to be refined. Nonetheless, the educative value of group psychotherapy for psychosomatic patients and their families has been confirmed throughout the literature (7, 58, 59, 61–64), and psychological as well as physical results have been highly promising. In addition, in a controlled research study (6) the feasibility of the group as a therapeutic modality in terms of increased patient motivation and length of stay was "unequivocally affirmative" as indicated by an 84% acceptance rate, 69% attendance rate over 1½ years, and follow-up of 85% of the group. This augurs well for the future use of group psychotherapy as an alternative to, or adjunct of, other therapeutic regimes in the psychosomatic treatment armamentarium.

COMPARISON SURVEYS

Most psychosomatic outcome studies have at best researched a single disorder treated by a single psychotherapeutic modality, and cumulated work with any one disorder or modality has not been systematically generalizable to others. This has left the issue of comparative findings essentially unexplored. Although to my knowledge there is no single controlled psychosomatic study to date that comprehensively compares the effects of more than one psychotherapeutic modality, the following surveys shed some light on this essential, but as yet embryonic, area (8–11, 70).

Early collective findings in the use of psychotherapy for psychosomatic disorders contrasted the effectiveness of brief flexible psychotherapy and long-term psychoanalysis for a variety of disorders (8). The overall outcomes of both forms of psychotherapy ranged from poor to good, but when the outcomes were further analyzed according to illness, suggestive patterns emerged. Asthma studies showed consistently good results with either type of treatment and hypertension studies consistently poor results. Outcomes with rheumatoid arthritis patients were poor using brief flexible therapy, but promising using psychoanalytic treatment. For patients with hyperthyroidism brief flexible psychotherapy helped, but psychoanalytic treatment did not. However tantalizing the differential findings, they were faulted by methodological and research limitations.

Chalke's similar review format (9) for the subsequent 15-year period (1950–1965) revealed that psychotherapy had by then been applied to many medical conditions other than the classical psychosomatic disorders, e.g., chronic vasomotor rhinitis, migraine headaches, neurodermatitis, and spontaneous habitual

abortion. This review led to differential findings of another order: only for these newer psychosomatic categories was psychotherapy sufficient treatment; for the classical psychosomatic illnesses psychotherapy was significantly useful; but essentially as an adjunct treatment. This was particularly evident in ulcerative colitis and asthma patients, for whom psychotherapy was especially beneficial for the secondary emotional crippling and related personality and familial problems, although it did not necessarily materially affect the *physical* component, e.g., the asthmatic attacks.

These newer studies had the scientific advantage of having been conducted under somewhat improved controlled conditions, but definitive findings were not yet possible. In particular, the criteria for rating outcome were still admittedly vague. However, by locating instances of "psychological" versus "somatic" improvement that differentially occurred in different disorders, these studies inadvertently served to place the crucial question of outcome criteria in bolder relief, e.g., are there instances when therapy for psychosomatic disorders can still be considered viable treatment although the *somatic* component remains essentially unaltered? Although it is seemingly obvious, this issue has not been sufficiently addressed within the deliberative context of research designs.

A decade later, surveys of controlled studies covered a wide spectrum of new treatment modalities (and ills) (10, 11, 70). Although Malan's uniformly positive findings across several psychological formats and disorders were the most clinically and scientifically encouraging, they precluded the emergence of any specific patterns; too few studies of long-term follow-up were available (11). However, others (10, 70) uncovered some suggestive relationships between illness and treatment technique. Goldstein and Stein (70) concluded that systematic desensitization is differentially the most effective treatment for bronchial asthma, biofeedback for hypertension, autogenic training for migraine headaches, and psychotherapy for ulcerative colitis. Within the domain of the psychotherapies, psychoanalytically oriented treatment had the best differential outcomes in well-differentiated patients, and nondirective and supportive techniques fared best with symbiotic and regressed patients. Kellner's conclusions (10), based on a substantially greater number of studies, were more complex and cautious. Some disorders (e.g., asthma, ulcers, and migraines) appeared to be more amenable to psychotherapy than others (e.g., hypertension); some techniques fared better than others for different disorders (e.g., group directive techniques for peptic ulcers versus support and relaxation for muscular pain); and there seemed to be distinct individual and psychopathological differences in receptivity to different types of treatment.

Finally, my review, arranged according to therapeutic modality, serendipitously suggests that the differential pairing of disorder and treatment has often been implicit in the literature. It is not entirely clear whether this has occurred by accident or design. Thus group therapy techniques have been preferentially applied and reported with positive results primarily in the treatment of myocardial infarctions and migraines; family therapy techniques for diabetes, eczema, and anorexia nervosa; and individual therapy techniques for ulcerative colitis and peptic ulcer.

On the basis of such interesting but inconclusive findings, it is becoming clear that ultimately the efficacy of psychological interventions for psychosomatic disorders (as well as for other medical ills) will have to be based on careful comparisons of the outcomes obtained by different forms of psychotherapy in direct contrast with other therapeutic (psychological and medical) regimes.

CONSIDERATIONS FOR PRACTICE

In an assessment of the entire field of psychosomatic medicine in the 1970s, the development of increasingly effective therapeutic techniques was one of the major features noted (71). Most recommendations in this regard have pertained to *flexibility* of the therapeutic approach to psychosomatic patients, perhaps more so than would be required for psychoneurotic patients (1, 3, 23, 72). As has occurred in work with patients with personality disorders (23), borderline patients (72), and latent psychotics (3), most theorists and therapists in the field have ultimately relinquished, modified, or combined classical psychoanalytic techniques with essentially supportive approaches within the dyadic framework (1–3, 73), and/or have increasingly added to the therapeutic repertoire other than individual psychotherapeutic approaches, e.g., mother-child, family, or group approaches (3, 7, 33, 47–56, 58–67).

Wolff (1), who regards the main technical problem of such therapy as how to handle these patients' tendency toward regression, has suggested that the psychosomaticist must locate the middle ground between gratification and interpretation. Sperling (3, 33) cited psychoanalysis as her treatment of choice because it is capable of eliminating or resolving the psychosomatic patient's dependency needs, whereas supportive therapy merely shifts it onto the therapist. However, she felt that psychosomatic disorders may be treated by either therapy, particularly if one is aware of the assets and limitations of each. When using supportive therapy, guidelines include working with the patient on a conscious level only; providing insight by showing the connection between the occurrence of symptoms and affects without any interpretation of specific conflicts; helping the patient to direct his or her aggression so that it is not released solely through somatic symptoms; not dealing with transference phenomena; and not interfering with the patient's dependency relationship with the therapist or others in his or her environment (3). Others have concurred with these recommendations and other modifications of psychodynamic therapy, such as sitting face-to-face with the patient, not being silent, actively offering reassurance, and

seeing family members (2). It has also been suggested that the therapist interpret the patient's body posture and movements as descriptive of his or her affect, as an alternative to the direct interpretation of the emotion behind the affect (6).

More specifically, Alexander (57) has suggested that alternative approaches could greatly facilitate the clinical management of these patients, particularly when they are based on precise knowledge of the psychophysiological interaction and nuclear conflict of different types of patients. For example, ulcer patients, for whom accepting help from others mobilizes shame and guilt, react favorably to authoritative management; and the hypertensive patient's inclination to submit himself or herself to unrewarding and strenuous life situations that he or she inwardly resents can be useful in teaching the patient to avoid these types of stress-provoking circumstances.

While such suggestions have often provided the basic theoretical and technical orientation for approaching different types of psychosomatic patients, more recent recommendations are less contingent on the type of patient befitting a specific disease entity, nuclear conflict, or characteristic interaction. They have instead advocated major attention to the fit between patient and therapist (47), consideration of the nature of the patient's psychopathology (perhaps more so than the nature of the physical disorder) (10), and approaches that are not oriented to the illness but to the person and situation (68).

In a comprehensive study of therapeutic factors in the psychotherapy of ulcerative colitis patients, Karush and associates (42) found that the techniques of interpretation, support, emotional abreaction, and direct suggestion were variably effective with different patients, albeit with the same basic disorder. The more individuated patients did well with interpretation and abreaction, whereas the more symbiotic patients did better with support, suggestion, and graded abreaction. The latter also needed concrete verbal and behavioral evidence of their therapists' attachment and availability; and they were flooded by reactive anxiety about retaliatory punishment, and their symptoms intensified, after a cathartic outburst. Essentially, it was the fit between the patient's dependency needs and style of expressing them, and the therapist's response to them, that was particularly important to outcome. The best overall results were obtained by those therapists who were most flexible, that is, they could be active with some patients and passive with others.

It has been suggested that the overriding goal in dealing with all psychosomatic patients is the restoration of unity between psychic and somatic experience regardless of specific symptoms (68). Psychotherapeutic techniques would thus include the following recommendations: directly communicating to the patient a concern for his or her bodily symptoms by empathizing with his or her fears of being physically ill and allowing opportunities to express fantasies of illness; exploring life circumstances at the onset of illness; showing sensitivity to psychosomatic phenomena by linking psychic or feeling experiences with accompanying bodily sensations; and providing awareness of the link between life stresses and physical illness by shifting concentration from the illness itself to personal problems and painful feelings connected with the patient's life situations. Additional recommendations include deemphasizing physical symptoms that only reinforce the split between psychological and physiological events; recognizing the patient's need for physical contact (more so than sexual contact) and the way it is symbolically expressed to the therapist; and understanding transitional phenomena, especially in the patient's turning to his or her body or parts of it, or to the doctor, as transitional objects. Finally, the therapist must convey to the psychosomatic patient that he or she is continually available (although not necessarily for formal sessions). Psychosomaticists currently advocate person-related care and respect for the patient (68), upholding the central importance of the relationship between patient and therapist (42), and recognizing individual differences in receptivity to different treatment modalities (10). The major point of view is represented in the conclusion that "flexibility is the hallmark of effective treatment" (68, p. 245).

CONCLUSIONS

The overall state of the art of psychotherapeutic intervention for the medically ill has been generally marked by a paucity of systematic investigation and research. Although the data, format, and results of studies of psychotherapy for the medically ill have been highly variable, it is possible to draw some conclusions, albeit cautious ones, about the applicability of psychotherapeutic interventions to medical illnesses. Some patients with medical disorders may respond positively to psychological treatment, either physically or psychologically. Some medical disorders appear to be more amenable to psychotherapy than are other disorders. Some therapeutic modalities appear to be more effective than others. Some individuals appear to be more responsive to psychotherapy than others, especially in relation to the nature of their psychopathology, rather than their physical pathology.

This amalgam of results has profound implications for the future of clinical practice, and suggests the following requirements in future research studies:

1. The need for more comparative research in order to systematically evaluate all approaches, for example, the use of the same technique with different diseases and the use of different techniques for the same disorder.

2. The need for several detailed and consistent criteria of outcome in the psychological, social, and physical domains (in order to clarify findings that suggest psychological or social effectiveness but no physical change, or vice versa).

3. The need for longitudinal study and follow-up at various points, especially of several years' duration (in order to clarify and extend findings that suggest immediate positive or negative effects but no sustained results, as well as positive results from long-term treatment when short-term treatment does not suffice or even temporarily produces exacerbations of illness).

4. The need for more attention to the therapeutic process, not merely the endpoint or final outcome of therapy for the patient (in order to more precisely arrive at the mediating factors during the therapeutic endeavor that produce the particular results).

5. The need for greater specificity of the variables in each treatment situation that may influence the response to particular kinds of therapy (e.g., medical disease, nature of the psychopathology, patient/therapist factors).

It may then be possible to more critically explore and comprehend the role of psychotherapy in the treatment of medically ill patients.

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Primary Prevention in Perspective

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Discussion of primary prevention has been made difficult by lack of clarity of underlying concepts and assumptions. Is the purpose to prevent diagnosable mental illness or to prevent unhappiness and social incompetence? What are the implications of the assumption that societal stress causes mental illness? Is there a clear distinction made between major and minor mental illness? Except for a few specific conditions there is little evidence that primary prevention has been effective. The authors feel that research and program evaluation in prevention is sorely needed but should be funded separately and with discretion. Scarce mental health funds should not be diverted from direct treatment for this purpose.

PRIMARY PREVENTION of mental illness—actual avoidance of the occurrence of cases—has a powerful, almost irresistible attraction. Appealing slogans such as "Curing is costly—prevention, priceless!" and recognition that there will never be enough mental health professionals and facilities to treat all current and expected cases of mental illness in the United States have led to a search for and recently an enthusiastic acceptance of prevention as an alternative to direct treatment. However, given our present state of knowledge, is primary prevention of mental illness really possible? This crucial and obvious question too often goes unanswered or even unasked during discussions of program planning. The prominence that the President's Commission on Mental Health, the Mental Health Association, and the National Institute of Mental Health have recently given to prevention suggests that a review of the issues is needed.

Clearly, much of the discussion and debate over primary prevention is related to the fuzziness of the concepts and definitions underlying the issues. Unless care is taken to distinguish prevention of diagnosable mental illness from prevention of unhappiness, feelings of distress, or social incompetence, discussants will often be examining several different phenomena while thinking they are focusing on one. Unless it is clear what position the discussants are taking on the assumption—which is at yet unproved—that difficult life circumstances lead to mental illness, much of the debate must be fruitless because this assumption is basic to most (although not all) primary prevention efforts.

To be sure, some of the dedicated and sophisticated proponents of primary prevention recognize these distinctions (1), but all too often the cautious voices of these experts are drowned out in the excitement over the prospects of preventing substantial amounts of mental illness. Although some experts may be restrained in their claims for prevention, a good part of the general interest in prevention undoubtedly stems from the expectation of reducing the number of seriously ill, hospitalized, and disabled individuals—not simply because prevention may lead to a happier life for the average person. It is crucial that the distinction between preventing mental illness and preventing general emotional distress remain clear, not only so that we are clear as to what we are trying to accomplish, but also so that we are able to make intelligent decisions about priorities and the allocation of scarce funds.

Those concerned with primary prevention in the mental health field face first a complex problem of definition: the boundaries of mental health. When used by public health practitioners, the word "prevention" almost always refers to illness. Mental health services, on the other hand, usually deal not only with individuals with diagnosable illness but also with those who have no recognized psychiatric illness but who want help with their interpersonal problems and concerns of everyday living that cause them distress and unhappiness. Some mental health specialists go still further and feel that mental health services should be concerned with resolving basic social problems and with improving the quality of life. However, the cause and effect relationship between social conditions and mental illness is extremely questionable. It may well stretch both the definition of mental health and the public health concept of prevention beyond their useful limits to speak of mental health services and primary prevention in these areas. Moreover, the concepts, techniques, and expertise necessary for effective resolution of social problems are in no way related to those used by clinicians.

Concern with primary prevention in psychiatry has been with us for many years—at least since the 1920s. Interest has waxed and waned several times, but recently there has been an upsurge of interest again, counteracting the disillusionment with prevention of the early 1970s (2). The disenchantment in the 1970s

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seems to have been a result of a combination of factors: the absence of any good evidence to confirm the effectiveness of primary preventive efforts; the recognition that primary prevention has been seriously oversold; and the shifting of interest to newer fads. Despite all this there are now again those who say that this is the time when primary prevention will come into its own. For instance, a national survey in 1974 among professionals and paraprofessionals in 515 community mental health centers and 193 state hospitals showed that respondents felt prevention was the third most critical current mental health issue, preceded only by children's services and adolescent services (3). The respondents estimated that in five years prevention would be the second most critical issue preceded only by children's services. Many articles, reviews, and books on primary prevention have recently appeared, most of which describe or advocate a particular type of preventive program. The final report of the President's Commission on Mental Health (4) recommended that increased funding be made available for primary prevention.

Despite this activity and concern, another critical issue remains—demonstrated effectiveness of primary prevention, or the lack of it. There seems to be general agreement that research has only begun to reveal something about the causes of most kinds of mental illness. Without knowledge of cause, primary prevention programs can only be shots in the dark. Moreover, recent research has increasingly suggested the operation of genetic and biochemical factors in the production of mental illness, and these factors are not targets of typical primary prevention programs.

THE SCOPE OF PRIMARY PREVENTION

Primary prevention must be distinguished from the other forms of prevention with which it is often confused. Public health practitioners divide preventive activities into primary, secondary, and tertiary (5); these terms were later adopted by mental health practitioners (6). Secondary prevention involves enabling people to regain their normal level of functioning and preventing further development of illness after its occurrence; early diagnosis and treatment is a sine qua non. Tertiary prevention involves preventing or reversing the sequelae of illness, i.e., disability. Secondary and tertiary prevention, despite their importance, do not have the glamour that primary prevention has, especially with regard to treating the major mental illnesses such as schizophrenia, nor do they offer the same potential for alleviating these disorders. For example, in the case of smallpox secondary and tertiary prevention do not hold out the same promise of widespread relief of suffering for a relatively small amount of effort that vaccination, the primary prevention, does.

Primary prevention has itself been subdivided into two categories—activities that promote health generally and thereby increase resistance to disease and activities that are aimed against the occurrence of specific illnesses.

In the area of mental health, most—if not all—successful primary preventive activities have been aimed at specific diseases. In contrast to the situation in large areas of physical illness, there is no evidence that it is possible to strengthen "mental health" and thereby increase resistance to mental illness by general preventive activities. Despite massive efforts to combat poverty (at least partly in the name of mental health), to increase social welfare and Social Security benefits, and to change the educational systems and methods of child rearing, there is no indication of a decrease in frequency of any of the functional mental illnesses. Nor is there obvious evidence that other countries with stronger social welfare systems and different child-rearing practices have different rates of mental illness. Thus, as far as we can see, the major functional mental illnesses (as well as the very frequent diagnosable minor illnesses) remain untouched by primary prevention in the sense of trying to strengthen mental health.

THE SHAKY UNDERPINNINGS OF PREVENTION

Many of the primary preventive activities currently proposed or in progress are based on theories that are far from demonstrated and no longer universally accepted in the field of mental health. In particular, we refer to the fact that most people who write or work in the field of primary prevention have a developmental orientation. They believe that later psychopathology, including the psychoses, is to be understood in terms of earlier life experiences. They "suspect that mental disorders are primarily a social phenomenon, that social factors drive people crazy. People most often become neurotic or psychotic in relation to other people" (7). As we see it, it is certainly true that the manifestations of mental illness and the social responses to mentally ill people are "in relation to other people." However, it does not necessarily follow that prevention can best be carried out in the social arena or that the principal cause of mental illness is societal stress.

This brings us to an important distinction that must be made between major mental illness, the psychoses, and minor mental illness, the neuroses and character disorders. Recent research, in particular the adoption studies of Kety and associates (8), indicates that major mental illness is probably in large part genetically determined and is probably therefore not preventable, at most only modifiable. Even that it can be modified is questioned by many and there is little hard evidence one way or the other.

When we leave the major mental illnesses we enter an area that is even less clear. There appears to be a greater likelihood that such minor mental illnesses as neuroses and character disorders are preventable by modifying societal factors that produce stress. Some

uncritically embrace this possibility as a "commonsense notion" (9). Others take an opposite point of view, again without hard evidence, that psychosocial influences have only a minor role, if any, in the causation of minor psychiatric problems. There are others, however, who take a middle course. For instance, Dunham (10) feels that

> There are certain forms of behavior, certain adult character disorders, certain patterns of nonconformity, certain symptomatic patterns that we label as psychoneuroses that may well have their roots in the character of our social life. But even here, the cause of these links is by no means conclusively established. We have numerous hypotheses, we have various perspectives and we have a certain amount of evidence attempting to link these psychological disturbances with the conditions of social organization. But there are many gaps in our knowledge here. These gaps will be particularly noticeable if we attempt to develop therapeutic procedures oriented with respect to the community that are supposed to cope with these mental ill-health conditions that have developed and are developing among certain persons in the community. (10)

The degree to which currently discussed primary preventive efforts can be effective depends on which of these hypotheses is correct. In the absence of strong evidence favoring social factors as the primary cause of neurosis, a major investment in primary prevention in the social arena is at best a hazardous venture.

As we have noted, there are those who strongly defend the relationships between environment and mental illness and some who have conducted studies to provide data in support of this hypothesis. The most notable and comprehensive of these studies have been those of Leighton and associates (11). They have shown that in the cross-sectional studies there is a relationship between the number of people who report symptoms commonly associated with mental illness and indicators of sociopsychological stress or, in their terms, "social disintegration." These indicators are weak and absent leadership, lack of recreational facilities, high crime and delinquency rates, widespread ill health, extensive poverty, cultural confusion, and a high frequency of broken homes. What these studies have failed to demonstrate is that there is a *causal* relationship between the particular stressful environmental factors under consideration and the occurrence of mental illness.

Other important studies (12, 13) have demonstrated that socioeconomic class is highly correlated with frequency of major mental illness; the bulk of the cases occur in the lowest class position, class V. Some have used this finding as evidence of a causal relationship between social class and major mental illness. Again, however, there is no evidence for such a conclusion (14).

There are several conceivable explanations for the statistical relationships found in these various studies. These include the possibility that the illness itself accounted for the person being in the stressful or undesirable situation (i.e., the hypothesis that schizophrenia accounts for one's class position rather than the class position's accounting for schizophrenia) or that the culture or sociopsychological environment influences not the occurrence of mental illness but the likelihood with which it will be reported, recognized, and labeled as such.

Aside from the fundamental flaw of a lack of good evidence of a causal relationship between societal factors and mental illness, there are a number of other problems in undertaking primary prevention that are almost as important. If it is granted for the sake of discussion that there is such a causal relationship, it remains to be demonstrated that the preventive efforts proposed will actually have a sufficient effect on the environment to change the occurrence of mental illness. Quite conceivably the work of a mental health consultant—or an army of mental health consultants—will never overcome the effects of such massive social forces and conditions as poverty, unemployment, and discrimination, any or all of which might lead to mental illness.

Again, if it is granted that environmental modification has an effect on mental illness, the question arises as to whether the types of preventive efforts that can be undertaken by mental health professionals are the most effective and appropriate ones to deal with the factors needing change. For example, assuming that one of the variables of concern for prevention is quality of education and/or student-teacher relationships, can a commonly prescribed preventive intervention—mental health consultation in the schools—actually have a significant effect on quality of education and/or student-teacher relationships? May there not be more powerful approaches to attaining the same result? Such activities, if they exist and if they fall outside of the jurisdiction of mental health professionals, are hardly likely to be called primary prevention, but their results are likely to be more to the point and their effectiveness greater.

In summary, then, much of primary prevention is based on theories that cannot be shown to account for most cases of mental illness and in particular for the major mental illnesses. Even if the underlying theory of environment as a cause of illness is accepted, there are still serious logical inconsistencies in the usual understanding of how primary prevention could work.

PRIMARY PREVENTION: PROVEN AND PROBABLE

There are primary prevention techniques in psychiatry that have been shown to be extremely effective. Psychiatric complications of syphilis and vitamin deficiency are seldom seen today in developed nations. Decreased rates of birth injury and improved prenatal care have lowered the incidence of major psychiatric problems resulting from congenital brain damage. Elimination of lead in house paint has reduced the

number of children suffering from organic brain syndromes, and control of industrial toxins has virtually eliminated "mad hatters" and other such problems.

There are newer preventive programs that undoubtedly are also effective in reducing the number of cases of illness. For instance, counseling prospective mothers not to delay pregnancy until the later childbearing years is likely to reduce the incidence of mongolism.

Other primary prevention programs show promise but have not yet been proven effective. In some cases the evidence in support of them is yet to be gathered. The present basis of such programs is theory, but this theory seems highly plausible and is generally accepted. Professional intervention and other programs for abusing parents such as Parents Anonymous seem likely to be effective in breaking the cycle of child abuse, which has been shown to be socially transmitted from generation to generation. Bowlby (15), Provence and Lipton (16), and others have demonstrated the deleterious effects of raising infants in impersonal institutions or without a consistent mother figure over a long period of time. Programs to replace institutions for homeless children with long-term high-quality foster care or adoption should prevent personality disturbance. With the mounting evidence of genetic influence on the occurrence of schizophrenia and manic-depressive psychosis, the appropriate use of birth control and genetic counseling should be effective in preventing the births of individuals who would be at high risk for development of these illnesses.

"PREVENTIVE" PRACTICES OF DOUBTFUL VALUE

Claims have been made that child-rearing practices will affect the likelihood of mental illness. These run the gamut from authoritarian to permissive, from bottle feeding to breast feeding, from mothers spending more time with their children to placing children in day nurseries while mothers find other outlets so they can appreciate their children and show more love to them on evenings and weekends. There is no evidence that any particular child-rearing practices affect the incidence of any of the mental disorders.

Mental health education, once a bright hope of community mental health, has in recent years been in eclipse, probably due in no small part to a book by James Davis titled *Education for Positive Mental Health* (17). After a thorough review of the literature, Davis concluded that in mental hygiene campaigns concerned with techniques of improving personal adjustment and prevention of mental illness, the important problem is not determining the most appropriate and effective means of communication and persuasion, but the fact that "mental health educators have little or nothing specific and practical to tell the public." For instance, even if environmental and situational stresses do play an important role in causing emotional distress, mental health educators still do not possess a body of knowledge that enables them to say more than, "Do not be poor or ignorant, and stay out of armed combat."

Crisis intervention has been considered a fertile area for efforts in primary prevention. To a large extent, however, crisis intervention is really secondary prevention, the provision of treatment services to people who are already in difficulty to enable them to regain their normal functioning and avoid further deterioration. It has been hypothesized that people are more amenable to learning more effective coping skills at times of crisis and that they will be better prepared to handle future crisis without further breakdown. Unfortunately, there is conflicting evidence that intervention will make a difference.

On the one hand, there is research evidence that it does not. Polak and associates (18) conducted a controlled study in which families who had experienced the sudden death of a family member were given crisis intervention services and compared at follow-up with two untreated control groups. Crisis intervention had not decreased the risk of psychiatric illness, disturbed family functioning, or social cost to the families. On the other hand, there are studies that have shown just the opposite. For instance, Raphael (19) showed that intervention with recently bereaved subjects decreased morbidity, compared with a control group, in terms of feelings of panic, excessive tiredness, weight loss, doctor visits for general symptoms, and diminished work capacity. Clearly, further research is needed to clarify the relationships, if any, between crisis intervention and primary prevention.

Many programs that have been called preventive programs are geared toward the development of competence in individuals. Although this is a worthy goal in and of itself, there is no evidence that developing competence prevents mental disorder. This point remains controversial, and the fact that many emotionally disturbed persons are educationally incompetent leads many to believe there is a cause and effect relationship. One often cited example is the Head Start program. Although there is no agreement as to the effectiveness of Head Start, there is some evidence (20) that this program can influence early school achievement. On the other hand, there is no evidence that this increase in achievement is related to a lowered incidence of mental illness in these individuals, although this conclusion is tempting.

Finally, there is a group of (often highly popular) activities commonly considered to be primary preventive whose effectiveness in preventing mental illness is, in our opinion, questionable to unlikely.

Foremost among these programs are a variety of measures undertaken by mental health agencies to eliminate socially undesirable factors in their communities, such as poverty, poor housing, inadequate educational services, violence, and unresponsive political systems. As Elaine Cumming (21) has pointed out,

"No one knows whether or not vile living conditions actually cause mental illness, but in a civilized society they should be found intolerable just because they are vile. All citizens, not just psychiatrists, should be appalled at them." There can be no doubt that the prevalence of symptoms associated with mental illness is higher among poor people and that rates of admission to state hospitals and of diagnosis of the major mental illnesses also are higher among this group. What remains to be unequivocally demonstrated, however, is that poverty is somehow causative. The situation is analogous to the relationships between social class, quality of environment, and mental illness, and the arguments are the same. For instance, people who are ill or on the verge of illness may gravitate toward poverty-stricken neighborhoods (downward drift).

Even if there were a clear demonstration of poverty as a causative agent of mental illness, this would not make poverty a condition to be dealt with by mental health professionals. Again, to quote Elaine Cumming, "There is a certain arrogance in someone who has been trained to heal the sick, imagining that he, therefore, has an expertise beyond that of any other thoughtful citizen in patching up the cracks in society." A more appropriate role for mental health professionals would be to emphasize the need for changes to those professional and political groups who have expertise and authority for implementing these changes.

It might well be asked why there is any need to be concerned with the definition of primary prevention and with the question of effectiveness of worthy social programs in preventing mental illness. If all agree that a program to eliminate poverty, for example, can only lead to an improvement in the quality of life and in any case is a good thing in itself, why raise these questions at all?

Why indeed! Mental health programs now and for the foreseeable future are operating in a world of shrinking resources. There is not enough money to go around, not even enough to support the basic mental health direct service programs that all recognize as fundamental and essential. To undertake programs that are expensive and quite likely not even preventive of mental illness out of funds allocated to mental health services seems to be a dangerously foolish gamble. If such programs are deemed socially desirable and politically popular, they should be supported from funds other than those allocated to mental health services.

CONCLUSIONS

A review of the field leaves only unanswered questions—many of them hidden from professional and public scrutiny by mountains of testimonials and hopes. Perhaps the foremost question is that of social policy—how much of scarce public funds and how much of our professional credibility are mental health professionals willing to risk on prevention programs that might be completely ineffective?

Unfortunately, we do not yet possess the techniques necessary to make primary prevention a reality except in a few limited areas. When allocating funds to preventive activities, therefore, we need to distinguish carefully among those programs which are proved, those which are not yet proved but seem well on the way to attaining this status, and those which are at best experimental. We need to make a distinction between programs preventive of diagnosable mental illness and programs aimed at prevention of unhappiness or social incompetence so that we can be clear as to what we are trying to accomplish and so that we are able to make informed and intelligent judgments about our priorities. We need to distinguish between prevention programs and programs designed to alleviate social and ethical problems. The latter group may well demand attention and support from mental health professionals, but primarily as concerned citizens and not as scientific experts. Prevention's appeal must be resisted by mental health professionals lest it become a glamorous rationalization for avoiding treatment of difficult, mentally ill persons, such as chronic psychotic, alcoholic, and addicted patients. Evaluation of basic and applied research and of programs in prevention is sorely needed, but it ought to be funded separately from direct treatment programs (22). Research and program evaluation, when properly carried out, is so expensive that such studies cannot be grafted on to existing programs as "minor" additions.

Finally, it seems that there is no easy way out of the problems that those of us concerned with serving the mentally ill face. Our difficult patients will not magically go away. We will simply have to struggle with each of the many discouraging if not overwhelming complexities and obstacles to overcoming mental illness and resist the temptation to go prospecting for the pot of gold at the end of the rainbow.

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Relationship of Plasma Levels of Chlordiazepoxide and Metabolites to Clinical Response

BY KEH-MING LIN, M.D., AND ROBERT O. FRIEDEL, M.D.

The authors report on the relationship between the antianxiety effects of chronically administered chlordiazepoxide (CDX) and plasma levels of CDX and two of its metabolites, desmethylchlordiazepoxide (DMCDX) and demoxepam (DMX). Fifteen subjects with moderate to severe anxiety were studied in a double-blind, placebo-controlled, crossover design. Significant correlations were found between anxiety reduction and DMCDX and DMX plasma levels. No such correlation was observed between anxiety reduction and CDX levels. These data suggest that in chronically treated subjects, DMCDX and DMX have significant antianxiety properties which surpass those of CDX itself.

BENZODIAZEPOXINES have been generally accepted as effective in anxiety reduction (1) and are widely used in both psychiatry and general practice (2). However, the influences of drug absorption, distribution, metabolism, and excretion on the efficacy of these anxiolytic agents are still far from clear. In what is to our knowledge the only previously published report of this type involving chlordiazepoxide (CDX), Gottschalk and associates (3) studied the effects of a single 25-mg dose of this drug on anxiety; this study included evaluation of CDX blood levels. No placebo-drug difference in anxiety reduction was noted in the entire group. However, the 11 subjects who had CDX blood levels greater than 0.70 μg/ml 50 minutes after drug ingestion showed a significant placebo-drug difference in anxiety reduction, suggesting that a minimal blood concentration of CDX is required to obtain an antianxiety effect after acute treatment. Greenblatt and associates (4) evaluated the correlation between blood levels of CDX and its metabolite desmethylchlordiazepoxide (DMCDX) and behavioral symptoms in 10 asymptomatic healthy males after a single 25-mg dose of the drug. Self-ratings of "feeling spacey" at 1.0 hour after the dose were highly correlated with 0.5-hour blood levels of CDX plus DMCDX, but not with 1.0-hour blood levels.

A search of the literature revealed no reports of studies evaluating the relationships between the antianxiety effects of chronically administered CDX and plasma levels of CDX and its major metabolites. Since chronic administration is the most common dosage regimen of this widely used drug, such a study would seem indicated. Therefore, we have evaluated the relationship between the antianxiety effects of CDX and steady-state plasma levels of this drug and two of its metabolites, DMCDX and demoxepam (DMX), in 15 symptomatic unpaid volunteer subjects using a double-blind, crossover, placebo-controlled design.

METHOD

An advertisement was placed in the general news section of a large urban newspaper giving a clear description of the study. It included a 58-item Hopkins Symptom Checklist (HSCL) (5) as an initial screening instrument. Respondents with scores greater than 11 on the anxiety subscale of the HSCL (items 2, 17, 23, 33, 50, and 57) and less than 6 on four depression items (15, 19, 30, and 54) were contacted by telephone by the study nurse to elicit demographic information and a brief medical, psychiatric, and drug history, and to evaluate their willingness to follow the study protocol. After telephone screening, suitable subjects were referred to the study physician for the predrug evaluation, which included a medical and psychiatric interview, a physical examination, and completion of the Hamilton Anxiety Rating Scale (HARS) (6). Blood and urine samples were taken for electrolytes, glucose, liver-function tests, urinalysis, and drug screening. A tentative appointment was made one week later for the first session of the study pending results of laboratory studies.

Additional criteria for inclusion in the study were age between 18 and 50 years; a high-school diploma or equivalent; an HARS score greater than 12; the pres-

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ence of sufficient symptoms so that, in the study physician's clinical judgment, treatment with an antianxiety medication was indicated; and willingness to return for subsequent visits. A signed consent form was obtained after the nature of the procedure had been explained to the subjects. Criteria for exclusion from the study were the presence of a significant physical impairment or medical disease; symptoms and signs indicating the history or presence of a borderline or overt psychosis, severe depression, or organic brain syndrome; the use during the previous month of any drugs, including marijuana, more than twice weekly, or of alcohol, more than one drink per day; pregnant women or nursing mothers; women of childbearing age not practicing reliable physical or chemical contraception; and a history of convulsive disorders, narrow-angle glaucoma, or allergy to benzodiazepines.

Fifteen subjects meeting the criteria were selected to participate in a double-blind, placebo-controlled, crossover study. Subjects were randomly assigned to receive identical capsules of either chlordiazepoxide hydrochloride or placebo; crossover was done at the end of 1 week. Subjects were instructed to return the bottle and unused medication at the end of the week, and a pill count was then recorded. The recommended dose of CDX was two capsules t.i.d. (20 mg t.i.d., 60 mg/day), with the last dose taken approximately 6 hours before each interview. If excessive sedation or other adverse effects were noted, the subject was instructed to take one capsule in the morning and at noon and two capsules at night (40 mg/day). If side effects persisted, the noon dose was to be omitted (30 mg/day). Subjects were advised to take at least 30 mg/day unless serious adverse effects persisted (this did not occur during the study).

Anxiety was evaluated by the following instruments: the 58-item Hopkins Symptom Checklist, a self-rating scale with subscales covering symptoms of anxiety, depression, obsessive-compulsiveness, somatization, and interpersonal sensitivity (5); and the Hamilton Anxiety Rating Scale, a physician-administered scale with items covering 14 symptom categories of anxiety, which can be divided into psychic and somatic subscales (6).

The severity of drowsiness was rated on a 4-point scale, with 0 = none, 1 = mild, 2 = moderate, and 3 = severe. The numerical values indicating the total magnitude of this side effect were computed by multiplying its severity by its duration in days (e.g., severe drowsiness lasting 7 days would give a score of 21).

Three interview sessions were conducted during the 2-week period of investigation, one before treatment and one at the end of each week of treatment. In all three sessions the subject first completed the HSCL, and then one of the investigators (K.L.M.) conducted the interview and rated the HARS. After the third (last) session was over, a discussion was held with the subject about his/her problems, and further treatment or referral was provided if necessary.

After each session, a urine sample was obtained for drug screening. At the end of the second and third sessions, blood samples were drawn for determination of drug plasma levels. Ten ml of blood were drawn from each subject into heparinized vacutainer tubes (Becton-Dickinson, East Rutherford, N.J.). Plasma was separated immediately and frozen. The Koechlin and D'Arconte (7) method as modified by Schwartz and Postma (8) was used for detection of CDX, DMCDX, and lactam metabolite (DMX). The sensitivity of this method for all three compounds is 0.2 μg/ml.

Differences between means were determined using Student's t test and the Wilcoxon sign test (9). Pearsons' product-moment correlation coefficients and Spearmans' rank-order correlations (9) were computed between variables such as symptom reduction, side effects, demographic variables, and plasma drug levels.

RESULTS

Seven men and 8 women, aged 28–50, completed the study protocol (see table 1). No subjects dropped out of the study. The mean reported daily dose of CDX for subjects during the drug week was 53.4 mg for women and 57.6 mg for men. Pill counts taken at the end of each week were generally in good agreement with the reported dosage for both placebo and drug with two exceptions (subjects 9 and 10).

Placebo-drug differences in anxiety levels as measured by the HARS and the HSCL for each of the 15 subjects are presented in table 2. Most subjects reported and were rated as having less anxiety after medication than after placebo. The placebo-drug differences in mean reduction in anxiety as measured by the total HARS (36%), the HARS psychic (39%) and somatic (31%) subscales, the total HSCL (24%), and the HSCL anxiety subscale (35%) were all statistically significant (see table 2) and were independent of any placebo-drug ordering effect.

Eight subjects reported drowsiness as a side effect (table 1). In these subjects it occurred more often in the drug than in the placebo week, with statistical significance approaching the .05 level ($t=1.98$). Other side effects were rare and transient, with 2 subjects reporting bitter taste after medication (subjects 3 and 12), and 1 each reporting thirst (subject 5), headache (subject 13), and questionable eyelid edema (subject 15), in the drug week. There was no relationship between the occurrence of side effects and rating scale changes, which ruled out the possibility of score assignments being contaminated by the presence of side effects. Age was found to be correlated positively both with drowsiness ($p<.05$) and with numbers of pills omitted ($p<.05$). There was also a trend toward a positive correlation between drowsiness and numbers of pills omitted ($p<.1$), suggesting a relationship between these three variables.

Apparent steady-state plasma levels of CDX and its metabolites (DMCDX and DMX) showed marked in-

TABLE 1
Dosage of Chlordiazepoxide and Drug Plasma Levels of Chlordiazepoxide and Its Metabolites

Subject	Sex	Age (years)	Mean CDX Dose (mg/day)	Remaining Pills		Drowsiness*		Drug Plasma Levels (μg/ml)**			
				Drug	Placebo	Drug	Placebo	CDX	DMCDX	DMX	Total
1	M	33	59	1	0	4	6	1.6	1.6	1.0	4.2
2	M	30	60	0	2	0	0	0.4	1.2	0.2	1.8
3	M	35	59	0	0	0	0	3.7	1.9	0.9	6.5
4	F	37	57	2	0	2	0	2.1	1.3	1.1	4.6
5	F	46	51	7	2	14	0	2.4	1.4	0.7	4.5
6	F	36	60	0	3	7	2	1.6	1.3	0.0	2.8
7	M	30	60	4	0	0	0	1.8	1.2	0.3	3.3
8	F	42	46	8	2	0	0	2.4	1.1	0.5	4.0
9	F	44	57	7	11	14	2	4.5	2.0	1.2	7.7
10	M	47	60	8	0	0	0	3.6	1.0	0.5	5.2
11	F	34	57	2	0	0	0	3.0	2.2	0.7	6.0
12	M	30	45	10	0	5	0	2.5	1.2	0.8	4.5
13	M	28	60	0	2	0	7	2.8	0.7	0.3	3.8
14	F	45	57	2	10	0	0	0.0	1.7	1.3	3.0
15	F	50	41	9	5	14	0	1.4	1.5	0.5	3.4
Mean		37.8	55.3	4.0	2.5	4.0	1.1	2.25	1.41***	0.67	4.35
SD		7.3	6.4	3.7	3.6	5.6	2.3	1.20	0.40	0.38	1.53

*Score indicates severity rated on a 4-point scale (0=none, 3=severe) multiplied by duration in days.
**Significant difference in mean plasma levels of CDX, DMCDX, and DMX ($F=15.9$, $p<.01$).
***Significant correlation with DMX ($r=.58$, $p<.05$).

TABLE 2
Analysis of Differences in Rating Scale Scores Following Chlordiazepoxide and Placebo Administration

Subject	Mean CDX Dose (mg/day)	Rating Scale Change Scores*				
		HARS			HSCL	
		Total	Psychic	Somatic	Total	Anxiety
1	59	−5	−1	−4	−16	−6
2	60	−2	−1	−1	−52	−9
3	59	−7	−3	−4	−37	−10
4	57	−1	−1	0	−21	−7
5	51	−5	0	−5	13	0
6	60	−5	−4	−1	−20	0
7	60	3	0	3	26	9
8	46	3	1	2	0	0
9	57	−12	−5	−7	−18	−1
10	60	−12	−7	−5	−22	−4
11	57	−20	−10	−5	−59	−9
12	45	−7	−4	−3	−17	−9
13	60	−4	−5	1	−3	−6
14	57	−18	−8	−10	−39	−9
15	41	−11	−8	−3	−27	−1
Analysis**						
Mean change		−6.9	−3.7	−2.8	−19.5	−4.1
SD		6.8	3.4	3.5	22.6	5.3
t**		−3.99	−4.24	−3.09	−3.33	−3.04
Significance		p<.01	p<.01	p<.001	p<.01	p<.01

*Minus sign indicates symptom reduction during drug period compared to placebo period.
**Due to the relatively small sample, a nonparametric method (the Wilcoxon test) was also used, with similar results.

terpatient variations, with standard deviations of 53%, 29%, and 58%, respectively (see table 1). Mean plasma levels of CDX and metabolites were significantly different; CDX levels were higher than DMCDX levels, which were higher than DMX levels. There was poor correlation between plasma levels of CDX and its two metabolites, DMCDX ($r=0.19$) and DMX ($r=0.16$).

TABLE 3
Correlations Between Symptom Reduction and Chlordiazepoxide and Metabolite Plasma Levels

Rating Scale	Correlation Coefficient (r)*			
	CDX	DMCDX	DMX	Total
HARS				
Total	−0.12	−0.63**	−0.42	−0.3
Psychic	−0.11	−0.39	−0.15	−0.23
Somatic	0.06	−0.64**	−0.65**	−0.39
HSCL				
Total	0.13	−0.51	−0.21	−0.09
Anxiety	0.04	−0.24	−0.40	−0.14

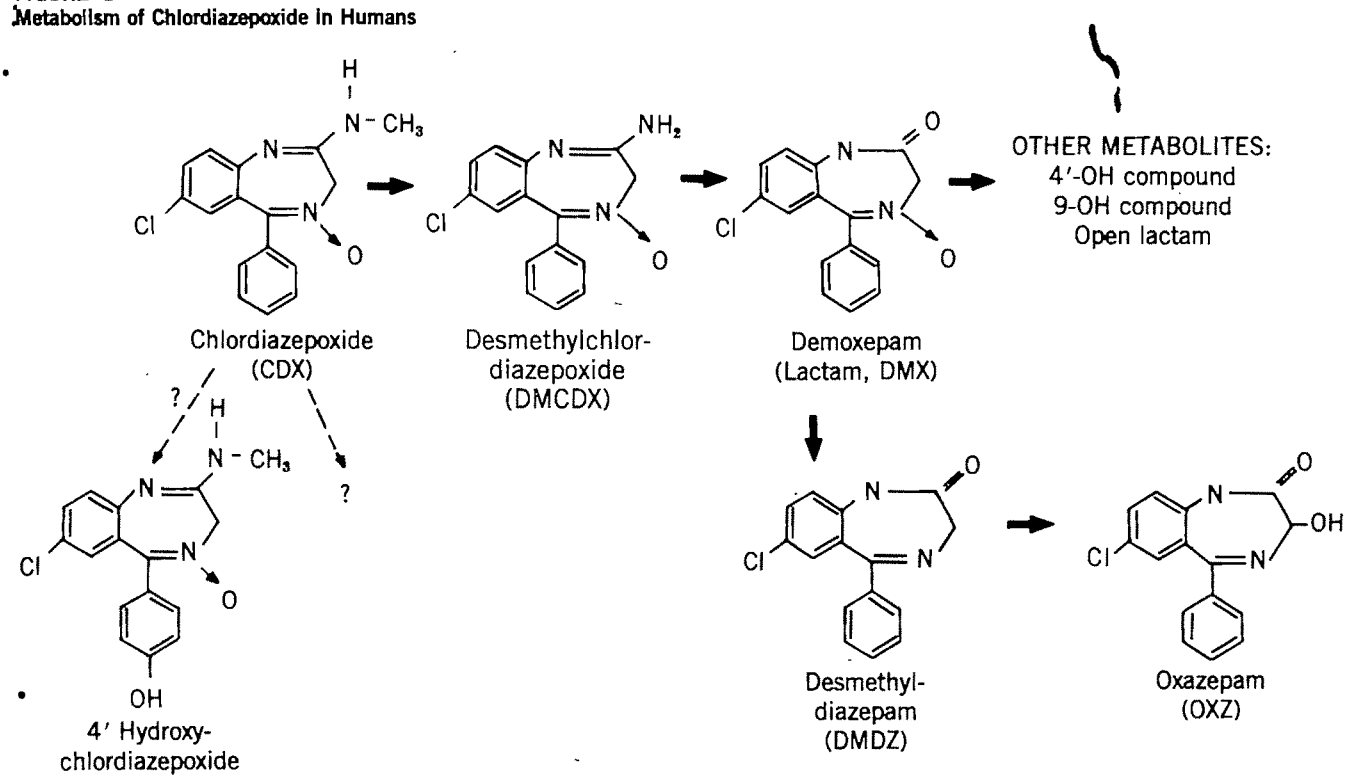
*Nonparametric analysis with Spearman's rank-order correlation yielded identical results.
**$p<.02$.

The correlation between plasma levels of DMCDX and DMX was statistically significant (see table 1).

Correlations between symptom improvement (placebo-drug differences) and drug and metabolite plasma levels are presented in table 3. There was no correlation between antianxiety effect and plasma levels of CDX, whereas the correlations between DMCDX and total and somatic HARS change scores and between DMX and the somatic HARS change score achieved statistical significance. This correlation also exists between the total and somatic HARS change scores and the sums of DMCDX and DMX. Improvement in the HSCL rating scores did not correlate with plasma levels of CDX or its metabolites. The ratios of plasma levels of CDX to DMCDX, and DMCDX to total plasma levels were also tested against symptom reduction on both the HARS and the HSCL; these comparisons did not achieve statistical significance.

The plasma levels of CDX and metabolites do not manifest any significant correlation with sex, age, drug

FIGURE 1
Metabolism of Chlordiazepoxide in Humans



dosage as determined by pill count, and incidence of side effects.

DISCUSSION

To our knowledge this is the first reported evaluation of the relationship between anxiety reduction and plasma levels of chlordiazepoxide and its two metabolites, desmethylchlordiazepoxide and demoxepam, after chronic drug administration. The data indicate that the antianxiety effect of CDX after 1 week of administration is significantly correlated with plasma levels of its two major metabolites, DMCDX and DMX (table 3). In addition, there is a trend toward a positive correlation between symptom reduction and plasma total drug levels. There appears to be no suggestion of a correlation between anxiety reduction and CDX plasma levels. The significant correlation between DMCDX plasma levels and the total HARS and the HARS somatic subscale change scores, as well as a trend toward a correlation between DMCDX levels and symptom reduction measured by the HARS psychic subscale, suggests that DMCDX may affect somatic and psychic components of anxiety. This is in contrast to the relationship of DMX plasma levels with the reduction of only somatic symptoms of anxiety. The importance of CDX metabolites to therapeutic response with chronic treatment may be even more complicated, since it has been demonstrated that DMX is metabolized further to desmethyldiazepam, which is then metabolized to oxazepam (10) (figure 1), both of which are also known effective antianxiety agents. It would be important to also evaluate plasma levels of these metabolites in future studies of CDX.

These data may explain the failure of Gottschalk and associates (3) to demonstrate a significant antianxiety effect of CDX after a single dose of the drug, and a lack of correlation between CDX plasma levels and symptom reduction in their total sample. However, their finding of a significant antianxiety effect at plasma levels of CDX greater than 0.7 μg/ml does suggest that CDX itself has antianxiety properties, although possibly less than DMCDX or DMX. Our data in humans agree in part with studies performed in lower species. The antipentylenetetrazol activity of CDX in the rat appears to parallel brain levels of DMCDX rather than those of CDX (11). However, in the monkey, taming effects of CDX appear to parallel brain concentration of CDX better than that of the metabolites (12). Since other studies have demonstrated significant interspecies differences in the pharmacological effects of CDX and its metabolites (13), extrapolation of such data to humans is problematic.

The major therapeutic implication of these data is that one would not anticipate a rapid antianxiety effect after administration of CDX, since a significant amount of metabolism to its metabolites may first be required. This interpretation is consistent with the clinical observation of a delay in onset of action of CDX compared with diazepam. It may also explain the relative preference for diazepam over CDX by physi-

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cians and patients (14, 15) and its greater incidence of abuse (16). A rigorously controlled study comparing the temporal antianxiety effects of CDX, DMCDX, and diazepam and the relationship of these effects to plasma drug and metabolite levels would be most useful in clarifying this important point.

It may be argued that the lack of correlation of CDX plasma levels with antianxiety effect observed in this study is a function of the pharmacokinetic differences known to exist between CDX and its two major metabolites after chronic ingestion. Boxenbaum and associates (17) have recently reported that under these conditions CDX plasma levels vary more with time than those of DMCDX and DMX because of the shorter half-life of CDX. Therefore, this greater variability, which would be sensitive to the time lapsed between the previous dose and the taking of the blood samples, might mask a correlation between CDX plasma levels and pharmacologic activity. Although an effort was made in this study to ensure subject drug compliance and to obtain the blood sample approximately 6 hours after the previous dose, this is still a possibility. The high degree of intersubject variability of CDX plasma levels we have observed (SD=53%) and the lower variability of DMCDX (SD=29%) would seem to support this interpretation. However, the reverse is true for DMX (SD=58%), which correlates with antianxiety effect, and for total plasma levels (SD=35%), which do not. It would be important to control carefully for this possibly confounding factor in future studies.

There is no significant relationship between drug dosage and behavioral response. This is not surprising, since plasma drug levels are a function of drug absorption, distribution, metabolism, and excretion (18). Of these four factors, only the amount of drug absorbed is related to drug dosage. Most subjects reported that they took close to the maximum amount of drug prescribed (these reports were supported by pill counts), with a mean daily dosage of 55 mg (table 1). Therefore, it would seem likely that the intersubject variability in apparent steady-state plasma levels of CDX, DMCDX, and DMX is not related to differences in drug dosage but rather to interpatient differences in the pharmacokinetic processes noted above.

The blood levels of CDX reported probably represent steady-state levels, since most subjects had been taking the same dosage of medication for at least 4 days before the blood sample was taken. Steady-state levels are achieved in approximately four half-lives of a drug if it is administered at dosage intervals no greater than the half-life (19). Since the mean half-life of CDX has been reported to be 7 to 28 hours (20–22), steady-state levels probably had been achieved at the end of the drug treatment week. However, since the elimination half-lives of CMCDX and DMX appear to be considerably longer (17, 20, 22), it is possible that the plasma levels reported for these substances do not represent steady-state values, although they are probably very close (17). The lack of correlation between plasma levels of CDX and either DMCDX or DMX suggests that another metabolic pathway for CDX exists in man. For example, it is possible that hydroxylation of CDX to 4-hydroxy CDX also occurs (see figure 1). Although such a pathway has not been reported previously in man, it has been described in the rat (23). That DMX is the major metabolite of DMCDX is supported by the significant correlation between plasma levels of these two substances (table 1).

In this sample, plasma drug levels did not appear to be influenced by demographic factors such as sex and age. However, since the sample was limited to ages 28–50 and to subjects in good physical health, the lack of an age-related effect on plasma drug and metabolite levels should not be extrapolated to elderly patients. This cautionary note is supported by the recent data of Shader and associates (22), which indicate that absorption and metabolism of CDX are significantly reduced in the elderly.

We were not able to relate significantly the side effects reported in this study to plasma levels of CDX or of its metabolites or to total drug plasma levels. However, the correlation between age and the number of pills omitted and drowsiness suggests that older subjects are more sensitive to this side effect and that they reduced the intake of medicine to lessen its severity. This is consistent with the recent findings of Greenblatt and associates (24), indicating an increased incidence of residual drowsiness and other toxic symptoms in elderly subjects compared with younger ones treated with flurazepam. In our study, had subjects not been instructed to decrease medication in the presence of side effects, the incidence and severity of drowsiness may have been greater, and a positive correlation with plasma levels of CDX or its metabolites might have been achieved.

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Papers on Hospitalization to Appear in *H&CP*

The 1978 APA annual meeting included a session titled Hospital Treatment of Emotional Disorders: Circa 1978. The group of timely and important papers presented at this session will be published in the February 1979 issue of APA's specialty journal, *Hospital & Community Psychiatry*.

The titles and authors follow:

"National Trends in Hospitalization," by Gerald L. Klerman, M.D.

"Intensive Psychiatric Care for Adolescents and Young Adults: A Model for Treatment and Evaluation," by John S. Strauss, M.D., T. Wayne Downey, M.D., and William H. Sledge, M.D.

"Short-Term Hospitalization and the Medical World," by Marvin I. Herz, M.D.

"The Hospital and Optimal Chemotherapy in Schizophrenia," by Theodore Van Putten, M.D., Philip R.A. May, M.D., and Stephen R. Marder, M.D.

"Hospitals in the 1980s: Service, Training, and Research," by Ira D. Glick, M.D., and William A. Hargreaves, Ph.D.

The Changing Role of Psychiatrists in Community Mental Health Centers

BY WALTER W. WINSLOW, M.D.

The community mental health center (CMHC) movement, largely conceived by psychiatrists to improve standards of mental health care nationally, appears to be going through an evolutionary phase in which there is decreasing utilization of psychiatrists and increasing utilization of other mental health professionals. The author discusses some factors that may have influenced this trend. In order to counter it CMHCs must become a recognized, essential, and respectable part of a pluralistic system of mental health care, and psychiatrists must be willing to assume their responsibility for involvement in all segments of mental health care, including the private and public sectors, in both leadership and clinical positions.

THE COMMUNITY mental health center (CMHC) movement could be seen as having its beginning in 1946 when President Truman signed into law the National Mental Health Act, creating the National Institute of Mental Health (NIMH). This was a significant milestone for mental health, for it marked the beginning of a unique national mental health program in research, education, and service.

In the early 1950s the leaders in psychiatry realized that much remained to be done, since there were still periodic exposés of "snake pit" care in the nation's state mental hospitals. As a result of increasing public concern, Congress passed the Mental Health Study Act, signed into law by President Eisenhower in July 1955, which authorized an appropriation to NIMH for a study and recommendations regarding mental health and mental illness in the United States. Out of this action came the Joint Commission on Mental Illness and Health, with a mandate to analyze and evaluate the needs and resources of the mentally ill in the United States and make recommendations for a national mental health program.

The Joint Commission study resulted in the landmark document *Action for Mental Health*, published in 1961 (1). This report urged an expanded program of services and long-term research. It attacked the concept of large state hospitals and proposed that they be converted into smaller regional intensive care centers. It recommended the development of community mental health centers, including emergency psychiatric services, for both chronically and acutely ill patients. The Joint Commission report also proposed massive financial participation by the federal government in the care of mentally ill patients, a responsibility that had previously fallen to the states. It outlined the principle of matching grants to facilitate federal involvement; it recommended that grants be awarded according to criteria of merit and incentive to be formulated by an expert advisory committee appointed by NIMH.

With the release of the Commission's report the stage was set for federal action. In 1963 Congress passed the Community Mental Health Centers Act (Public Law 88-164), which was signed into law by President Kennedy in October of that year. The original act authorized a total of $150 million in construction funds for a 3-year period, but financial assistance to communities for services was not provided until 1965 when Congress amended the act through Public Law 89-105 to provide grant funds for initial staffing costs for professional and technical personnel. These staffing grants were authorized on a decreasing matching basis for a period of 51 months, after which time it was expected that the CMHCs would have gained enough local financial support to continue operations without further federal funds.

In 1970, when it was realized that most centers could not become financially self-sufficient in 51 months, the duration of all staffing grants was extended to 8 years (Public Law 91-211). The legislative authority for new grants under the CMHC program expired on June 30, 1974, and was replaced by Congress with the 1975 amendments (Public Law 94-63), which authorized funds to create new CMHCs and to continue existing centers. The 1975 amendments were detailed and complicated, and regulations to implement this legislation often appeared not to support the intent of Congress, which was to have a community mental health service in every section of the country by 1985.

The community mental health center legislation—a "bold new approach" (2)—had a significant impact on the development of health care and contributed to what has been called "the third mental health revolu-

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tion" (3). It modified the delivery of mental health services by deemphasizing institutionalization and transferring most of the mental health services to the community.

While the CMHC has expanded, the psychiatrist's influence and involvement in the CMHC seem to have diminished. It appears that other mental health professionals have assumed roles traditionally held by psychiatrists.

TRENDS IN CMHC MENTAL HEALTH MANPOWER

Psychiatric manpower in the United States has increased from 3,000 psychiatrists in 1939 to approximately 27,000 in 1975 (4). Much of this growth can be attributed to federal training monies made available through NIMH. The following data illustrate that psychiatrists are leaving CMHCs at a rapid rate and are being replaced by other mental health professionals.

On January 1, 1975, there were 434 federally funded CMHCs throughout all 50 states (5). This number had increased to 507 by July 1975 and to over 600 by 1977 (6). All 50 states have at least 1 CMHC, with California and Pennsylvania having 48 and 41, respectively, and Nevada having 1. The 434 CMHCs in 1975 employed 1,861 full-time equivalent psychiatrists, or about 6% of the total psychiatric manpower in the United States, serving catchment areas with a population of approximately 80 million people (7, 8).

Professional staffing patterns in CMHCs have shown a definite shift from 1970 to 1975. Table 1 reveals the relatively slow growth in the total number of psychiatrists and full-time equivalent psychiatrists employed in CMHCs from 1970 to 1975 and the more rapid growth in the number of psychologists, social workers, and other professionals.

Table 2 indicates that the average number of full-time psychiatrists per CMHC is diminishing, while the number of other mental health professionals is increasing. In 1970 there were 3.1 full-time psychiatrists per center; by 1975 this figure had dropped to 2.4. The decreasing number of full-time equivalent psychiatrists shown in table 2 probably also reflects decreasing part-time involvement, whereas the opposite is occurring for other mental health professionals. From 1970 to 1975 the number of CMHCs grew from 205 to 434 (over 100%), while full-time equivalent psychiatrists increased from 1,394 to 1,861 (approximately 50%). Over the same period the number of social workers increased more than 150% and the number of psychologists over 250%. The percentage of psychiatrists among total professional staff decreased from 25% to approximately 15%, while the other mental health professionals showed an increase.

The use of CMHCs as training sites for mental health professionals reflects similar changes. Fewer psychiatrists are receiving part of their training in CMHCs: 2.4 per CMHC in 1970 compared with 1.1 per CMHC in 1975. Psychology and social work professionals have increased their use of CMHCs as a training base over the same period. In 1970 there were 0.8 psychology trainees and 1.4 social work trainees per CMHC; by 1975 these figures had increased to 1.7 and 2.2, respectively.

TABLE 1
Number of Total Staff in CMHCs, 1970–1975*

Item	1970	1971	1972	1973	1974	1975
Number of centers	205	256	293	326	402	434
Psychiatrists	1,981	2,257	2,524	2,441	2,872	2,968
Full-time equivalents	1,394	1,462	1,583	1,599	1,848	1,861
Psychologists	1,314	1,593	2,331	2,654	3,781	4,685
Full-time equivalents	1,005	1,226	1,807	2,111	2,994	3,688
Social workers	2,361	2,834	3,712	4,147	5,251	6,262
Full-time equivalents	1,989	2,362	3,004	3,478	4,418	5,291
Other professionals	2,502	4,747	3,541	4,161	5,860	7,225
Full-time equivalents	1,825	2,831	2,599	3,231	4,557	5,850

*Based on data from reference 5.

TABLE 2
Average Number of Full-Time Staff per CMHC, 1970–1975*

Item	1970	1971	1972	1973	1974	1975
Number of centers	205	256	293	326	402	434
Psychiatrists	3.1	3.0	2.9	2.6	2.6	2.4
Full-time equivalents	6.8	5.7	5.4	4.9	4.6	4.3
Psychologists	3.8	3.9	5.0	5.4	6.3	7.2
Full-time equivalents	4.9	4.8	6.1	6.5	7.5	8.5
Social workers	8.1	8.0	8.9	9.3	9.7	10.7
Full-time equivalents	9.7	9.3	10.3	10.7	11.0	12.2
Other professionals	7.3	10.0	7.5	8.7	10.1	12.2
Full-time equivalents	8.9	11.1	8.8	9.9	11.4	13.5

*Based on data from reference 5.

In another trend, fewer psychiatrists are directing CMHC programs. In 1973, 56% of all centers had psychiatrist directors or executive directors, compared with only 35% in 1975 (9, 10). A survey conducted in March 1977 indicated a further decline, with only 22% of all centers having psychiatrist directors (W.W. Winslow, unpublished data, 1977).

Over this same 5-year period the number of patients per CMHC increased more than 50%, including a large percentage of inpatients with major mental disorders. This implies that the psychiatrist, with his or her unique medical and pharmacological expertise, ought to assume a larger role in the CMHC.

DISCUSSION

The CMHC movement, largely conceived by psychiatrists to improve the standards of mental health care nationally, appears to be going through an evolutionary phase in which there is decreasing utilization of the psychiatrist and increasing utilization of other mental health professionals. From the beginning the CMHC approach was conceived to be multidisciplinary and multimodality; appropriate professional, technical, and paraprofessional manpower were to

complement and supplement each other. It was envisioned that psychiatrists would be significantly involved; initially this was the case, but the situation is rapidly changing, and in many CMHCs they now have little involvement.

The data clearly show a consistent trend away from the use of the clinical skills and leadership abilities of the psychiatrist, both in full-time and part-time participation in CMHCs. The use of centers for psychiatric residency training also shows a downward trend. With fewer psychiatric residents receiving part of their education in CMHCs and with fewer psychiatrists working in centers to provide adequate role models, it would follow that even fewer psychiatrists will develop an interest in CMHCs in the future. In my March 1977 survey, only 25% of the centers in the United States had an affiliation with a medical school, whereas 32% had an affiliation with a nursing school, 30% with a clinical psychology school, and 37% with a social work school.

What are some of the factors that may be influencing this trend? Initially, NIMH mandated that CMHCs must be directed by psychiatrists; this was later broadened to include most mental health professionals. A larger available pool of other mental health professionals, the comparatively smaller pool of psychiatrists with skills and interests in administration, and the relatively lower salaries needed to recruit other professionals may have been factors leading to the relatively rapid replacement of the psychiatrist director by other mental health professionals. Many psychiatrists who in the past preferred to remain actively involved in center programs are now finding professional activities outside centers more attractive and the milieu inside centers less engaging and are entering the private sector. Because CMHC psychiatrists have traditionally been involved in independent salaried settings, they find leaving full-time CMHC positions easier than do other mental health professionals who have not worked in such settings. As the impact of psychiatrists on CMHCs is diminished, they may often feel that they are being underutilized and relegated to the task of signing prescriptions and medical records. They may perceive increasing hostility in the program staff and less control over their environment, and they may therefore turn their energies elsewhere.

Medical schools have often been reluctant to sponsor and operate CMHCs. Centers are often organizationally complex, fiscally frightening, and administratively taxing to a department of psychiatry. Community boards and community groups must be listened to; goals and objectives cannot be developed unilaterally. Some CMHCs view the university as an elitist organization and as unable to sponsor or direct a relevant community mental health program. Sometimes this is the case; often it is not. However, this polarization may have a significant influence on universities and CMHCs loosening or severing their affiliations. If this is the case, medical students and psychiatric residents may well get the message that CMHCs are becoming increasingly less desirable settings for psychiatrists and psychiatry, and they will be even more inclined to avoid these settings when they begin their careers.

Possible solutions to this problem do exist. Medical schools in this country must become more involved with CMHCs, must tighten their affiliations whenever possible, and remain heavily committed. If this can be accomplished, residents will be reassured that psychiatrists do have meaningful roles in CMHCs and, one hopes, will later become associated with them.

Psychiatrists with administrative interests and abilities should be encouraged to become competent mental health administrators so that a larger pool will be available from which to select leaders of CMHCs. A widespread attitude that physicians make poor administrators must be confronted. Physicians without interest, training, or experience in leadership positions may make poor administrators, but those with interest, ability, and training prove to be very effective. Although many CMHCs are ably directed by nonpsychiatrist professionals, there is no reason to believe that they are inherently more capable. Universities and other educational institutions must make high-quality educational programs, fellowships, workshops, and seminars more readily available so that psychiatrists, during their residency or later, can add administrative and leadership skills to their areas of expertise.

If psychiatry is to reverse the trend of decreased involvement in CMHCs, the centers must become a recognized, essential, and respectable part of a pluralistic system of mental health care. The centers must take their appropriate place with other segments of the public sector and become integrated with and accepted by the private sector. This will not easily be accomplished because there is an increasing wave of distrust from the private sector, and perhaps vice versa, as the perceived territories of the CMHCs and the private sector begin to overlap. As long as CMHCs provided care only to indigent patients the problem was minimal, but since fee-for-service became involved CMHCs have frequently been seen as competitive and threatening to the fiscal survival of psychiatrists in the private sector. The psychiatric profession, inside and outside the CMHC system, must learn to work together if patients' clinical needs are to be met. Society will eventually demand that some form of high-quality mental health services be available to all, probably through some form of national health insurance that will almost certainly include CMHCs. If the assumption can be made that psychiatrists must be an integral part of any mental health delivery system, then the trend of decreased psychiatric involvement in CMHCs must be reversed.

Solutions may come from Congress, the American Psychiatric Association, or universities. Solutions may be forthcoming only with a major attitudinal change in the psychiatrists of this country. Only when psychiatrists are willing to assume their responsibility for involvement in all segments of mental health

care—the private and public sectors, in both leadership and clinical positions—will their return to CMHCs be evident.

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Special Issue of *AJP* to Feature Depression and Alcoholism

A special issue of the *American Journal of Psychiatry* will be published in mid-April 1979. This issue, which will be distributed to all subscribers and is in addition to the regular April issue, will consist of a collection of articles on the subjects of alcoholism, depression, or both. Articles will deal with such topics as the treatment of depression with imipramine and ECT; bipolar illness: factors in drinking behavior; physical activity and plasma cyclic AMP in manic-depressive patients; childhood depression; life events at the onset of bipolar affective illness; psychiatric problems in women at alcoholic detoxification; lithium maintenance; efficacy of drugs, and psychotherapy in the treatment of acute depressive episodes; and life events, coping, stress, and depressive symptomatology.

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Impressions of Psychiatric Problems and Their Management: China, 1977

BY JOHN S. VISHER, M.D., AND EMILY B. VISHER, PH.D.

The authors, who visited the People's Republic of China in October 1977, describe the treatment methods used there, particularly in the Shanghai Mental Hospital, a 1,000-bed hospital for acutely ill patients. Psychiatric treatment there appears to be a blend of Western pharmacology, traditional Chinese medicine, and group therapy. The authors also speculate on the possible cultural sources of stress-related and interpersonal problems, provide a review of the literature regarding psychiatry in modern China, and outline the history of psychiatric treatment there.

IN OCTOBER 1977 we had the opportunity to visit the People's Republic of China. During our visit we spent a morning at the Shanghai Mental Hospital, visited general hospitals specializing in both Western and traditional Chinese medicine, saw neighborhood and factory dispensaries and clinics, and made inferences about the present status of mental health care in China. We also speculated about sources of stress in the population and the preventive techniques that may have a role in reducing the number of institutionalized mentally ill people in China.

PSYCHIATRIC LITERATURE

In recent years a small number of articles have described visits to China that were in some respects similar to ours. In May 1977 Leung and associates (1, 2) and Lowinger (2–4) reported on their visits to China. Leung and associates (1) concluded that mental health issues are managed through total support systems, usually involving family and work groups. Only the most serious illness comes to the attention of professionals. Lowinger (4) was impressed with the absence of evidence of neurosis in China and noted that such social and medical problems as drug addiction, alcoholism, prostitution, and venereal disease appear to have been virtually eliminated.

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In 1975 Walls and associates (5) wrote about a visit to China in 1973. One of these authors, L.H. Walls, is fluent in Mandarin, and the group was permitted to move freely without the constraints of translation difficulties. In 1973 David Ratnavale (6) also discussed a visit to the Shanghai Mental Hospital. His observations were in most respects consistent with our own.

A particularly valuable paper on the prevention and treatment of mental illness in the People's Republic of China was published in 1974 by D.Y.F. Ho (7), who is in the Department of Clinical Psychology at the University of Hong Kong. Dr. Ho has studied the history of psychiatry and the current status of the profession in China firsthand. He was impressed with the development of new methods to deal with emotional problems independent of traditional medical systems. He stated, "They have demonstrated what can be done when human resources are utilized to the fullest. They have shown that dynamic programs of treatment and prevention can be initiated when the whole society is mobilized, without having first to wait for more professionals to be trained" (7).

In 1973 Ruth Sidel (8) also discussed her experiences on two trips to the People's Republic. She pointed out that a major element in treatment there is the encouragement of psychiatric patients to feel that they are part of a force larger than themselves, namely, the Revolution. She said that patients are urged to take an optimistic view of their problems and to fight to understand and overcome their illness, not only for their own benefit but also for the sake of the Revolution.

HISTORY OF PSYCHIATRY IN CHINA

According to Ho (7), before Liberation, as the Chinese refer to the period before 1948, the mental health sciences were rudimentary; there were only a few small mental hospitals. Apparently there were only 4 in all of China, with a total of about 1,000 beds. The mentally ill were jailed, persecuted, or allowed to wander in the streets, where they often died of starvation and cold. After the Revolution, psychiatric care facilities and the number of psychiatrists expanded rapidly. In 1975, according to Walls and associates (5), there were about 60,000 psychiatric beds in China, located in 30 hospitals—2 for each province. Walls and associates also estimated that there were about 3,600 doctors specializing in psychiatry in China in 1975.

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A movement to humanize the mental hospitals, in some ways comparable to Pinel's work in Europe, took place between 1948 and 1958. Chains, isolation, and psychosurgery were largely eliminated. The hospitals began to use a combination of Western and traditional medical methods, emphasizing milieu and group experiences and including productive work as part of hospital treatment programs. An open-door policy was adopted, and efforts were made to "transform the hospital into a family-like institution so that patients can lead normal lives" (7). The hospital was seen as a battleground for the struggle against illness as well as a classroom for learning and practicing Communism. Family members were encouraged to become involved in the treatment, and great emphasis was placed on the patient's self-reliance. Patients were also encouraged to act as therapeutic agents for other patients (7). Outpatient therapy performed by psychiatrists or mental health workers does not exist in China as it does in the United States, although there are hospital-connected aftercare programs.

MANAGEMENT OF STRESS-RELATED AND INTERPERSONAL PROBLEMS

"Problems of living," personality disorders, marital conflicts, and interpersonal difficulties, which occupy so much of the time of outpatient mental health workers in the United States, are not seen in Chinese medical systems. These difficulties are handled by the extended family or by the work group, the neighborhood group, or the Revolutionary Committee.

It seems that people with physical or psychosomatic symptoms are treated in the regular medical system, in which patients can choose either Western medicine or traditional Chinese medicine. We heard reports that Western medical doctors might prescribe a tranquilizer such as diazepam, while traditional Chinese medicine would offer treatment techniques that include acupuncture with electrical current in the needles, moxibustion (a method of heating the acupuncture needle with burning wads of smoking incense), and "cupping," which involves external stimulation over painful shoulders or other joints. Traditional methods also include the use of herbal medicine. In this instance large packages of dried herbs or teas selected from more than 1,000 varieties of plants are carefully blended according to a prescription that takes into account the mythology regarding the emotional functions of each human organ.

The hospitals usually try to combine both Chinese traditional therapy and Western medicine whenever possible, but we learned that Chinese medicine is used more often for chronic illnesses and vague, nonspecific complaints. Hospitals varied according to the degree to which they used one or the other type of therapy. Often they used both simultaneously. "Cures" were attributed to Chinese medicine in medical conditions that were self-limiting or when palliation and calming effects were all one could provide, e.g., in a terminal cancer ward. As far as we could determine, no systematic effort is made to compare the different approaches.

SOURCES OF CULTURAL STRESS

It is interesting to speculate on possible sources of stress in Chinese society. Obviously, we were able to make only superficial observations in this area. In addition to the stress brought on by the fact that politics pervades every aspect of life in China, the recent changes in political structure, and changes in age-old systems and beliefs, we would add the stress of living so closely with others. Literally one is almost never alone in China. Crowds are everywhere, and large families live in cramped quarters like the two-bedroom, bath, and kitchen apartment we saw in Shanghai that housed 7 occupants who ranged from a 3-year-old child to an 82-year-old father-in-law.

Both premarital and extramarital sexual relations are illegal in China, and we were told there was no sex education aside from occasional high school hygiene classes. One wonders what is done with sexual tensions that have no apparent outlet until marriage, which is delayed to age 26 for women and 28 for men.

The competitive urges that certainly exist are expressed in sports and in working for the country. However, personal striving for advancement and personal wealth or security are considered no longer necessary because of the new social order. Workers strive for achievements that benefit the work group and result in increased production. "Model workers" are publicly acknowledged, and production records are posted so that everyone can see them. One holds his job for life and has little or no opportunity for change in either position or geographic location. One evening a worker who could speak a little English told us he hated his job. It would seem that the inability to change jobs except at the suggestion of a doctor would cause considerable stress.

Closeness between men and women, even in marriage, does not seem to be the norm. The government sometimes separates couples by means of geographically separate job assignments for long periods of time. The changed status of women, virtually all of whom work outside the home in neighborhood workshops or state factories from the time they graduate from middle school until they retire at age 50, may cause some stress. Husbands are expected to share the household chores and child care. According to our observations, many do so to an impressive extent. However, there may be some resentment. One man told us he did not want to marry because of the amount of time household chores would take. He preferred living with his mother, who did most of the housework.

Every Chinese person is a member of one or more overlapping small groups of 15 to 20 people. These groups spend 2 or 3 hours a week in meetings at which

the works of Marx, Lenin, and Chairman Mao and the latest principles of the Central Committee in Peking are studied in great detail. The discussion at these meetings often takes the form of "criticism-self-criticism" sessions, which we did not observe but which apparently are very much like leaderless encounter or therapy groups. The criticism is often very direct, and the person criticized is expected to respond with a frank confession of negative actions or thoughts that may have in some way contributed to the difficulties being discussed. There is much group pressure to conform to expected norms and to change deviant behavior and thoughts. A Chinese person is a member of a group first and an individual second.

The small group is obviously important from the standpoint of social control and communication of political ideas and guidance from above; for some individuals this may cause stress. On the other hand, the group can also offer a supportive and even therapeutic situation in which interpersonal conflicts, neighborhood problems, and job-related difficulties can be discussed. Feelings can be expressed and accepted, and the group rewards individuals for "good behavior." Neighbors and fellow workers do not stand aloof from the individual in trouble—as long as the individual accepts the group's guidance and conforms to the group ethic.

SHANGHAI MENTAL HOSPITAL

Severely mentally disturbed individuals are sent to mental hospitals in their geographic locale. We spent a morning at 1 of the 2 mental hospitals in Shanghai, a city of 11-12 million people. This hospital was established in 1958 and has 1,000 beds and 14 wards. The other hospital in Shanghai has approximately 1,200 beds and serves chronic cases only. The Shanghai Mental Hospital we visited treats acute cases only; the average stay is 2 or 3 months. Equal numbers of male and female patients are admitted, and there are no waiting lists for admission. The outpatient department handles about 500 outpatients every day, most of whom have been discharged from the hospital. The staff consists of 754 people and includes 86 doctors, 295 nurses, and 42 medical technicians in the X-ray department, laboratory, and pharmacy.

The hospital is a large complex of 3- or 4-story buildings set in a parklike compound behind a high wall. This layout is typical of factories and public institutions in China. We were impressed with how modern and clean it looked in contrast to other hospitals and public buildings we had visited. The wards were airy and light with long lines of beds in precise rows. We saw no personal articles or decoration of any kind in the patients' sleeping areas, although there were political slogans and schedules on the freshly painted walls. The furniture was very simple. One section at the end of each of several large unlocked wards we visited was for more disturbed patients. We saw a few patients in locked isolation rooms and 1 or 2 lightly restrained in beds. There were more staff on the disturbed wards. The staff were dressed in clean but unpressed white hospital coats and smocks and white surgical caps. Patients were dressed in ordinary street clothing; the women were dressed in more colorful clothes than the usual street clothing, which tends to be drab and plain. Nearly all of the patients were in the day rooms; they were not allowed in the sleeping quarters during the day. All of the patients were busy at activities that looked as though they had been arranged for our visit. Some were playing cards, some were assembling matchboxes or paper flowers. These tasks were work production projects from a nearby factory; we were told that the revenue from this labor reverted to the patient fund.

In the disturbed wards a few patients were posturing or gesturing, and nurses reacted quickly when a patient became excited. One young manic woman sang several songs for us in Chinese and then asked in perfect English, "Are you from Pakistan?" Another patient tried to join our group as we walked along, but she was spotted by a staff member who gently led her away. Although no patients seemed drugged or toxic, some seemed apathetic; almost everyone was quite docile and controlled. Doctors pointed out patients with diagnoses of depression, schizophrenia, and senile dementia in the disturbed wards. Most of the patients we saw were young. There were no children; we were told there is no need for children's psychiatric facilities any longer.

Relatives bring patients to the hospital with the help of their neighborhood or work groups. Each patient is given a thorough checkup, and a complete history is taken. (Compulsory involuntary admission of patients does not occur in China except in criminal cases.) The doctor makes a decision regarding hospitalization after he or she makes a diagnosis. In complicated cases consultation and/or a period of observation may be required before a diagnosis can be made. Cooperative patients are usually treated as outpatients. However, doctors told us that mental patients usually cannot cooperate because they do not admit that they have mental problems and refuse to take medicine. These patients are first taken to small wards where they are isolated because they are anxious and confused. The doctors use Western medicine to treat these patients. In 7-10 days they let them join in activities with other, less disturbed patients.

Eighty percent of the patients in the hospital are diagnosed as schizophrenic; these are primarily young people 14 years old and older. The number of young schizophrenic patients is steadily increasing, and the doctors are baffled as to the reason. They do not consider societal stress as a precipitant. Some patients are diagnosed as having "neurasthenia." These patients exhibit symptoms we would consider depressive, but they are usually treated as outpatients. Patients with epilepsy are also treated as outpatients unless they have an associated psychosis. Epileptics make up only

.1% of the total number of inpatients. Manic-depressive patients comprise only 1.5%.

Various treatment modalities are used. We observed four male patients being treated with acupuncture. They were seated around a small box providing low voltage current to wires leading to needles in their heads, necks, or limbs. In another room a patient was conferring seriously with a doctor and a nurse on a one-to-one basis. Elsewhere there was a group session with 20 or 30 patients and a nurse seated in a square, with one patient standing and telling the history of her illness and her delusions while the others held Chairman Mao's "little red book" in hands folded carefully on their laps. We also saw patients receiving their herbal tea.

We were told that there is a therapeutic milieu in which patients and staff are regarded as comrades in arms fighting in the struggle against mental disease. Patients and staff also work together to plan programs and accomplish the necessary hospital chores.

In the acute phase of illness Western medicine is used, and a combination of Chinese medicine and Western medicine is employed later. The doctors report that this combination has resulted in an overall improvement in effectiveness of treatment. When patients are hallucinating and highly disturbed, haloperidol, chlorpromazine, and perphenazine are used. Lithium carbonate is prescribed for manic patients. These medicines are combined with strong-voltage acupuncture twice daily. After 2 or 3 weeks, as symptoms improve, lower voltages are used and acupuncture treatments are less frequent. Combined treatments are given according to the judgment of the doctor, who emphasizes one modality or another. Apparently the hospital is not engaged in research to determine which modality works best, and there is no emphasis on keeping statistical records on the difference.

Special nurses organize patients into study sections, and each class lasts for about 1–2 weeks. The classes study Chairman Mao's philosophical works and articles written on how to handle "the contradictions among the people," which refers to interpersonal difficulties. The doctors also provide the patients some basic knowledge about psychosis so they can analyze their own case or course of disease. The patients are said to come to understand the reasons for their psychosis, which is seen as "subjectiveness and one-sidedness." The doctors believe that patients have arrived at wrong conclusions because they have not made thorough investigations. Knowledge of psychosis, therefore, helps the treatment and also helps patients know how to correctly handle the "contradictions" that might occur after they leave the hospital, thereby preventing recurrence of their illness. The hospital personnel feel that patients are harmed by being in the hospital too long and that patients should be discharged as soon as possible and looked after by society at large. After discharge the doctors think it is better for patients to be back at work even if they have not fully recovered and need to continue medication.

Doctors at the hospital stated that one of their important tasks was to help families and neighborhood groups understand the needs of the mentally ill and fit them into life at a level where they could continue to make a contribution to society. We heard of a case in which a fellow worker was assigned as a companion to someone who was psychotic. This worker lived and worked with the individual to help her conform to acceptable standards.

Hospital doctors often pay visits to patients living at a distance from the hospital who have been discharged to help them consolidate the effectiveness of the treatment and to reduce recurrences of illness. Outpatient visits are scheduled every 2–3 weeks following discharge, and doctors from the inpatient department will also visit the family at home if necessary. Before leaving the hospital, patients are given guidance about how to take medicines and how to manage their daily lives. When patients are considered able to return to work the doctors sometimes make suggestions to the leaders of the patient's work place for changes in schedules or jobs.

Since the Cultural Revolution (1966–1969), the hospital has been sending medical teams on regular tours through the factories and countryside to deliver medicine and medical care, to teach prevention of mental disease, and to train a number of barefoot doctors or paramedical personnel.

Patient care is paid for by the place where they work. The cost is 170 yuan a month, in contrast with the average rate of pay, which is 50 yuan a month.[1] However, patients pay the cost of their food in the hospital. For the first 6 months of hospitalization the patient's full work pay is continued, and after 6 months the patient receives 60% or 70% of his or her work salary, depending on seniority. Payment for nonworking patients is partly covered by the place of work of their spouse, and retired people receive free medical care.

Doctors in China receive less formal training than doctors in the United States. We were told that most of the doctors in the Shanghai Mental Hospital were trained in the Shanghai Medical College before the start of the Cultural Revolution in 1966. The length of training in medicine at that time was 5 years; now it is 3 years. Students in the medical college before 1966 studied the basic medical subjects and spent 2 weeks in a psychiatric hospital. After graduation, doctors who were assigned to work in the mental hospital were required to have 1 year of practice under the supervision and guidance of other doctors at the hospital. Under the shortened program, the study of psychiatry takes only 1 week, so after graduation doctors are required to have a longer period of hospital practice under supervision. Some doctors are selected from among the nurses with "rich experience"; these

[1]It is difficult to estimate the equivalent of 50 yuan in U.S. currency. The fact that a bicycle costs 200–300 yuan might provide a sense of its relative value.

nurses are required to have 1½ years of further study to become doctors.

COMMENT

Most acute cases of mental illness in China are effectively treated in large mental hospitals similar in many respects to our large state hospitals of 15 or 20 years ago. Treatment methods are a mixture of Western psychotropic medication and traditional Chinese medicine, including acupuncture, milieu, group, and activity therapy. Aftercare is the responsibility of hospital outpatient clinics, and community care by families and work groups provides continuing supervision and support. The medical care system offers care and treatment for physical illnesses related to stress, while life crises and problems of living are dealt with by the individual's neighborhood or work group.

Shared goals, values, and aspirations reduce the need to choose from among a variety of beliefs and lifestyles, but other personal, political, and cultural stresses apparently exist and result in emotional conflicts and symptoms for a number of individuals. However, the Chinese social experiment has resulted in an improvement in the quality of life for the vast majority of the Chinese people. Great progress has been made to provide all of the people with food, clothing, shelter, education, and low-cost or free medical care.

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Differential Bonding: Toward a Psychophysiological Theory of Stereotyping

BY CHARLES A. PINDERHUGHES, M.D.

The author uses the concept of affiliative-affectionate bonding and the concept of differentiative-aggressive bonding to answer questions regarding the human tendency to stereotype. He states that the human processes of affiliation, introjection, identification, and affection appear to be psychophysiological elaborations of approach-seeking physiology and that differentiation, projection, and aggression appear to be psychophysiological elaborations of withdrawal-avoidance physiology. He also points out that some measure of stereotyping and paranoia may be necessary because differential bonding appears to be essential for adaptation.

STEREOTYPING IS UBIQUITOUS as a central component of group-related thinking and behavior. Among social scientists a stereotype is defined as a simplified and standardized conception or image invested with special meaning and held in common by members of a group. The prefix *stereo* is from the Greek word for "solid," and the word "stereotype" was originally applied to a process for producing solid metal printing plates from many separate pieces of type.

Allport (1, pp. 28–66, 106–137, 184–199) defined a stereotype as "an exaggerated belief associated with a category. Its function is to justify (rationalize) our conduct in relation to that category" (1, p. 191).

Stereotypes may be false or may contain some element of truth. They may be favorable or unfavorable. They may grow and persist in defiance of all evidence and may interfere with even the simplest rational judgments. All human minds must think with the aid of categories. It is not possible to differentiate objects or to handle the countless stimuli, impressions, relationships, and other experiences of life without doing so. Allport (1, pp. 20–22) indicated that the process of categorization has five important characteristics: 1) It forms large classes and clusters for guiding our daily adjustments. 2) It assimilates as much as it can to the cluster. 3) The category enables us quickly to identify a related object. 4) The category saturates all that it contains with the same ideational and emotional flavor. 5) Categories may be more or less rational.

Ordinarily, at two years of age children play with one another without regard for sex or race or other categories that they subsequently learn to use. Between the ages of four and seven they generally establish preferences for play with one sex. A favorable stereotype is applied to the preferred sex and an unfavorable one reserved for the other. Children who prefer girls may say they are gentle, kind, and loving and that boys are too loud, rough, and mean. Those who prefer boys may say they are active, enterprising, and stimulating and that girls are too quiet, uninteresting, or silly. In adolescence, youths who earlier had these preferences and stereotypes often reverse them. Learning and cultural factors play an important role in these developments. Ordinarily, association with one sex is facilitated more than association with the other sex.

During adolescence, if not earlier, many children in a heterogeneous society may start to show racial, religious, class, caste, and ethnic group preferences and to apply stereotypes. Learning and cultural factors play an important role in these developments as well. Association of members of the same group generally has been facilitated more than association between members of different groups. Parenthetically, it may be noted that wherever a specific group or stereotype is mentioned in this paper, the content may be applied equally as well to other racial, ethnic, religious, national, caste, or class groups and stereotypes.

CATEGORIZING AND STEREOTYPING

I have a number of questions in connection with these observations:

1. If we accept Allport's definition of a stereotype as an exaggerated belief associated with a category, why is the belief exaggerated when there seems to be equal opportunity for more realistic appraisal?
2. Why do people who take pride in their rational judgment hold so tightly to false beliefs in defiance of all evidence?
3. In the process of categorization, why does the mind assimilate as much as it can to the cluster, including some characteristics that are not present in the category?

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4. In the process of categorization, why is everything in the category saturated with the same ideational and emotional flavor?

5. Since sexual, racial, religious, class, caste, and ethnic stereotypes are not present at an early age but appear, intensify, or decline in the course of development, what relationship do they have to developmental processes and stages?

Although social psychologists, social psychiatrists, and sociologists have studied stereotypes in social and psychological fields extensively, they provide no answers to these questions. Psychoanalysts studying in psychological and social fields (2–7) have developed insight into the dynamic interaction of thoughts, feelings, and behavior associated with stereotypes. They have thrown light on the primitive and irrational mental processes through which all individuals pass and the persistence of these into adult life with dynamic variations in extent, intensity, and form under different conditions. Although they have not answered the questions, they have offered many hints that the answers to the questions lie deeper than we have tools to probe.

Although relatively well-developed psychoanalytic, psychological, and social psychological concepts of stereotypes have been produced, there has been virtually no development of psychophysiological concepts of stereotypes. We have very little ability to study the psychophysiology of a feeling and are virtually unable to study the psychophysiology of an idea. At psychophysiological levels we have no reliable information about the nature of stereotypes. In this absence I would ask what observations and ideas can aid those of us who suspect that part of the answer to our questions lies in somatic terrain.

My readings in biology over the years have been helpful in trying to answer this question. Studies of fish, birds, and animals led Lorenz (8) to perceive that animals which form attachments or bonds often show alternation of aggression and closeness and display aggression to one object and closeness to another. Closeness to a mate often follows aggression to a neighbor, and a bond to one may be enhanced by aggression to another. Lorenz wrote,

> The principle of the bond formed by having something in common which has to be defended against outsiders remains the same, from cichlids defending a common territory or brood, right up to scientists defending a common opinion and—most dangerous of all—fanatics defending a common ideology. In all these cases aggression is necessary to enhance the bond. (8, p. 190)

Lorenz conceptualized bonding in affiliative terms but not in terms of aggression. Biological research has also focused on attachments to territory as well as to mates and generally does not deal with aggressive behavior as bonding phenomena. Research on imprinting processes has also focused on development of bonds primarily in terms of following-seeking-affiliative behavior, as did Bowlby (9) and the Harlows (10) in their studies of attachment in humans and monkeys, respectively. Although approach-avoidance researchers seldom focus on bonds, Schneirla (11) noted that "biphasic mechanisms facilitating approach and withdrawal adjustments are present in all functional systems, from sensory to motor, in multicellular animals."

Schur and Ritvo (12) noted that Freud drew on his biological research experience in using withdrawal-avoidance concepts in his unpleasure principle and approach-seeking concepts in his pleasure principle. They noted that an inborn apparatus guaranteeing approach and withdrawal responses is necessary for survival. Freud's concept of links that become established between mnemonic images of perceptions and memory traces of body excitation (13) has aided the conceptualization of an organic relationship between body physiology and the external world. Schilder's work with the body image (14) and Deutsch's work with conversion process (15) made additional contributions to my conceptualization of the attachments human bodies develop to people, possessions, and ideas.

Human psychophysiological processes of affiliation, introjection, identification, and affection appear to be psychophysiological elaborations built on the bedrock of approach-seeking physiology. Differentiation, projection, and aggression appear to be built on the bedrock of withdrawal-avoidance physiology. Concepts of this kind are also consistent with Hess's division of body and behavioral organization into "ergotropic" and "endophylactic trophotropic" patterns (16).

DIFFERENTIAL BONDING THEORY

The above observations and ideas are consistent with psychosomatic and psychosocial concepts I have worked on since 1950. They were concisely expressed in 1971 (17) and in 1973 (18) as a series of hypotheses. Some of the hypotheses can be paraphrased as a theory of differential bonding or discriminatory physiological linkage that may offer a useful approach to my five unanswered questions.

Hypothesis 1

The physiological patterns that mediate approach-affiliative-affectionate behavior and those which mediate avoidance-differentiative-aggressive behavior are opposing physiological patterns that impose on all thinking and behavior a drive to dichotomize—to seek, join, introject, and make part of oneself on the one hand and to avoid, differentiate, and oppose on the other.

Hypothesis 2

The drive to dichotomize universally results in fluctuating physiology and ambivalence in early behavior with other persons and objects. Ambivalence can be

resolved only by differential physiological bonding. Affiliative-affectionate physiology is linked to mental representations of one object, while differentiative-aggressive physiological patterns are linked to mental representations of another. Such discriminatory physiological linkage results in greater constancy of relationships and of physiology.

Developmentally, these discriminatory physiological linkages become recognizable in diverse parts of the world by the eighth month after birth (19). The capacity to relate differentially to the familiar and the strange reflects early differential bonding. Subsequently, differential bonding may develop toward the images of any persons, groups, material things, sensory stimuli, body parts or processes, ideas, patterns, modes, or styles that may have representation in the CNS. (A physiological bond to the mental image of an object may be subjectively experienced as a physiological link to the object itself, although this is, of course, only an illusion.)

Differential bonding is generally incomplete, and reversion to ambivalence often occurs. This provides potential for a shift of bonds, for mourning and other separation processes, and for realignment and reorganization of relationships. Differential bonding may also show shifts with changes in the field with which a given image is associated. Two ethnically different Americans who relate as members of different groups when they meet in the United States may relate as members of the same group while they are engaged in a common interest, while they face a common adversity, or when they meet in a foreign country.

All object-directed behavior and all mental processes depend on the intricate dynamics of differential bonding. Compared with differential bonding in other animals, these processes in humans involve a greater range of images, greater capacity to bond and retain diverse images, greater capacity to bond images in various patterns, and greater capacity for sequential shifting of bonds from one image to another.

Each animal has its own characteristic potentials for differential bonding. These unfold and interact with experience in ways that define affiliations, aversions, friends, and enemies. Imprinting, grouping processes, territorial attachment, and mating behavior can be examined and formulated in terms of differential bonding, with affiliative and differentiative components differing in strength and in the amount of focus or diffusion.

Hypothesis 3

The object of the affiliative-affectionate linkage is aggrandized and the object of the differentiative-aggressive linkage is renounced as paired paranoid processes accompany the discriminatory physiological linkages. These processes are paranoid in the sense that they produce false beliefs based on projections that aggrandize objects of identification and denigrate renounced objects.

Paired dichotomous concepts, such as good-bad, right-wrong, in-out, high-low, front-back, and white-black, reflect the drive to dichotomize, but these regularly have one member of the pair preferred and the other devalued depending on the projections involving each member of the pair. Thus, differential projection is generally associated with differential bonding.

Hypothesis 4

All unambivalently constant relationships are basically paranoid, as objects of affiliative-affectionate bonds are aggrandized and objects of differentiative-aggressive bonds are denigrated.

Hypothesis 5

Groups are characterized by affiliative physiological linkages between group members or to common territory or common beliefs and by differentiative physiological linkages to common renounced targets. Nonpathological group-related paranoias are commonly found in groups. They bind together and socialize the group members and are to be distinguished from idiosyncratic pathological paranoias that isolate and alienate the believer.

Hypothesis 6

Behaviors disruptive to one's social group (such as sexual arousal, hostile discharges, excretory activity, and sudden loud noises) are discouraged in the group context. Images of these behaviors are likely to be renounced, repressed, and projected in social contexts. The association of mental representation of these behaviors with differentiative linkages facilitates their association with outsiders, reinforcing differentiative-aggressive bonding to outsiders and offering group members illusions of purity and superiority that reinforce affiliative-affectionate bonds between group members.

Hypothesis 7

Positive and negative feedback in life experiences determine what becomes associated with affiliative linkages and given high value and what becomes associated with differentiative linkages and given low value.

Hypothesis 8

Because of common linkages to affiliative physiological patterns, the more valued component of each paired relationship is associated with the more valued component of other paired relationships. Thus, the white, the high, the in, the front, the right, the head, the top, and the free are associated with one another in mental life. The dark, the low, the out, the behind, the left, the foot, the bottom, and the repressed are similarly associated in human minds because of common linkages to differentiative-aggressive physiological patterns.

Hypothesis 9

Once falsely positive or falsely negative attitudes and beliefs about people develop, relationships, social

structures, and the environment are modified wherever possible into a contrived reality that expresses, perpetuates, and seems to validate the false beliefs.

THE ANSWERS

On the basis of this speculative excursion into territory where psychology, physiology, and biology come together, it is now possible to construct some speculative answers to the five questions formulated toward the beginning of this paper. I will deal first with question 2: Why do people who take pride in their rational judgment hold so tightly to false beliefs in defiance of all evidence? The beliefs are organically linked to the body in the body image; to change them would thus introduce an instability in physiology that must be resisted. Clinicians evaluating somatic complaints often hear that some important trusted person had "deeply hurt" the patient or "caused great pain" by behavior that altered cherished beliefs and illusions. Denial is commonly used in attempts to ward off somatic discomfort associated with the perception of an unpleasant reality. Another answer to question 2 is that valued relationships with fellow believers would also be threatened by a change in belief.

Question 4 asked, In the process of categorization, why is everything in the category saturated with the same ideational and emotional flavor? In the course of relating in an unambivalent and physiologically stable way to any mental representation, including the mental representation of a category, it is necessary to relate either with affiliative-affectionate physiological linkage or with differentiative-aggressive physiological linkage, but not with both. Thus, categories linked in the body image to affiliative processes will be saturated with affiliative ideational content and affectionate emotional flavor. Categories linked in the body image to differentiative processes will be saturated with differentiative-aggressive content and flavor.

Questions 1 and 3 may be taken together: Why exaggerate beliefs when realistic appraisal is possible and why does the mind assimilate as much as it can to the cluster, including some characteristics that are not present in the category? If each of us has affiliative and differentiative twins in our physiology, minds, relationships, and outside world, we can expect that there will be aspects of our bodies, aspects of our minds, aspects of our relationships, and aspects of the outside world that we will value highly and have linked to affiliative physiological processes. Among the many such affiliative (introjected) categories with which we associate ourselves, easy transfers may take place from one to another because in the body image they are all united as a part of our acknowledged identity. There will also be aspects of our bodies, minds, relationships, and outside world that we will renounce, assign negative value to, and have linked to differentiative physiological processes. Easy transfers may also take place among the many such differentiative (projected) categories from which we renounce association because in the body image they are all united as part of the differentiated identity of outsiders. Thus, there is a psychophysiological tendency to impute to any category with which one identifies characteristics from other categories with which one identifies. There is also a twin psychophysiological tendency to impute to any category that one renounces and projects characteristics from other categories that are renounced and projected. Under these circumstances, exaggerated beliefs and the presence of characteristics that do not belong in the category can be expected regularly in the process of categorization.

When a group of people is excluded from social interactions as unacceptable, images of those people tend to be linked in unconscious thinking processes with those ideas, feelings, and body products which tend to be excluded from social interactions as unacceptable. Contact with the socially excluded people may be accompanied by a sense of something forbidden, bad, and possibly dirty or dangerous. Unconscious sexual, aggressive, and other repressed feelings may tend to emerge into consciousness in their presence, and stereotypes of the excluded people may contain all of these elements.

This brings us, finally, to question 5: What relationship does the development of stereotypes have to developmental processes and stages? Discriminatory physiological linkages occur at different rates at different times. They proceed more rapidly at points in development where psychophysiological instability has increased and can be corrected and stabilized by differential bonding. Thus, it occurs at a rapid rate in early childhood during resolution of the oedipal conflict, when mental representations associated with one parent are linked primarily to affiliative physiological processes and mental representations of the other parent are linked primarily to differentiative physiological processes. More rapid discriminatory linkage also takes place in adolescence in association with identity formation and participation in new grouping processes. Marked psychophysiological instability commonly precedes and perhaps activates increases in differential bonding and in the associated paranoia. Increased differential bonding and increased paranoia appear to help stabilize the psychophysiological instabilities that occur during healthy development as well as those which occur in acute pathological states. Stereotypes change with shifts in identifications and group relationships or as expressions of defensive operations against a shift in identity or relationships.

Data from many sources strongly support the conclusion that human beings are generally paranoid as well as sometimes wise. In fact, humans use their intelligence most frequently in support of their various nonpathological paranoias. The paranoias often gain expression in contrived relationships and in contrived social, political, economic, and educational structures. Stereotypes constitute one expression of them. These paranoias are not accessible to reason, and attempts to

alter them lead to confrontations and, often, to violence.

We should come to terms with the fact that healthy and normal people function with paranoid processes, especially in their group-related behavior. If we become aware that the processes with which we are involved are paranoid, we will be in a better position to cope with and manage them and to clarify what future generations need to do about them. Certainly the exploration of these processes in family therapies has helped many family members to better understand and better manage them. For example, I have described a family with frequent violent interactions (20) in which the hysterical wife had renounced control and discipline in her search for freedom of spirit and expression. She experienced her obsessional husband's disciplined and structured life as an attempt to stifle her and deprive her of life. The obsessional husband had renounced and warded off emotions and spontaneity. He perceived the hysterical wife as primitive, sick, uncontrolled, irresponsible, manipulating, and evil. Each marital partner was relating to a renounced and repressed aspect of himself or herself that had been projected on the other, who helped to maintain and reinforce the repressions and projections. Understanding this process and perceiving the mate as an aspect of self undermined the projection, reduced the conflict, and improved opportunities for better management.

It may well be that some measure of stereotyping and paranoia is necessary for the integrity of our bodies because differential bonding by means of discriminatory physiological linkages appears essential for adaptation and because stereotyping and paranoia go hand in hand with differential bonding. The biological process of differential bonding offers stability and certainty at the expense of objectivity and validity, producing a biologically mature organism that may know it is right even when it is wrong. This, in my opinion, constitutes the main source of bias in human thinking and behavior and the primary basis for social conflict.

In appraising these ideas we do well to keep in mind Freud's statement:

> The deficiencies in our descriptions would probably vanish if we were already in a position to replace the psychological terms by physiological or chemical ones. It is true that they too are only part of a figurative language; but it is one with which we have long been familiar and which is perhaps a simpler one as well.
>
> On the other hand, it should be made quite clear that the uncertainty of our speculation has been greatly increased by the necessity for borrowing from the science of biology. Biology is truly a land of unlimited possibilities. We may expect it to give us the most surprising information and we cannot guess what answers it will return in a few dozen years to the questions we have put to it. They may be of a kind which will blow away the whole of our artificial structure of hypotheses. (21, p. 60)

Freud's statements still hold true. He learned, as we should, that the ideas we hold now are very likely to prove false or at least biased. This does not deter some of us from speculating and taking steps to develop hypotheses that seem consistent with the existing data, although they are not yet testable.

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Einstein's Creative Thinking and the General Theory of Relativity: A Documented Report

BY ALBERT ROTHENBERG, M.D.

A document written by Albert Einstein has recently come to light in which the eminent scientist described the actual sequence of his thoughts leading to the development of the general theory of relativity. The key creative thought was an instance of a type of creative cognition the author has previously designated "Janusian thinking." Janusian thinking consists of actively conceiving two or more opposite or antithetical concepts, ideas, or images simultaneously. This form of high-level secondary process cognition has been found to operate widely in art, science, and other fields.

ALBERT EINSTEIN was clearly one of the world's most creative scientists. Among his many accomplishments, the formulation of the general theory of relativity stands out as one of the most far-reaching scientific events in the modern era. As a new theory of gravitation, it embraced Newton's classic theory as a special case and allowed for extensive and grand conclusions about the universe as a whole. At the very least, the accomplishments and accumulated knowledge of modern astrophysics can be traced to this theory and the dramatic confirmations of its predictions in 1919 from data collected during a solar eclipse.

Because of his importance and the exalted nature of his accomplishments, many speculations have arisen about the roots of Einstein's genius and creativity (1–7). Much has been made of the rather remarkable but now generally known facts that Einstein learned to speak late, did not perform well in his early school years, and was not highly proficient in verbal skills throughout his life. Emphasis has been placed on the visual nature of his thinking, which he reported to his colleague Hadamard (8) and to Wertheimer (7). Because of Einstein's gentle and somewhat introverted personality and because visual thinking is often erroneously considered more primitive and more characteristic of childhood than verbal thinking, some have asserted that Einstein thought as a child thinks. This type of conclusion roughly coincides with many psychoanalytic formulations about creativity that postulate the regressive, primitive roots of creative thinking (9–11).

Tracing specific factors of creative thinking is highly complex and difficult, especially in science. Scientists rely so heavily on the contributions of their colleagues and predecessors and so much good scientific work derives directly from perseverance, memory, and logical capacity that it is hard to isolate and identify special kinds of thinking or skills in an individual discovery. Moreover, even when something is known about a scientist's personality, cognitive modes, and outlook, specific data are needed before it is possible to connect any particular personality factors or modes to a specific attainment or discovery. The assertions of childish, primitive thinking in Einstein's discoveries are especially based on surmise and speculation. Even the emphasis on visual and geometric thinking, plausible as it may be, derives only from Einstein's sketchy and general descriptions to Hadamard. As with most scientists, Einstein gave out very little information during his lifetime about the circumstances or specific sequences of thought connected with any of his attainments.

An important document has recently come to light, however. This document provides a direct and detailed account by Einstein himself of the actual thinking that led to his discovery of the crucially important general theory of relativity. Still unpublished in its entirety, it was written in Einstein's own hand in approximately 1919 and titled by him (in translation from the German) "Fundamental Ideas and Methods of Relativity Theory, Presented in Their Development." It was found after Einstein's death among his personal effects and includes an unusually explicit personal statement by a man whose writings are almost invariably scientific or impersonal. Even Einstein's autobiographical notes, written at age 67, consist primarily of an exposition of his theories (12).

The section I shall quote was subtitled by Einstein The Fundamental Idea of General Relativity in Its Original Form. Prof. Gerald Holton published other

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translated versions and portions of these passages in 1973 (5) and 1975 (13). The entire document is in the Einstein Archives at the Princeton Institute for Advanced Study. The section I shall quote was translated by Professor Holton and was made available to me by the executors of the Einstein Estate, Otto Nathan and Helen Dukas. The section begins with Einstein's discussion of considerations involved in his discovery of special relativity theory, the theory he expanded and applied to gravitation in his general theory of relativity. These considerations involve a seeming discrepancy of interpretation arising from the Maxwell-Lorentz theory explaining Faraday's work on electromagnetic induction:

> In the development of special relativity theory, a thought—not previously mentioned—concerning Faraday's work on electromagnetic induction played for me a leading role.
>
> According to Faraday, when a magnet is in relative motion with respect to a conducting circuit, an electric current is induced in the latter. It is all the same whether the magnet moves or the conductor; only the relative motion counts, according to the Maxwell-Lorentz theory. However, the theoretical interpretation of the phenomenon in these two cases is quite different. . . .
>
> The thought that one is dealing here with two fundamentally different cases was, for me, unbearable [*war mir unerträglich*]. The difference between these two cases could not be a real difference, but rather, in my conviction, could be only a difference in the choice of reference point. Judged from the magnet there certainly were no electric fields; judged from the conducting circuit there certainly was one. The existence of an electric field was therefore a relative one, depending on the state of motion of the coordinate system being used, and a kind of objective reality could be granted only to the electric and magnetic field together, quite apart from the state of relative motion of the observer or the coordinate system. The phenomenon of the electromagnetic induction forced me to postulate the (special) relativity principle.
>
> When, in the year 1907, I was working on a summary essay concerning the special theory of relativity for the Yearbook for Radioactivity and Electronics I tried to modify Newton's theory of gravitation in such a way that it would fit into the theory. Attempts in this direction showed the possibility of carrying out this enterprise, but they did not satisfy me because they had to be supported by hypotheses without physical basis. At that point there came to me the happiest thought of my life, in the following form:
>
> Just as in the case where an electric field is produced by electromagnetic induction, the gravitational field similarly has only a relative existence. *Thus, for an observer in free fall from the roof of a house there exists, during his fall, no gravitational field* [Einstein's italics]—at least not in his immediate vicinity. If the observer releases any objects, they will remain, relative to him, in a state of rest, or in a state of uniform motion, independent of their particular chemical and physical nature. (In this consideration one must naturally neglect air resistance.) The observer is therefore justified in considering his state as one of "rest."
>
> The extraordinarily curious empirical law that all bodies in the same gravitational field fall with the same acceleration immediately took on, through this consideration, a deep physical meaning. For if there is even one thing which falls differently in a gravitational field than do the others, the observer would discern by means of it that he is falling in it. But if such a thing does not exist—as experience has confirmed with great precision—the observer lacks any objective ground to consider himself as falling in a gravitational field. Rather, he has the right to consider his state as that of rest, and his surroundings (with respect to gravitation) as field-free.
>
> The fact, known from experience, that acceleration in free fall is independent of the material is therefore a mighty argument that the postulate of relativity is to be extended to coordinate systems that are moving non-uniformly relative to one another.

The happiest thought of Einstein's life as described here was the formulation that provided the foundation for the general theory of relativity. From the exposition, it is clear that this "happiest thought" was his underscored (here italicized) phrase, "*Thus, for an observer in free fall from the roof of a house there exists, during his fall, no gravitational field.*" Einstein was not referring to the analogic thought in the immediately previous sentence because, in earlier paragraphs, he had made it clear that he had had in mind for some time the idea of an analogy between electromagnetic induction and gravity. The analogic thought, in other words, was the formulation of the problem, and the underscored idea was the solution. It is important to emphasize this because the exposition indicates many factors pertaining to Einstein's capacity to make this discovery, such as his full and complete knowledge of theories and facts, his extraordinary intelligence and lucidity, his passionate devotion to solving the problem, and his emotional involvement in the substance of the matter—seen in his statement, "The thought that one is dealing here with two fundamentally different cases was, for me, unbearable." All this is worthy of attention and analysis, but Einstein's creative leap of thought is discovered here for the first time clearly specified, and it is this thought—the happiest one of Einstein's life—which requires explication. The leap of thought Einstein described is an instance of a psychological process I have previously (in 1969 and 1971) defined, discussed, tested experimentally, and designated "Janusian thinking" (14–18).

JANUSIAN THINKING

Janusian thinking consists of *actively conceiving two or more opposite or antithetical concepts, ideas, or images simultaneously*, both as existing side by side and/or as equally operative or equally true. In order to clarify the creative nature of this type of thinking, I shall first briefly review the research that led to its discovery and then return to show how it is manifest in Einstein's dramatic formulation.

Janusian thinking was discovered through empirical

studies of the creative process consisting of extensive and intensive psychiatric research interviews focused on creative work in progress (19), analyses and reconstructions—both statistical and psychological—of literary creative processes of the past with predictions assessed in other types of research interviews (14) and experimental studies (17, 18). In the research interviews focused on work in progress, I regularly and systematically met with creative individuals over an extended period—usually months or years—and, in a collaborative effort (not a treatment or a psychotherapy), we explored the thoughts, feelings, fantasies, dreams, and events in their lives that were connected to the creative work they were engaged in at the time.

On the basis of these interviews, which total 1,665 hours to date, new experimental techniques were constructed and particular controlled experiments designed to elicit cognitive patterns were performed, both with the individuals who had participated in the interviews and with 149 other creative individuals and control subjects (17, 18, 20). Although differing criteria were used for assessing the creativeness of the individuals involved in the various researches, selection of individuals for the interview series was based primarily on two factors: high creativity ratings by peers and endorsement by society at large. For the creative writers and scientists who were my interview subjects, this meant roughly that they had achieved some major honor or award, such as the Nobel Prize, Pulitzer Prize, National Book Award, Bollingen Poetry Prize, membership in the American Academy of Arts and Letters, in the National Institute of Arts and Letters, in the American Academy of Arts and Science, or in the National Academy of Sciences, and that they were considered highly creative through an objective survey of their writer and scientist peers.

The term "Janusian thinking" is based on the Roman god Janus, whose multiple faces (two, four, or six) (21) were turned in several opposite directions at once. In apparent defiance of logic or matters of physical impossibility, the creative person formulates two *or more* opposites or antitheses coexisting and simultaneously operating, a formulation that leads to integrated concepts, images, and creations. This form of thinking, apparently unique to creative people, plays a constant role in diverse types of creative processes. From the reports of my subjects and from other documented sources, it clearly operated during the creation of poems, novels, plays, paintings, or sculptures, and it operated during the creation of new scientific theories and creative leaps of thought connected to very important scientific discoveries (20). Occurring at crucial moments during the process of creating, usually at the moment of inspiration, this form of thinking has effects that are not always overtly manifest in the final product. The Janusian formulation, in other words, is often a crucial step or way station that later undergoes a good deal of transformation and revision.

Before I describe the revisions and transformations as well as the functions of Janusian thinking, it is important to note that many untransformed Janusian formulations are clearly manifest in some of the great creations of the world. In religious creations, for instance, there is the Yin and Yang formulation of the oriental Taoist religion: two opposite and universal forces or principles operating together as a single force. Nirvana and Samsara are the dominant unified principles of Buddhist theology: Nirvana, the end of the cycle of rebirth, is opposed to and simultaneous with Samsara, the endless series of incarnations and reincarnations of living things. In the Middle East Zoroastrianism, the major religion of the vast Persian Empire, had as its basic tenet the simultaneous presence from the beginning of all things of the twin but opposite gods Ormazd, the god of light and goodness, and Ahriman, the god of darkness and evil. The precepts and descriptions in various aspects of Western theology of a simultaneous operation of God and the Devil, insofar as they are theological creations of the human mind, are also direct manifestations of Janusian formulations.

In philosophy simultaneous opposition and antithesis are manifest in the pre-Socratic conceptions pertaining to Being and Becoming, Nietzsche's Dionysian and Apollonian principles, Kierkegaard's belief by virtue of the absurd, and Sartre's representation of Being and Non-Being. In psychology there are Freud's formulations of the conscious operating together with the unconscious and the theory of the dually functioning but opposed instincts of sex and aggression. Direct manifestation of simultaneous opposition is probably most apparent in the poetry produced by great creators throughout the world and throughout the ages. Many major poets and critics, including William Blake, Samuel Taylor Coleridge, Cleanth Brooks, Robert Graves, Amy Lowell, Robert Penn Warren, Allen Tate, and John Crowe Ransom, have emphasized that poetry is fundamentally based on elements of opposition or a close relation, paradox.

All these examples suggest the workings of Janusian thinking in diverse types of creations, but my evidence is not based merely on consideration of these final created products. It is derived from experimental data and empirical observations of the constant use of this type of thinking in the achievement of creative ideas. For instance, plagued all his life by the suicide of a roommate in his youth, a suicide apparently precipitated by the roommate's wife's infidelity, a great playwright came to realize that the suicide was also motivated by guilt over a simultaneous but opposite wish—the man had wanted his wife to be unfaithful. The formulation resulting from this realization—the idea that his former roommate had both wanted and not wanted his wife to be unfaithful—instigated the writing of one of this playwright's most highly esteemed plays, and the idea directly influenced that play's structure and substance (14). Another noted playwright decided to write a comedy shortly after his father's death. He structured this highly successful play around characters who were both living and dead at the same time.

These characters were not to be represented as ghosts but as lost persons struggling to survive and to understand what had happened to them (20). Another subject of mine, a novelist, during an early phase of thinking about a novel's plot, conceived of a revolutionary hero as responsible for the deaths of hundreds of people but killing only one person with his own hand, a person who had been kind to him and whom he loved. Much of the subsequent novel became an elaboration of this early idea.

One subject, who was a poet, conceived the central image of a poem when he thought of horses as both human and beasts simultaneously, that is, horses were beasts but they lived human lives, they were not human and were human at the same time (20, 22). Another poet, walking on a beach, came on some rocks and thought: the rocks are heavy, they are weapons of violence but, *at the same time*, they feel like human skin. This inspiration led to the construction of a poem concerning the relationship between sex and violence in the world. That sex and violence have much in common was an immediate formulation at the time of perceiving the antithetical qualities of the rocks. For an example from painting, a careful analysis of Picasso's drawings and the successive stages of his creation of the great mural *Guernica* leads to the conclusion that his earliest conception, visually formulated, was of a woman (holding a torch in the completed mural) looking both inside a room and outside onto a courtyard at the same time (20). In context, all of these creative constructions involved high-level, conscious, abstract thinking. As a psychological process, Janusian thinking is a complex, nonprimitive form of cognition.

EINSTEIN'S CREATIVE COGNITION

The sentence following Einstein's underscored and happiest thought is, "The observer is therefore justified in considering his state as one of 'rest.' " Adding this, Einstein clarified the essence of his thought. As he surely knew, the observer falling from the roof of a house is in a state of motion, he is moving during his fall. However, as Einstein conceived it in this formulation, the observer is simultaneously in a state of rest; the observer is thus *in motion* and *at rest* at the same time. This was Einstein's observation about a physical circumstance that allowed him to modify Newton's theory of gravitation so that it would fit into relativity theory. It imbued the "extraordinarily curious empirical law that all bodies in the same gravitational field fall with the same acceleration" with a "deep physical meaning." From the formulation that an observer in free fall is both at rest and in motion at the same time, Einstein was able to postulate the relativity of motion in coordinate systems. Thus, the crucial step in the development of the general theory of relativity, Einstein's creative leap, was a formulation of the opposite states of motion and rest operating simultaneously, an instance of Janusian thinking.

That Einstein knowingly formulated a condition of simultaneous antithesis is clear from the discussion in context. In ordinary experience, falling or being in motion and being at rest are completely antithetical. He therefore devoted several sentences to explaining the particular considerations allowing the moving observer to—as he states twice—"consider his state as that of rest." As Einstein knew, it was a rather shocking and dramatic breakthrough.

The complete development of the general theory of relativity depended on the previous conception of the special theory, as Einstein here made clear, and that theory depended, as Gerald Holton has shown (5), on the direct and indirect influence of Maxwell, Lorentz, Mach, Poincaré, and Föppl as well as important scientific, sociological, and philosophical currents of the time. Other factors included Einstein's enormous capacities for deductive and inductive logic, for combining and separating categories and symbols, and for intense concentration as well as his profound understanding of the rubrics of science and mathematics. As Einstein made clear in this passage, however, the key creative step consisted of finding a way to connect gravitation to relativity on the basis of empirical or physical reality. This key step was formulated all at once as a simultaneous antithesis.

As an instance of Janusian thinking, the formulation was not developed through a stepwise weighing of physical or conceptual alternatives, such as drawing cumulative inferences from a series of observations or deriving conclusions directly from a theory. Nor, as is intrinsic to a dialectical process (dialectical and Janusian thinking are not the same), did Einstein consider the opposites or antitheses in systematic fashion in order to synthesize or combine them. Although he may have engaged in such systematic thinking about the problem at other times, he stated he was working on a summary yearbook essay when he conceived the entire idea.

There was nothing primitive, childlike, or regressive in the creative leap. Absent from the account are any suggestions of an altered state of consciousness or the intrusion of ego-alien material. There is nothing to suggest primary process material appearing in consciousness, no sudden idea followed afterward by critical and logical evaluation. Einstein was fully aware of the logic and reality of the issue at the moment he had, in his words, his "happiest thought." He did not magically conceive of the equivalence of opposites. Visual thinking, which Holton (5, 13) and others (7, 8) have emphasized, and imagery seem to have been present. The idea of a person falling from the roof of a house suggests a visual conception. Rather than the primary process visual imagery emphasized by regression theories of creativity, however, Einstein's probable visualization in this case very likely involved another type of high-level creative thinking (Homospatial thinking), which I have described extensively elsewhere (22).

Einstein's account also clearly conveys both the aesthetic orientation and emotional investment ("The

thought that one is dealing here with two fundamentally different cases was, for me, unbearable.") connected with his scientific thinking that has been emphasized by Holton (5). An interest in economy and simplicity and what Holton has described as a sensitivity to asymmetry and an interest in symmetry seem to play a general role. Janusian thinking is related in an instrumental and critically facilitative way to these general factors. As it is extensively operative in aesthetic creation, as it has definite and specific connections with emotional sources and emotional goals (15, 20), and as simultaneous antitheses or oppositions are intrinsically symmetrical, Janusian thinking is a more primary creative factor than the general ones. Holton's observations are important; aesthetic goals operate ubiquitously in scientific creation. Looking for economy or rejecting asymmetry or being emotionally invested are not, however, the same thing as deriving the particular formulation of simultaneous antithesis that provides key conceptions and dramatic creative leaps.

In its fully worked-out form, the general theory of relativity presented what physicists today consider the principle of equivalence of gravitation and inertia. According to this principle, there is no way to distinguish the motion produced by inertial forces—acceleration, deceleration, centrifugal forces—from motion produced by gravitational force. In blind-flying in an airplane, for instance, it is impossible to separate the physical sensation of pulling out of a dive and that of executing a steeply banked turn at high speed; one cannot separate the effects of inertia from those of gravitation. In both cases blood is drawn away from the brain and the body is pulled down heavily into the seat. The fully worked-out theory, moreover, gave a picture of the universe as a four-dimensional space-time continuum, a construct that changed previous conceptions such as that of the universe as an island of matter in a sea of space. Revolutionizing science, philosophy, and man's view of the nature of physical experience, this construct is the basis for all modern cosmological conceptions. The theory not only allowed for predictions about deflections of the light of stars seen in a solar eclipse, it explained an observation about the behavior of the planet Mercury that had not been accounted for by Newton's laws. It provided a means of dealing with high speed velocities and intense gravitational fields, and it provided an understanding of the shape and size of the universe. Much elaboration, deduction, and calculation went into the development of the entire general theory of relativity, and there has been much empirical testing and conceptual elaboration since it was first constructed. However, as Einstein's exposition now makes clear, the key thought allowing for the formulation, the creative leap giving his incomplete ideas a physical basis and bringing them together in a meaningful formulation, was the specific conception of opposites operating simultaneously—an observer being in motion and at rest at the same time. Such a formulation is a cardinal instance of the creative cognitive process of Janusian thinking.

The cognitive process I have described is not the same as that reported by other investigators. It is not at all merely a manifestation of Guilford's "divergent thinking" (23), Koestler's "bisociation" (3), DeBono's "lateral thinking" (24), or Mednick's formulation of the bringing together of remote associates (25). It is a directed thought process involving active formulation rather than association or bisociation. Moreover, it involves active espousal of specifically antithetical or oppositional entities rather than only divergent or "lateral," unrelated, or remote ones. Nor is Janusian thinking an illogical or prelogical process. In distinction to schizophrenic thinking or the thinking in dreams, for instance, it is a logical postulating of what, on the surface, seems illogical. It is not a form of primary process thinking nor does it derive directly from the unconscious. The creative person actively brings together opposites and antitheses as a means of formulating scientific and other problems, initiating and facilitating aesthetic tasks, creating theories, making discoveries, and constructing works of art.

As seen in Einstein's description, during the creative process the creative person is in neither a trance nor an altered state of consciousness, nor is there any indication that unconscious material erupts directly into consciousness. This is not primary process or free association material brought into consciousness by suspension of secondary process thinking and/or logical and critical judgment. Nor is it correct to say that Janusian thinking consists of unconscious or preconscious material appearing in consciousness during an altered state and then later subjected to logic and critical judgment. Critical assessment and logical elaboration are always necessary to construct and articulate a full theory, discovery, or work of art, but, as Einstein's description again makes clear, the creator is also fully logical and capable of full critical judgment at the moment of the Janusian formulation. Janusian thinking is a form of secondary process cognition. Creative individuals are fully aware that the elements of their thought would be, in a particular context, antithetical, but they engage in what may be called a logic-transcending or "translogical" leap of thought.

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1978 Anniversaries

BY GEORGE MORA, M.D.

The author recalls 1978's anniversaries of events and individuals prominent in the history of medicine, psychiatry, and psychology and examines their practical and theoretical contributions.

1578

William Harvey was born in Folkestone, England. After receiving a thorough classical education at the King's School in Canterbury and Caius College in Cambridge, Harvey studied in Padua, Italy, where he graduated in medicine in 1602. Back in England in the same year, he married the daughter of the King's personal physician. In 1607 he was elected Fellow of the College of Physicians, in 1609 became associated with St. Bartholomew's Hospital, and in 1618 became physician to King Charles I, with whom he developed a close relationship. For 40 years, beginning in 1616, he held the Lumleian lectures on anatomy and physiology at the College of Physicians. Greatly upset by the Civil War, he remained faithful to Charles I. He died in 1657.

Harvey is universally known for his discovery of the circulation of the blood. His discovery, based on solid experimental research, was described in his famous *De Motu Cordis* (1628). Among his other writings are the *Prelectiones* (notes on physiology and pathology related to his Lumleian lectures) and *Exercitationes de Generatione Animalium*, in which he expressed his belief in the theory of epigenesis.

An acute observer of human psychology, especially of women (undoubtedly fostered by his gynecological practice), Harvey followed contemporary views, based on the Galenic tradition, on the knowledge of the nervous system. In the field of psychiatry proper, he anticipated concepts of modern psychosomatic medicine. In *Exercitatio de Motu Cordis et Sanguinis* he wrote that "every affection of the mind that is attended with either pain or pleasure, hope or fear, is the cause of an agitation whose influence extends to the heart." Elsewhere he related the observation of a

> strong man, who having received an injury and affront from one more powerful than himself, and upon whom he could not have his revenge, was so overcome with hatred and spite and passion, which he yet communicate to no one, that at last he fell into a strange distemper, suffering from extreme oppression and pain of the heart and breast . . . and in the course of a few years . . . became tabid and died.

Harvey also described the physiological signs of fainting related to emotions and stated that

> in almost every affection, appetite, hope or fear, our body suffers, the countenance changes, and the blood appears to course hither and thither. In anger the eyes are fiery and pupils contracted; in modesty the cheeks are infused with blushes; in fear, and under sense of infamy and shame, the face is pale but the ears burn as if for evil they heard or were to hear; in lust how quickly is the member distended with blood and erected!

Harvey's psychological insight is particularly evident in regard to symptoms of hysteria. "Women occasionally become insane through ungratified desire, and to such a height does the malady reach in some, that they are believed to be poisoned, or moonstruck, or possessed by a devil." Having stated that the uterus is a most important organ, and brings the whole body to sympathize with it, he said,

> How dreadful, then, are the mental aberrations, the delirium, the melancholy, the paroxisms of frenzy, as if the affected person were under the dominion of spells, and all arising from the unnatural state of the uterus. How many incurable diseases also are brought on by unhealthy menstrual discharges, or from over abstinence from sexual intercourse when the passions are strong!

Harvey also left a beautiful description of a case of "hysteric" pregnancy:

> a young woman . . . who experienced in her own person all the usual symptoms of pregnancy; after the fourteenth week, being healthy and sprightly, she felt the movement of the child within the uterus, calculated the time at which she expected her delivery, and when she thought, from further indications, that this was at hand, prepared the bed, cradle, and all other matters ready for the event. But all was in vain. Lucina . . . tutelar deity of childbirth . . . refused to answer her prayers; the motion of the fetus ceased; and by degrees, without inconvenience, as the abdomen had increased so it diminished; she remained, however, barren ever after.

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Finally, in the case of an 18-year-old girl who had "lost the sense of feeling in the external parts of her Body," obviously on a hysteric basis, Harvey "advised her Parents (who sent her not thither out of poverty) to take her home, and provide her a Husband, by whom, in effect, she was according to his Prognostick, to many Mans wonder, cured of that strange Disease."

1778

Jean Jacques Rousseau died in Ermenonville, France. Rousseau's mother died at his birth in 1712 in Geneva. Having received an uneven education from his unstable father, he fled Geneva and was helped for many years by Mme. de Warens, toward whom he developed a strong attachment. After having held several jobs, he became involved with an ignorant servant girl, by whom he had five illegitimate children, all of whom were sent to a foundling home. In the meantime he became acquainted with some of the leading representatives of the French Enlightenment. His early writings reveal his dissatisfaction with the failure of scientific progress to improve the condition of mankind. His two main works, *Le Contrat Social* and *Émile,* appeared in 1762. After the latter work was burned in Paris by the Catholic authorities, Rousseau accepted an invitation from philosopher David Hume to make his home in England in 1765. Shortly thereafter Rousseau quarreled with Hume and began to show evidence of emotional disturbance. He returned to France and spent his last few years in Paris.

Basic to Rousseau's thinking was his rejection of the cult of reason advocated by the Enlightenment as leading to happiness; in contrast, Rousseau advocated a return to the state of nature. Hence, Rousseau issued his proposal that social life be based on a contract allowing maximal freedom to the individual. A necessary postulate for this is the development of mature individuals through a proper process of education. It is obvious that Rousseau's educational ideas were closely linked to his social goals.

Four stages of education are clearly defined in the four parts of *Émile*. In the first, from birth to 2 years, the child begins experiencing things; in the second, up to 12 years, the senses develop under the influence of pleasure and pain. In the third stage, from 12 to 15 years, the child learns not in an abstract, but in a concrete, way; in the fourth, from 15 to 20 years, the child develops rationally and acquires the ideas of values and religion.

Thus for the first time it was clearly stated that childhood should be studied with its own criteria rather than from the viewpoint of the adult. Childhood should not be seen as an age of imperfection, since it had its own uniqueness and adequacy based on feelings and instinctual needs. As Rousseau put it: "Nature wants children to be children before being men. . . . Childhood has its own ways of seeing, thinking and feeling." Moreover, he felt that a truly positive education began only when the child became aware of his or her relationship with other people.

Although written with a passionate style and presented in an uneven way, *Émile* had a tremendous impact not only on education but on the newly developing field of psychology as well. It was only at the beginning of our century that Rousseau's idyllic concept of childhood was substantially modified by the psychoanalytic emphasis on sexuality and aggression as basic characteristics of childhood. It is likely that Rousseau's revolutionary views were influenced by the unhappy circumstances of his childhood and by the vicissitudes of his life.

Christian Friedrich Nasse was born in Bielefeld, Germany. Proving at an early age to be highly intelligent, he showed an interest in music and modern languages. After studying at several universities, he received his medical degree in Halle in 1800. In 1815, after several years of general practice, he accepted the position of professor of medicine and chief physician at the hospital of the medical school of Halle. In 1819 he was called as clinical professor of medicine to the newly established University of Bonn, where he remained for the rest of his life. There he directed his energies toward two main goals: the care and treatment of patients and the training of students according to the then-modern direction of experimental research on animals, work in the laboratory, and bedside teaching. A prolific writer, he published books and articles on various aspects of medicine. Among his publications on psychiatry is the volume *On the Treatment of the Mentally Ill by Non-Physicians* (1844). He founded and directed the *Journal for Psychological Physicians* (1818–1826), *Annals of Anthropology and Pathology and Therapy of Insanity* (1830), and the *Journal for the Diagnosis and Treatment of Pathological Mental Conditions* (with Jacobi, 1838). He died in 1851.

At various times Nasse called his system psychophysiology, psychosomathology, and anthropology—names that emphasize the unity of the personality. He viewed psychosis as entirely dependent on a physical condition; as mind and body are indissolubly united, it is impossible to speak of mental disorders in the strict sense of the term. He also assumed a critical stance toward mechanistic and spiritualistic, and monistic and dualistic attitudes. Under the influence of the philosopher Herbart, he attempted to build a psychology based on mathematical foundations and void of philosophical postulates. His principles of clinical medicine were based on direct observation of the patient, assessment of environmental factors, and formulation of the individual diagnosis. In the Clinic for Mental Disease, which he opened in Bonn in 1819, he created a small community of patients, teachers, nurses, and students. For him, the psychiatrist should be an "anthropological physician" as, indeed, he was himself: a specialist in welfare medicine, clinical matters, teaching, and legal issues.

Along with other representatives of German organicistic philosophy of psychiatry (Reil, Friedreich, Jacobi), Nasse was opposed to the so-called "mentalists" (Heinroth, Ideler, Groos). He denied the existence of prenatal psychological influences and viewed all deductions of psychologists on the infantile psyche as pure fantasy. For him, the etiology of mental disorders had to be found in disorders of the various organs of the body, especially the brain. Yet under the influence of Herbart, he accepted the notions of "threshold of the unconsciousness" and, consequently, of repression and inhibition. He was also interested in the study of dreams and somnambulism. In the practical handling of the mentally ill he was eclectic and open-minded, making use of dietetics, limited means of restraint, psychological influence, and occupational therapy.

1878

Claude Bernard died. Born in St. Julien (Rhône, France) in 1813, he received essentially a humanistic education. After working as an assistant pharmacist for some time (and unsuccessfully as a writer), he studied medicine in Paris, where he became assistant to François Magendie. Under his influence Bernard became involved in experimental physiology, as indicated by his medical thesis on the role of gastric juices in nutrition (1843). He developed the notion of "physiological autopsy," a technique aimed at isolating the properties of tissues and organs with the help of chemical substances. Among his most important discoveries in the field of neurophysiology during the succeeding years were the etiology of diabetes, the constricting and dilating functions of the vasomotor nerves, and the action of curare on the voluntary muscles (in recent years this action has had application in anesthesia and electroconvulsive therapy).

Soon recognized as the leading experimental physiologist in France, Bernard received a number of awards and was appointed to prestigious positions, first at the Sorbonne (chair of general physiology), then the Academie des Sciences and the Collège de France (chair of experimental medicine). In time he produced a number of volumes, the most important of which remains the *Introduction à l'étude de la médecine expérimentale* (1865), preceded by the *Lectures on the Physiology and Pathology of the Nervous System* (1858). In these works he developed the concepts of "inner secretion," "inner environment," and "reciprocal inhibition," which were to become basic to the understanding of the functions of the organism. As a matter of fact, he always gave preeminence to function over form in his painstaking search for the essence of life. In later years he taught at the Museum of Natural History (1868) and was appointed senator of the empire (1869) and president of the Académie de France (1869). Of his many pupils, some became outstanding scientists in France, Germany, Russia, and the United States.

Stanley Hall received the first American doctorate in psychology from Harvard University. Hall was born in 1844 and raised on a farm in Ashfield, Mass. Endowed with great personal ambition, he attended Williams College, where he became enthusiastically inspired by philosophy and evolution. In 1867 he studied for the ministry at Union Theological Seminary in New York. He then spent some time learning theology and philosophy, as well as physiology and physics, in Bonn and Berlin. Back home in 1871, he took his degree in theology but soon resigned from his first pastorate. For three years he taught French and German languages and literature at Antioch College. His interest in psychology was spurred by reading Wundt's *Physiological Psychology*. Eventually he came under the influence of William James at Harvard, where in 1878 he presented his dissertation on the muscular perception of space. Following that he spent two years in Leipzig as Wundt's first American student.

Now definitely committed to the study of psychology, Hall embarked on an investigation of children's thoughts and beliefs through interviews and questionnaires. He slowly gained renown through a series of Saturday morning talks on education given at the invitation of the president of Harvard. In 1881 he accepted an offer to lecture at Johns Hopkins, and three years later he became full professor there. In 1883 he founded at Hopkins what is usually considered the first American psychology laboratory; a number of promising young men, such as Cattell and Dewey, were trained there. In 1887 he began to publish the *American Journal of Psychology*, the first journal of psychology in this country and still an important one today. Appointed the first president of the new Clark University in 1888, Hall aspired to develop a research center modeled on the style of the German universities. There he taught psychology and founded the *Pedagogical Summary* (now the *Journal of Genetic Psychology*) in 1891. The next year, with a few others, he founded the American Psychological Association, of which he was elected the first president.

The results of the many studies on children conducted by Hall and his associates were published in 1894 in *The Content of Children's Minds on Entering School*, a pioneering endeavor in the field of child psychology. It was followed by the monumental two-volume book *Adolescence* in 1904. By that time Hall was intrigued by the novelty of the psychoanalytic theories. In spite of the opposition of many of his colleagues and much of the public, he managed to invite Freud, Jung, and some other psychoanalysts for a series of conferences on the occasion of the 20th anniversary of Clark University in 1909. His longstanding interest in religion led him to publish the book *Jesus the Christ in the Light of Psychology* (1917), which was severely criticized by organized religion. He continued to work even after his retirement in 1920. In 1922 he published his two-volume book *Senescence*, the first work on geriatric psychology. Two of his books, *Recreation of a Psychologist* (1920) and *The*

Life and Confessions of a Psychologist (1923), are autobiographical. Hall died in 1924.

In retrospect, Hall's interests may appear rather fragmentary and scattered. However, he should be credited with two main accomplishments: he aroused interest in new fields, notably developmental psychology and psychoanalysis through his excellent skill as an organizer and publisher, and he maintained the evolutionary theory as the constant focus of his multifarious activity.

John B. Watson was born in Greenville, S.C. A rebellious and independent spirit from early years, at age 16 he entered Furman University, from which he obtained a master's degree in 1900. At that time he was already opposed to any religious influence. He then entered the University of Chicago, which was then dominated by George Mead and John Dewey. There he was particularly influenced toward experimental psychology by James R. Angell and toward a materialistic orientation of biological studies by Jacques Loeb, professor of physiology and originator of the theory of "tropism." Watson received his Ph.D. in 1903. In that same year he married a woman who belonged to a prominent family and continued to work at the University of Chicago as an instructor in experimental psychology and director of the psychological laboratory.

In 1913 Watson published his famous manifesto "Psychology as a Behaviorist Views It." In it he proclaimed that psychology "is a purely objective experimental branch of natural science. Its theoretical goal is the prediction and control of behavior." The following year he produced *Behavior: An Introduction to Comparative Psychology,* a defense of animal psychology and of the place that it occupies in the behavioral sciences. In this way Watson was advocating a new kind of psychology "in the third person," which was at variance with Wundt's psychology "in the first person" (the school of introspection) and Freud's psychology "in the second person" (based on the transference situation between the individual and the analyst).

The forceful and at times polemic way in which Watson presented his ideas had a great impact on the entire field of American psychology. Elected president of the American Psychological Association in 1915, he announced his support of the theory of conditional reflexes brought forward by the Russian neurologist V.M. Bekhterev. (Pavlov's main work on conditional reflexes did not become available in English until 1927.) The climate seemed ready for a shift in psychology from a speculative to a concrete orientation.

After a period of difficulties toward the end of World War I related to his criticism of a neurological test for pilots, Watson began the study of children at the Henry Phipps Clinic in Baltimore. He concluded that the emotional reactions of children can be reduced to three types: love, elicited by the gentle stimulation of the skin; rage, elicited by physical restraints; and fear, elicited by loud noises and loss of support. In 1919 he published his most famous book, *Psychology from the Standpoint of a Behaviorist.*

In 1920, at the peak of his career, Watson was divorced and soon afterward married Rosalie Rayner, his coworker at Hopkins. The reaction of the academic community was so strong that he was dismissed from his position and had to work as a salesman. He obviously revealed talent in this new occupation, for in three years he rose to the position of vice-president of a large advertising agency. He remained in the field of business until his retirement in 1946.

Watson's main interest, however, continued to be psychology, and he pursued it through lecturing (at Clark University and the New School for Social Research) and writing. His book *Behaviorism* (1925) contains the following famous statement: "Give me a dozen healthy infants, well-formed, and my own specific world to bring them up in and I will guarantee to take any one at random and train him to become any type of specialist I might select." As far as is known, this experiment was never carried out. In 1928, he produced *Psychological Care of the Infant and Child,* a popular book for the general public. It was followed the next year by *The Battle of Behaviorism,* a polemic against William McDougall, then professor at Harvard and the exponent of the doctrine of purposive behavior. Watson spent the last years of his life in Westport, Conn. He died in 1958.

Although Watson's theories were highly controversial, they were very stimulating for both his adherents and opponents. During his life neobehaviorism was flourishing, mainly through the work of C.L. Hull. After his death the spread of behavior therapy gave new meaning to Watson's work.

Kurt Goldstein was born in Kattowitz, Germany (now Poland). After obtaining a medical degree from the University of Breslau in 1903, he taught at the University of Königsberg (1907–1915) and then worked as director of the Institute for Research into the Aftereffects of Brain Injuries. He later became professor of neurology at the University of Frankfurt (1916–1929), followed by a short stay in Berlin (1929–1933). His firsthand experience with large numbers of brain-injured soldiers during World War I, coupled with the influence of the then-emerging movement of Gestalt theory, led him to consider the organism as a whole regardless of any particular deficit. Together with Wertheimer, Köhler, Koffka, and Gruhle, he was one of the cofounders of the Gestalt psychology journal *Psychologische Forschung* (1921–1938).

Forced by the Nazis to leave Germany, Goldstein emigrated to Amsterdam (1934–1935), where he wrote his classic volume *The Organism: A Holistic Approach to Biology Derived from Pathological Data in Man* (1934; translated into English in 1938). In it he presented the concept of the inborn potential toward self-realization as a driving force that puts the organism into action and achieves its optimal state when there is "adequacy" between the organism and the

world. Good gestalt is "a form of coming to terms of the organism with the world, that form in which the organism actualizes itself, according to its nature, in the best way." Poor gestalt, "the tendency to discharge any tension whatsoever is a characteristic expression of a defective organism, of disease." In fact, aphasic and brain-injured persons lack the ability to name objects as class symbols rather than experiencing the loss of words and word images, which indicates that while defects can be localized, functions cannot. In particular, Goldstein described the "catastrophic reaction" of brain-injured patients as a state of tremendous anxiety accompanied by a sense of "losing existence" or feeling unable "to be." By dividing human acts into the abstract and the concrete, he postulated that impairment in abstract thinking (attention, planning, discrimination, hypothesizing) leaves concrete operations unaffected, and vice versa. The Goldstein-Gelb and Goldstein-Scheerer tests were devised to measure the ability to form concepts and to diagnose brain lesions. Goldstein suggested that schizophrenics' behavior may be either concrete or abstract, but that it tends to become concrete in anxiety-arousing situations.

From 1936 to 1940 Goldstein was professor of psychiatry at Columbia University and chief of the Neurophysiological Laboratory at Montefiore Hospital in New York City. During the period 1938–1939 he delivered the William James lectures, which were published under the title *Human Nature in the Light of Psychopathology* (1940). He later served as professor of neurology at Tufts Medical School in Boston (1940–1945). From 1946 until his death in 1965 he taught at the College of the City of New York, Columbia University, and the New School for Social Research, and was guest professor at Brandeis University in Waltham, Mass. In 1962 he was one of the cofounders of the *Journal of Humanistic Psychology*. Among those particularly influenced by Goldstein were Abraham Maslow, the well-known promoter of "third force (humanistic) psychology," and Fritz Perls, who was the founder of Gestalt psychotherapy. Goldstein's *Selected Papers* were published posthumously in The Hague in 1971.

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SPECIAL SECTION: The Psychobiology of Depression—NIMH-Clinical Research Branch Collaborative Program

Introduction: Overview of the Clinical Studies Program

BY MARTIN M. KATZ, PH.D., AND GERALD L. KLERMAN, M.D.

THE IMPETUS for the NIMH-Clinical Research Branch Collaborative Program on the Psychobiology of Depression stemmed from the growing concern with depression as a U.S. public health problem and the growing awareness that there had been some striking changes over the past 20 years in our conception of the nature and etiology of the affective disorders. New findings in the area of genetics and biochemistry inspired hypotheses implicating the chemistry of the endocrine and central nervous systems in ways quite different than had been thought in the past. There was reason to believe that we were on the verge of linking physiology, psychosocial factors, and behavior in these disorders, which would eventually make possible the development of a coherent and valid psychobiologic theory of depression. However, serious methodologic problems and the practical difficulties intrinsic to clinical research had seriously retarded progress toward this objective.

Because of these developments and the perceived obstacles, NIMH staff convened a conference in 1969 (1) to review progress and to consider ways of accelerating the pace of research in this sphere. The conferees were asked to analyze the state of the field and to develop a set of recommendations to guide future research. Their consensus regarding the major affective disorders was

1. That they are heterogeneous both in regard to their nature and their etiology.
2. That there is a subclass which is almost certainly hereditary in nature.
3. That a form of derangement of central nervous system chemistry is implicated in certain severe forms of the disorder.
4. That significant progress has been made in issues of diagnosis. There are now several systems of classification, e.g., the primary-secondary and bipolar-unipolar systems, which appear valid for different purposes; no one system, however, meets all of the requirements for research and for clinical practice.
5. That despite the advances in the conceptualization of nosologic systems, diagnostic practice remains sufficiently unreliable across clinical settings to make comparison of results of different studies consistently hazardous.
6. That the compelling hypotheses in the field which implicate genetics, biochemistry, and/or psychosocial factors in the etiology or nature of the depressive disorders derive from small studies and, thus, require replication.
7. That settings having a sufficient number and diversity of patients, adequate technical resources for conducting clinical and biological studies, and the willingness to conduct such replications would likely be rare, if they existed at all.

These observations, which span issues of nosology, clinical methodology, and the role of psychosocial factors, led to a broad set of recommendations on which this highly diverse group of basic scientists and clinical investigators again had reasonably high agreement.

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Drs. Katz and Klerman are Co-Chairmen, NIMH-Clinical Research Branch Collaborative Program on the Psychobiology of Depression. At the time this work was done, Dr. Katz was Chief, Clinical Research Branch, NIMH; he is now Research Professor, Department of Psychology, Long Island University, Brooklyn Center, University Plaza, Brooklyn, N.Y. 11201. Dr. Klerman is Administrator, Alcohol, Drug Abuse, and Mental Health Administration.

This work was done as part of the NIMH-Clinical Research Branch Collaborative Program on the Psychobiology of Depression: Clinical Studies and was completed with the cooperation and participation of the Collaborative Program investigators: Martin M. Katz, Ph.D., and Gerald L. Klerman, M.D., Co-Chairmen; Robert M.A. Hirschfeld, M.D., Coordinator; Martin B. Keller, M.D., Robert W. Shapiro, M.D., and James Barrett, M.D. (Boston); Jan A. Fawcett, M.D., and William A. Scheftner, M.D. (Chicago); George Winokur, M.D., and Nancy C. Andreasen, M.D. (Iowa City); Robert L. Spitzer, M.D., and Jean Endicott, Ph.D. (New York); and Eli Robins, M.D., Paula J. Clayton, M.D., Theodore Reich, M.D., Amos Welner, M.D., and Jack Croghan, M.D. (St. Louis).

RECOMMENDATIONS OF NIMH CONFERENCE

First, it was clear that the technical obstacles noted would significantly interfere with the capacity to generalize results from one study to another; more importantly, they would prevent certain kinds of research from actually getting done. The foremost technical problem was establishing consensus on and reliability of the measurement of the critical symptomatic and behavioral facets of the various subtypes of affective disorder. An interview method that would be sufficiently comprehensive and would be capable of reliable application across different clinical settings would have to be developed or adapted from prior instruments.

Second, there was need to resolve more basic nosologic issues and to clarify the relative values of the various systems of classification currently in practice in the field. Because new nosologic systems have arisen (based, for example, on genetic findings) that should, for purposes of research, replace earlier systems, it was necessary to determine the reliability with which they can be applied and to compare the new with the traditional systems vis-à-vis their relative validities for research and practice. The second recommendation, then, was to initiate research aimed directly at resolving the issues underlying the uncertainty of nosology in this field.

Third, aware of the powerful influence that the new genetic findings and biochemical theories had already had on the course of research in the entire field, the group saw a need to develop the means or capacity to conduct studies aimed at confirming or disconfirming critical findings arising from research in these areas.

Certain research objectives could not possibly be met without a situation in which a large number of patients of certain types were available. Research of a multisystem nature (i.e., simultaneous investigation of central nervous system, endocrine, and behavioral functioning) requires an array of patients, a range of technology, and an unusual combination of expertise—requirements that are not likely to be found in any one setting. Because of the multifaceted nature of this class of clinical conditions, research on its nature or etiology requires a somewhat unique strategy. It became clear that settings in which all of the clinical resources, i.e., expert clinicians, adequate samples of relevant patient groups, and such technical resources as biological and clinical scientists, were virtually nonexistent. It was also necessary that the interests and capacities to develop clinical methodology and to conduct replicative studies in the biological sphere be available.

What evolved from these discussions was the clear need to develop mechanisms of collaboration across disciplines, laboratories, and clinical settings that would maximize the capacity of the program and expedite the solution of these problems. In developing such a mechanism, it was important that it be designed to complement and to stimulate, not to stem or in any sense replace the efforts of independent laboratories, in moving forward on basic problems in this area.

The collaborative mechanism would, therefore, focus on methodologic problems currently not being studied, on the need for replication, and on conducting research that independent laboratories were not currently capable of carrying out.

With this set of recommendations as background, the Clinical Research Branch of the National Institute of Mental Health convened an advisory group on collaborative research on the psychobiology of the affective disorders. This initial group, formed in 1971, eventually divided into a biological studies group and a clinical studies group. The biological studies group concentrated on the development of replicative research to test hypotheses implicating CNS and endocrine chemistry and their links with behavior in depression. Through the collaboration of five investigative groups and NIMH staff, that research is currently in operation. Its rationale and design are described elsewhere (2).

THE CLINICAL STUDIES PROGRAM

The clinical studies group is comprised of centers at Massachusetts General Hospital and Harvard Medical School, Boston; Rush-Presbyterian-St. Luke's Medical Center, Chicago; the University of Iowa, Iowa City; the New York State Psychiatric Institute, New York; the Washington University School of Medicine, St. Louis; and NIMH, Rockville, Md. Through the years it has focused its efforts on

1. The development of assessment methodology to be applied to a wide range of clinical problems, primarily in defining the basic dimensions of psychopathology and the relevant clinical phenomena for the diagnosis of depression.
2. The design of research to test the relative validity of various extant nosologic systems in research and in clinical practice.
3. The development of studies to replicate findings and to test new hypotheses about genetic transmission.
4. The testing of hypotheses about the role of personality and environmental factors in influencing the course and nature of the depressive episode.

During the past 5 years of collaborative effort, the group has developed the conceptual bases for and designed research aimed at these objectives. The objectives of the current program are described in appendix 1. This group has also constructed assessment methods of major significance for research on diagnosis, e.g., the Schedule for Affective Disorders and Schizophrenia (SADS) (3) and the Research Diagnostic Criteria (RDC) (4), which are already widely used in the field and in clinical practice.

Having completed the groundwork, the group then initiated, with National Institute of Mental Health support, a major pilot effort across four of the centers to

refine and extend the method development and to assess the feasibility of a full-scale intercenter investigation to conduct collaborative research on nosologic, genetic, and psychosocial issues. That pilot study lasted 2 years and was successful from the standpoint of what it was able to accomplish across the four centers and what it indicated was not possible to accomplish at this time.

CONCLUSION

To communicate in broad outline the nature of the program, the emphasis in the set of articles presented here is more on description than on actual results. The articles include broad descriptions of the diagnostic methodology (the SADS and RDC), applications of their use, some preliminary work on the application of techniques for studying personality, and, in the examination of the varied meanings of the diagnosis of neurotic depression, an example of how the program proper will be applied to resolve current issues of controversy that cut across nosologic systems.

The major strength of the overall effort will in the long run lie in its capacity to stimulate and extend independent basic research on depression. We expect to contribute to this goal by providing the clinical methodology, by differentiating through replicative studies the hard from the soft findings, and by using the collaborative mechanism to conduct research that is not possible in one laboratory or clinical setting.

The depressions, like all severe mental disorders, are conditions of the whole organism; their etiologies are not likely to be determined by any one scientific discipline. Research involving the collaboration of disciplines and settings is essential in this field and complements our attempts through research in independent laboratories to progress more rapidly in resolving these critical public health issues. Although preliminary, we trust that these reports will serve as a stimulus and provide the base for the more definitive work to be carried out through this unusual collaborative effort over the next 5 years.

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APPENDIX 1
Objectives of the Clinical Studies Group of the NIMH-Clinical Research Branch Collaborative Program on the Psychobiology of Depression

I. Clinical psychopathology
  A. Comparative nosology
    1. Assess strengths and weaknesses of various classification systems
    2. Assess predictive validity
  B. Phenomenology
    1. Interrelate observations from clinical judgment, community behavior, and self-report
    2. Identify dimensions of depression
    3. Determine if there is a natural typology of depression
  C. Psychosocial factors
    1. Interrelationships of psychosocial stresses, social support systems, personality, and sociodemographic qualities
II. Genetics/family
  A. Family
    1. Correlate familial psychopathologic rates with nosology class of proband
  B. Genetics
    1. Determine specific mode of transmission through linkage and extended pedigree analysis (or studies)

Use of the Research Diagnostic Criteria and the Schedule for Affective Disorders and Schizophrenia to Study Affective Disorders

BY JEAN ENDICOTT, PH.D., AND ROBERT L. SPITZER, M.D.

In a pilot study of 150 manic or depressive patients, the authors used the Research Diagnostic Criteria (RDC) and the Schedule for Affective Disorders and Schizophrenia (SADS) to perform preliminary analyses of symptom pictures of the index episode of different diagnostic groups, joint diagnostic classification of the different subtypes of major depressive disorder, and differential outcome by diagnostic groups. The results suggest that schizophrenic symptoms in affective disorders do have diagnostic and prognostic significance, that the term "psychotic depression" should be limited to impaired reality testing without reference to degree of incapacitation, that situational-nonsituational and endogenous-nonendogenous classifications are separate depressive subtypes, and that it may not be true that patients with endogenous major depressive disorder have a better prognosis than patients with nonendogenous depression.

TWO PROBLEMS have plagued investigators in the study of affective disorders. The first and most important problem has been the absence of agreed upon sets of criteria that would enable investigators to compare the results of studies which use different ways of classifying patients. As a result, it is usually impossible to know if one investigator's patients who are characterized, for example, as having "severe endogenous depression," correspond to another investigator's patients described as having "psychotic depression." The second problem has been the absence of uniform procedures for collecting the information used to make diagnoses and to describe the characteristics of the study samples.

As part of the NIMH-Clinical Research Branch Collaborative Program on the Psychobiology of Depression, two new procedures were developed to minimize these problems: the Research Diagnostic Criteria (RDC) (1) and the Schedule for Affective Disorders and Schizophrenia (SADS) (2). This article focuses on the use of the RDC and the SADS in a pilot study that was part of the Collaborative Program and presents preliminary data on the use of these procedures for the study of affective disorders.

DESCRIPTION OF RDC AND SADS

The Research Diagnostic Criteria (RDC) provide sets of specific inclusion and exclusion criteria for a large number of functional psychiatric disorders, with particular emphasis on various ways of subtyping affective disorders. A single subject can be categorized in a variety of non-mutually exclusive ways, such as by the presence or absence of endogenous phenomenology, situational stresses, psychotic features, etc. We have described elsewhere (3) the rationale behind the selection of the RDC categories and criteria as well as initial interjudge reliability studies. The reliabilities obtained for the RDC categories are far above those generally obtained for clinical diagnosis in either joint interviews or test-retest interviews. Whereas most studies of diagnostic reliability yield kappas (an index of reliability that corrects for chance agreement) that range from .4 to .6 (4), the kappas for the RDC were usually above .7 and frequently above .8.

The Schedule for Affective Disorders and Schizophrenia (SADS) was developed in conjunction with the RDC in an effort to reduce information variance in both the descriptive and diagnostic evaluation of a

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This work was done as part of the NIMH-Clinical Research Branch Collaborative Program on the Psychobiology of Depression: Clinical Studies and was completed with the cooperation and participation of the Collaborative Program investigators: Martin M. Katz, Ph.D., and Gerald L. Klerman, M.D., Co-Chairmen; Robert M.A. Hirschfeld, M.D., Coordinator; Martin B. Keller, M.D., Robert W. Shapiro, M.D., and James Barrett, M.D. (Boston); Jan A. Fawcett, M.D., and William A. Scheftner, M.D. (Chicago); George Winokur, M.D., and Nancy C. Andreasen, M.D. (Iowa City); Robert L. Spitzer, M.D., and Jean Endicott, Ph.D. (New York); and Eli Robins, M.D., Paula J. Clayton, M.D., Theodore Reich, M.D., Amos Welner, M.D., and Jack Croghan, M.D. (St. Louis). This work was also supported by Alcohol, Drug Abuse, and Mental Health Administration grant MH-23864 from the National Institute of Mental Health and by the New York State Department of Mental Hygiene.

subject. The SADS is an interview schedule that interviewers use to ensure adequate coverage of critical areas of psychopathology and functioning.

Part I of the SADS is unique among rating scale procedures in that the items are used to describe the features of the current episode of illness when they were at their most severe, in contrast to most psychiatric rating scales, which cover a set time period regardless of the relationship of the time of the evaluation to the beginning and peak of the episode. In addition to the collection of data relevant to an evaluation of the diagnosis, prognosis, and phenomenology of a current episode of illness, part I of the SADS is also designed to measure change. Many of the scaled items are also judged for the level of severity during the week preceding the evaluation. This subset of items can then be used for subsequent evaluations to measure change using the Change version of the SADS (SADS-C). Part II of the SADS focuses primarily on past history, although some items describe current behaviors, and contains items relevant to prognosis and overall severity of illness as well as diagnosis. The rater should be able to make an RDC diagnosis at the end of the SADS evaluation.

The rationale and description of the development of the SADS and an initial summary scoring system, as well as the results of the reliability studies of the Collaborative Program, are described elsewhere (5). The reliabilities for the individual items and for five composite summary scales were found to be very high under both joint and test-retest conditions, with most reliability coefficients (intraclass R) .75 or higher.

PILOT STUDY

The RDC and SADS were used in a pilot study designed to assess the feasibility of the procedures, to determine their reliability, and to perform preliminary analyses regarding symptom pictures of the index episode of different diagnostic groups, joint diagnostic classification of the different subtypes of major depressive disorder, and differential outcome by diagnostic groups. The subjects were 150 newly admitted inpatients who had either a manic or depressive syndrome and met other screening requirements for entry into the study; all of the subjects gave informed consent. The data were collected by four of the centers participating in the Collaborative Program.

Ninety of the 150 patients had an RDC diagnosis of major depressive disorder for the index episode, and 9 were diagnosed as having schizo-affective disorder, depressed subtype. We focus here on subgroups of these 99 patients. As defined in the RDC, a diagnosis of major depressive disorder requires an episode of illness of at least one week's duration, in which there is a prominent and relatively persistent dysphoric mood or pervasive loss of interest or pleasure, accompanied by at least 4 of 8 specific features of the depressive syndrome. In addition, certain features that suggest schizophrenia must be absent. In contrast, the diagnosis of a schizo-affective disorder, depressed subtype, requires the presence of the positive features of the major depressive disorder as well as at least 1 of the symptoms suggesting schizophrenia.

CONTRASTS BETWEEN SCHIZO-AFFECTIVE DISORDER AND PSYCHOTIC MAJOR DEPRESSIVE DISORDER

There is no consensus as to how to define schizo-affective disorder or whether it should be defined as a separate group (6–10). Schizo-affective disorder, depressive subtype, is defined in the RDC as an illness in which there is a temporal overlap of both a major depressive syndrome and specified schizophrenic-like features. Psychotic major depressive disorder was defined as a major depressive syndrome with delusions or hallucinations or depressive stupor.[1] We tested two hypotheses regarding the characteristics of the index episode: 1) The onset of schizo-affective disorder is more often associated with stress and is more rapid than that of psychotic major depressive disorder, and 2) schizo-affective disorder is associated with more signs of disorganization, and the delusions associated with it are more numerous and of greater variety than with psychotic major depressive disorder.

Table 1 indicates which of the 120 scaled items in the SADS were significantly different for patients having schizo-affective disorder, depressive subtype (N=9) and those having psychotic depression (N=22). Items involved in the differentiating criteria are noted at the bottom of the table and, as expected, are significantly different. In contrast to the hypothesis, there were no differences on the two items evaluating stress (the rater's judgment and the patient's judgment). However, as hypothesized, the duration of the onset was significantly longer in the patients with psychotic major depression (one to two months versus one to two weeks). A higher percentage of patients in the schizo-affective group were found to be delusional (100% versus 54%), although the mean score was significantly higher only on the persecutory subtypes. The overall severity of delusions was also greater in the schizo-affective group, as was that of hallucinations and the global measurement of symptoms and impairment, the GAS score. The depressive syndrome of the psychotic major depressive patients was also characterized by more endogenous features than was that of the schizo-affective patients.

Although the RDC criteria differentiating the two groups of patients had little prior research support, the prior history and follow-up data of the pilot study suggest that the criteria used have some validity beyond that which is merely definitional. The schizo-affective patients had had more previous episodes and more residual impairment after previous episodes than the pa-

[1]The criterion of depressive stupor is no longer used.

TABLE 1
Mean Scores[a] on the SADS Part I Items That Differed Significantly for Patients with Schizo-Affective Disorder, Depressed Type and Patients with Psychotic Major Depressive Disorder[b]

Item	Schizo-Affective Disorder, Depressed Type (N=9)	Psychotic Major Depressive Disorder (N=22)	t[c]	ϵ
Not differentiating criterion				
Duration of episode	3.88	5.25[d]	2.36	.42
Duration of onset	3.57	5.20[d]	2.42	.45
Worthlessness	2.22	3.77[d]	2.34	.40
Poor appetite	2.44	3.91[d]	2.23	.38
Increased appetite	1.67[d]	1.00	2.87	.48
Weight loss	1.75	3.05[d]	2.05	.36
Weight gain	1.78[d]	1.00	2.81	.47
Persecutory delusions	2.11[d]	1.36	2.54	.43
Severity of delusions	5.00[d]	2.32	5.12	.71
Auditory hallucinations	2.67[d]	1.86	2.20	.37
Severity of hallucinations	4.44[d]	2.50	3.26	.53
Impaired functioning	4.50[d]	3.36	2.17	.38
Self-pity	1.00	2.00[d]	2.27	.39
GAS—worst period[e]	32.56[d]	38.95	2.12	.36
Differentiating criterion				
Delusions of control	2.22[d]	1.09	5.03	.71
People read mind	2.44[d]	1.14	5.41	.73
Thoughts broadcast	1.78[d]	1.09	3.05	.51
Degree to which delusions are bizarre	2.56 (N=9)[d,f]	1.15 (N=12)[f]	2.56	.46
Loose associations	1.56[d]	1.00	2.36	.40

[a]Scale of 1–6.
[b]Data from 150 cases in the pilot study; 120 items contrasted.
[c]t=2.04 at the .05 level, two-tailed.
[d]Most psychopathological score.
[e]Lower GAS scores indicate greater impairment.
[f]Delusional subjects only.

tients with psychotic major depression, and on follow-up two years later they were found to have more depressive symptoms and role impairment. Fifty-five percent of the schizo-affective group and 18% of the psychotic major depressive patients were found to be out of work because of psychopathology. Similarly, only 33% of the schizo-affective patients were found to have a GAS score above 60 whereas 50% of the psychotic major depressive patients had such scores.

The sample sizes in this study are small and there is obviously a need for replication. However, these findings do suggest that it may be premature to conclude, as have Pope and Lipinski (10), that "schizophrenic" symptoms occurring in the course of an affective disorder have no diagnostic or prognostic significance. Our findings are consistent with those of Croughan and associates (9), who also found that the presence of certain psychotic features in an affective illness was associated with a more chronic course, although their definition of schizo-affective disorders differs somewhat from that used in the RDC.

Contrasting Definitions of Psychotic Depression

Two of the criteria that have been used in defining psychotic depression are impaired reality testing and degree of incapacitation. These two criteria are split in the RDC into psychotic (having to do with reality testing) and incapacitating (having to do with functional impairment). A cross tabulation of these two subtypes revealed that 74% of the psychotic patients were incapacitated but only 40% of the patients with a diagnosis of incapacitating were also psychotic. Furthermore, the two groups differed in such important behavioral correlates as psychomotor retardation. Whereas only 48% of the patients with impaired reality testing had significant retardation, 70% of those who were functionally incapacitated were so described.

These findings indicate what most clinicians have long known: the broad definition of the term psychotic that was given official sanction in *DSM-II* (7) combines two very different concepts: impairment in reality testing and impairment in ability to function. Descriptively, it would seem to make better sense to restrict the use of the term psychotic to impaired reality testing, as done in the RDC and in the draft of *DSM-III* (11).

RELATIONSHIP BETWEEN ENDOGENOUS-NONENDOGENOUS AND SITUATIONAL-NONSITUATIONAL MAJOR DEPRESSIVE DISORDERS

The absence of stress and the presence of endogenous features are frequently used, in a confusing and inconsistent fashion, to define endogenous depressions. The RDC subtypes of major depressive disorder use both concepts. A situational major depressive disorder is defined in the RDC as a depression that is judged to have developed after an event or in a situation that seems likely to have contributed to the appearance of the episode at that time. An endogenous major depressive disorder is defined in the RDC on the basis of phenomenology of the current episode. There must be several of the clinical features that are repeatedly reported in the literature as characterizing depressive illnesses which arise in the absence of life events and which seem particularly responsive to somatic therapy, e.g., terminal insomnia, weight loss, and lack of reactivity of mood.

The two categories allow classification by both ways of subtyping as well as by a comparison of the features associated with the defining characteristics. Diagnoses of 45 of the 90 patients with major depressive disorder were subclassified as situational and 52 were called endogenous subtype. The cross tabulation of the subtypes indicates that 42% of the situational patients were also diagnosed as having endogenous subtype; 73% of the nonsituational patients were also called endogenous. Reversing the procedure revealed that 37% of the endogenous patients were called situational and 68% of the nonendogenous patients were called situational.

The cross tabulation data as well as those shown in tables 2 and 3 indicate that although there is considerable overlap in the characteristics of patients classi-

TABLE 2
Mean Scores[a] on the SADS Part I Items That Differed Significantly for Patients with Endogenous Major Depressive Disorder and Patients with Nonendogenous Major Depressive Disorder[b]

Item	Endogenous (N=52)	Non-endogenous (N=38)	F[c]	ϵ
Not differentiating criterion				
Degree of stress	3.07	4.06[d]	8.97	.30
Patient thinks stress is related	2.39	3.11[d]	9.46	.31
Outpatient treatment	2.60[d]	2.21	5.44	.22
Depressed mood	5.33[d]	4.76	4.14	.19
Lack of specific concerns	3.96[d]	2.76	10.16	.31
Increased appetite	1.04	1.50[d]	7.17	.26
Problem concentrating	4.37[d]	3.58	6.12	.23
Decreased involvement	4.06[d]	2.74	19.56	.42
Pleasure capacity when upset	4.67[d]	2.87	26.91	.48
Nonverbal manifestation of depression	3.04[d]	2.18	9.82	.30
Exaggerates	1.06	1.42[d]	6.70	.25
GAS—worst week[e]	37.69[d]	44.11	11.11	.32
GAS—week prior to admission	37.90[d]	43.47	7.77	.27
Differentiating criterion				
Quality of mood	3.26[d]	2.23	12.21	.35
Decreased mood	5.12[d]	3.34	42.71	.57
Reactivity of mood	4.42[d]	2.82	22.65	.44
Mood worse in morning	2.02[d]	1.21	11.35	.32

[a]Scale of 1–6.
[b]Data for 150 cases in the pilot study; 120 items contrasted.
[c]F value of 3.96 significant at the .05 level, two-tailed.
[d]Most psychopathological score.
[e]Lower GAS scores indicate greater impairment.

TABLE 3
Mean Scores[a] on Items of the Current Section of the SADS That Differed Significantly for Patients with Situational Major Depressive Disorder and Patients with Nonsituational Major Depressive Disorder[b]

Item	Situational (N=45)	Non-situational (N=45)	F[c]	ϵ
Not differentiating criterion				
Distinctiveness of episode	1.51	2.33[d]	17.33	.39
Patient thinks stress is related	3.27[d]	2.03	36.83	.55
Outpatient treatment	2.18	2.69[d]	10.30	.31
Lack of specific concerns	2.59	4.31[d]	24.45	.46
Number of suicide attempts	1.36	2.27[d]	5.83	.32
Weight loss	2.47	3.38[d]	6.69	.25
Somatic preoccupation	1.78[d]	1.33	4.08	.18
Loss of interest	4.00	4.73[d]	5.20	.21
Subjective anger	3.62[d]	2.87	5.66	.22
Reactivity of mood	3.38	4.11[d]	4.02	.18
Mood worse in morning	1.38	1.98[d]	6.06	.23
Antisocial behavior	1.13	1.42[d]	5.14	.21
Differentiating criterion				
Stress	4.20[d]	2.65	26.48	.49

[a]Scale of 1–6.
[b]Data from 150 cases in the pilot study, 120 items contrasted.
[c]F value of 3.96 significant at the .05 level, two-tailed.
[d]Most psychopathological score.

fied by the two sets of criteria, the results differ depending on the definition used. For example, there was no difference in the overall assessment of the severity of illness (Global Assessment Scale rating) and only one significant difference in the summary scale scores between the situational and the nonsituational subgroups, although one might expect that situational depressions (often called "neurotic depressions") would show less severe impairment. In contrast, the patients classified as endogenous were significantly more impaired than were those classified as nonendogenous, as would be expected. These findings support the separate use of these two clinical features in subtyping depressive episodes and are in keeping with Klein's focus (12) on phenomenology and suggested name change from endogenous to endogenomorphic depression.

Prognostic Implications of Endogenous Features

Another hypothesis we tested in a preliminary fashion was that patients with endogenous major depressive disorder will have a good prognosis in that they will have less symptomatology subsequent to episodes of illness than will patients with nonendogenous major depressive disorder. Two-year follow-up data on 33 patients with an index episode of endogenous major depressive disorder and 21 patients with an index episode of nonendogenous major depressive disorder did not confirm this hypothesis. If anything, the trends are in the opposite direction; more of the nonendogenous patients had GAS scores above 60 (indicating minimal or no symptomatology or impairment in functioning) than did the endogenous patients (81% versus 72%). Furthermore, 39% of the endogenous patients were unable to work at the time of follow-up because of psychopathology, whereas only 24% of the nonendogenous patients were so impaired.

Again, although the sample sizes are small, these findings call into question a clinical assumption frequently repeated in the literature regarding the prognostic significance of endogenous features.

CONCLUSIONS

The studies reported here demonstrate that the conjoint use of two newly developed procedures, the Research Diagnostic Criteria (RDC) and the Schedule for Affective Disorders and Schizophrenia (SADS), have considerable potential for the testing of various hypotheses regarding the classification of affective disorders. Although the sample sizes are small, the data from this pilot study of the Collaborative Program on the Psychobiology of Depression raise questions about widely held assumptions regarding cross-sectional symptomatology, course, and the relationship between subtypes of affective disorders.

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Neurotic Depressions: A Systematic Analysis of Multiple Criteria and Meanings

BY GERALD L. KLERMAN, M.D., JEAN ENDICOTT, PH.D., ROBERT SPITZER, M.D., AND ROBERT M.A. HIRSCHFELD, M.D.

Neurotic depression, the most commonly used psychiatric diagnosis, has multiple meanings that are often used interchangeably in clinical practice. The authors identify six different meanings of neurotic depression and present data from a study of 90 depressed inpatients to determine how many patients met several different criteria; 16 patients met four sets of criteria. The overlap that exists between the different meanings is higher than chance alone but not sufficiently high to allow complete interchangeability. Until new diagnostic classes are developed, the authors recommend that the term "neurotic depression" no longer be used clinically because of its vagueness.

AMONG THE AFFECTIVE disorders the nosologic status of "neurotic depressions" remains uncertain and controversial. Whereas considerable progress has been made in the past two decades in clarifying the status of other affective disorders, especially manic-depressive illness, neurotic depression remains a diagnosis of theoretical unclarity and clinical confusion. For manic-depressive illness the range of symptoms has been well described, and two main clinical forms have been distinguished—unipolar and bipolar. The validity and utility of the unipolar-bipolar distinction has been demonstrated by correlations with familial and genetic patterns, with response to treatment (especially to lithium and the tricyclic antidepessants) and biochemical and psychophysiological processes, and with clinical course and outcomes (1). However, manic-depressive illness occurs in only about 10%–15% of patients with affective disorders. Comparable data are not available for the neurotic depressions, and the absence of such information contributes to confusion in theory, research, and clinical practice.

Depressive neurosis (*DSM-II* code 300.4) is a very frequent clinical diagnosis, probably the most common diagnosis made among the affective disorders (2) (NIMH Biometric Division Report, 1975). In addition to its high treatment rate, the epidemiologic evidence from community surveys indicates a high prevalence of untreated cases in adult populations, especially among women (3). Another consistent finding from epidemiologic studies is that the neurotic forms of depression are far more common than manic-depressive illness or other psychotic forms of depression (4).

Even with the clinical frequency of diagnosis and the epidemiological evidence for high prevalence of depressive neurosis, there continues to be a lack of clarity and precision as to the range of phenomena to be subsumed by the nosologic class and vagueness as to the criteria for the diagnosis. This contributes to a relatively low reliability of diagnostic agreement among clinicians—in clinical settings if not in research efforts. Spitzer and Wilson (5), in reviewing diagnostic agreement among various clinical conditions; found the interrater reliability coefficients for neurotic depression to be .37, far lower than those achieved for mental deficiency, organic brain syndromes, or schizophrenic psychoses, all of which ranged above .5.

Disagreement exists about not only the diagnostic criteria but also the specificity of treatment. Those clinicians who presume that neurotic depressions have a psychosocial or psychodynamic etiology consider psychotherapy the treatment of choice (6, 7). Other observers, alleging that neurotic states including depression have high rates of spontaneous remission, question the necessity, desirability, or social value of psychotherapeutic intervention (8).

Comparable disagreement exists about the role of

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drug treatment. Recommendations regarding the value of drug treatment for neurotic depressions tend to follow the clinicians' and investigators' conceptions of the nature of the disorder. Those clinicians favoring psychogenic causation tend to be cautious, if not negative, toward the use of drugs. When drugs are prescribed by them, diazepoxide derivatives and other antianxiety drugs are used. In contrast, clinicians who regard neurotic depressions as mild forms of affective disorders are more prone to use drug treatment and to prescribe the tricyclic antidepressants or the MAO inhibitors (9). Although early research studies on antidepressant drugs and many Food and Drug Administration package inserts for tricyclics assert the specific value of tricyclic antidepressants for psychotic forms of depression, clinical experience and recent research trials increasingly support the value of tricyclics for neurotic depressions, and there has been a resurgence of interest in MAO inhibitors in atypical neurotic depressions—those with mixed anxiety or phobic features.

DEFINITIONS OF CRITERIA FOR NEUROTIC DEPRESSION

Reviewing these divergencies in diagnostic practice and treatment recommendations has led us to identify a number of different meanings for neurotic depression:

1. *Neurotic depressions are less socially incapacitating.* This usage is synonymous with a judgment of mild severity of social dysfunction. These depressions allow the individual to continue his or her social functioning, although there may be personal distress and inner misery.

2. *Neurotic depressions are nonpsychotic.* In this usage, neurotic depression is a residual category for those patients not showing psychotic features and is contrasted with a psychotic depression; the patient is considered to have a neurotic depression if there is an absence of hallucinations, delusions, confusion, memory impairment, or other signs of impairment of reality testing and intactness of higher mental functions.

3. *Neurotic depressions do not present endogenous symptoms.* This usage defines a clinical picture without "endogenous" symptoms, i.e., early morning wakening, weight loss, retardation, guilt. Some observers have asserted that in neurotic depression there is not only the absence of the endogenous symptom pattern but also the presence of a characteristic constellation of symptoms of its own, with self-pity, irritability, reactivity, and fluctuating symptoms (labeled the self-pitying constellation) (10).

4. *Neurotic depressions follow a stressful event that is usually, but not exclusively, psychosocial in nature.* This usage is synonymous with "situational depression" or "reactive depression." It is presumed that the stressor is the immediate proximate or contributing cause and has temporarily overwhelmed the previously normal individual's capacity to cope and adapt. Such depressions are seen as extensions of normal states, quantitatively rather than qualitatively different from the normal reactions of loss, separation, disappointment, and other precipitants of a normal mood shift.

5. *Neurotic depressions are the consequences of a long-standing maladaptive personality pattern.* These depressions represent merely the latest "ripple" on a long-standing wave of personality inadequacies and social maladaptations. This type of depression is sometimes called "characterological depression" (11) or "depressive personality." This concept emphasizes predisposition, in terms of long-standing preexisting personality structure and character pathology of the patient.

6. *Neurotic depressions are the result of unconscious conflicts, according to psychoanalytic theory.* These depressions depend on four basic factors: 1) mood changes following interpersonal loss, disappointment, or deprivation; 2) a fall in self-esteem; 3) conflicts over the aggressive drive; and 4) a premorbid personality structure involving narcissism, dependency, and ambivalence (6).

Because of these multiple alternative criteria, some investigators and clinicians have recommended discarding the diagnostic term (12). For example, the criteria developed by the Washington University group in St. Louis do not include neurotic depression (13). Furthermore, draft versions of *DSM-III* have eliminated "neurotic depression" and have instead classified such cases as "major depressive disorder," "chronic depressive disorder," "atypical depressive disorder," or "adjustment disorder with depressed mood."

Patients may receive a diagnosis of neurotic depression on the basis of any or all of these criteria. The implications as to etiology, clinical course, response to treatment, family history, psychosocial aspects, and other relevant variables of using one criterion set rather than another are unknown. In fact, even the approximate likelihood of meeting one criterion given that another has been met is unknown. For example, if a depression is nonpsychotic will it necessarily also be reactive? This paper addresses some of these issues. Specifically, a group of depressed patients was carefully diagnosed according to the Research Diagnostic Criteria (RDC) (14). The degree of overlap among the various neurotic depression groups was assessed and personality, symptomatic, social behavior, and other characteristics of the group were examined. It should be noted that the study focused on only the first four criteria and did not include assessments of long-standing maladaptive personality patterns.

METHOD

The data reported here derive from the pilot study conducted by the Clinical Studies group of the NIMH-

Clinical Research Branch Collaborative Program on the Psychobiology of Depression. The research groups at the four participating centers used a common protocol.

At the four centers consecutive admissions to the adult inpatient units were screened for depression and mania by a research assistant on the basis of a verbal report of mood, behavior, and associated symptoms. They were then evaluated with the Schedule for Affective Disorders and Schizophrenia (SADS) (15) and were diagnosed with the Research Diagnostic Criteria (14). Those patients who received a diagnosis of major depressive disorder were further diagnosed into the following nonmutually exclusive subcategories (as well as several others):

1. Psychotic major depressive disorder—must contain delusions, hallucinations, or depressive stupor (mute and unresponsive).

2. Incapacitating major depressive disorder—either A or B is required.

Group A. Because of severity of symptoms, the patient is *unable to function* at work or at school, or take care of the house for at least one week (or if hospitalized was so *impaired* that *obviously* could not work). (Do not count mere refusal to do tasks.)

Group B. Because of severity of symptoms, the patient is *unable* to feed or clothe himself or maintain minimal personal hygiene without assistance.

3. Endogenous major depressive disorder—from groups A and B a total of at least four symptoms for probable, six for definite, including at least one symptom from group A.

Group A. 1) Distinct quality to depressed mood, i.e., depressed mood is perceived as distinctly different from the kind of feeling the patient would have or has had following the death of a loved one; 2) lack of reactivity to environmental changes (once depressed, the patient does not feel better, even temporarily, when something good happens); 3) mood is regularly worse in the morning; 4) pervasive loss of interest or pleasure.

Group B. 1) Feelings of self-doubt or reproach or excessive or inappropriate guilt; 2) early morning awakening or middle of night insomnia; 3) psychomotor retardation or agitation (more than a mere subjective feeling of being slowed down or restless); 4) poor appetite; 5) weight loss (two pounds a week over several weeks or 20 pounds in a year when not dieting); 6) loss of interest or pleasure (may or may not be pervasive) in usual activities or decreased sexual drive.

4. Situational major depressive disorder—illness has developed after an event or in a situation that seems likely to have contributed to the appearance of the episode at that time. In making this judgment consider the amount of stress inherent in the event or situation, the cumulative effect of such stresses, and the closeness of the events to the onset or exacerbation of the depressive episode.

RESULTS

Sample Characteristics

A total of 150 patients (98 women and 52 men) were selected from the four participating hospitals. The median age was 33 years. The sample was predominantly white (82%). By religious background, 22% of the sample was Protestant, 45% was Catholic, 6% was Jewish, and 27% was unaffiliated. Two-thirds of the sample had been married, and one-third was single. Of those who had been married, however, a substantial percentage (27%) of the total sample was divorced or widowed. The sample was relatively well educated, 50% having had more than a high school education.

Of the 150 patients, the data analysis will focus on the 90 who had major depressive disorder. The remaining were schizo-affective, had a manic episode, or had a depressive syndrome not meeting the criteria for major depressive disorder.

Of the 90 patients with major depressive disorder, multiple noninclusive diagnostic assessments were made according to the RDC criteria on a number of different subtypes of current psychiatric interest (see table 1). Diagnoses were made independently by each of two clinicians on the basis of an interview conducted by one and silently observed by the other. Interrater reliabilities were very high (kappa=0.80) (14).

Relationship Among Alternative Diagnoses

Of the 90 patients with major depressive disorder, 42%, 53%, and 50% did not meet the criteria for endogenous major depressive disorder, psychotic major depressive disorder, or incapacitating major depressive disorder, respectively. Fifty percent were diagnosed as meeting the criteria for situational major depressive disorder.

The results clearly indicate that the alternative diagnostic criteria for neurotic depression yield different, although overlapping, groups of patients. This may seem a trivial finding; however, many clinicians and researchers use this term to refer to any or all of the groups and presume that they are identical.

Of the 38 patients who did not evidence endogenous clinical features, 79% were nonpsychotic, 66% were not socially incapacitated, and 68% had a clear precipitant. In parallel, of those patients who did not have psychotic symptomatology (N=67), less than half (45%) were nonendogenous, about two-thirds (63%) were not socially incapacitated, and about half (54%) had a precipitant. Of the nonincapacitated depressive patients (N=48), 52% were also nonendogenous, 88% were, not surprisingly, nonpsychotic, and 58% had a precipitant. Finally, of the 45 situational depressives, 58% were nonendogenous, 80% were nonpsychotic, and 62% were nonincapacitating.

Overall, the degree of overlap between any two groupings ranged between half and two-thirds, with the exception that it was rare for a psychotic depressive to be found among the other three groupings.

Conversely, among the 52 patients with endogenous

TABLE 1
Subtypes of Major Depressive Disorder (N=90) for the Present Illness*

Subtypes of Major Depressive Disorder	N	%
Primary	59	66
Secondary	31	34
Incapacitating	42	47
Endogenous	52	58
Agitated	17	19
Retarded	23	26
Situational	45	50
Simple	57	63

*Diagnoses were counted as present if they were rated probable or definite.

symptoms, 37% were also diagnosed as having a situational precipitant. This goes against one of the major classical criteria for endogenous depression, which holds that the depression comes on in the absence of a situational life stress. Of the 90 patients with major depressive disorders, only 18% (N=16) met all four criteria for neurotic depression.

DISCUSSION

The findings confirm that the various criteria for neurotic depression are not interchangeable. Some degree of overlap of patient groups does exist; this degree is greater than would be expected by chance alone, but it is not sufficiently strong to allow complete interchangeability.

It is important to underscore that the lack of agreement among the various definitions of neurotic depression is not due to observer variance. As previously noted, the interjudge symptomalogic and diagnostic reliabilities were very high. This was due in large part to the use of a semi-structured clinical interview, explicit diagnostic criteria, and training programs. The degree of agreement is far above that reported by Spitzer and Wilson in their review of previously published literature on neurotic depression (5). This confirms the expectation that when explicit criteria are used, the meanings of neurotic depression are specified, and the clinical interviewers are trained, the agreement rises substantially into acceptable levels. Thus, the lack of agreement simply reflects the fact that these various criteria define different groups.

Several prior investigators have developed affective nosologies based on clusters of cross-sectional symptoms. Overall (16), for example, has proposed three depressive types. Two of the three types (namely, anxious and hostile) might be regarded as variants of neurotic depression. Paykel (17), using a cluster analysis technique, devised a four-fold typology, three of which might also be regarded as neurotic (nonpsychotic). Derogatis (18), using factor analysis, derived three types of neurotic affective disorder to include patients with a mixture of anxiety and depression. The Derogatis typology is based on data from patient self-reports, the Hopkins Symptom Checklist.

After a number of alternative diagnostic groups subsumed under the rubric of "neurotic depression" have been identified, these groups can be compared as to symptom profile, family background, follow-up study, or personality profiles. These diagnostic schemes, in addition to the one described in this paper, were developed on the basis of symptomatology and, to some extent, psychiatric history. With the exception of the Overall system, for which there were some differential drug response data reported, there are no validation data such as clinical course, follow-up, familial psychopathology, or personality data. Such research is now underway as part of the clinical studies of the NIMH-Clinical Research Branch Collaborative Program on the Psychobiology of Depression.

Our objection to the term neurotic depression involves theoretical as well as empirical criticisms. It has tended to carry with it various etiological assumptions. For the most part, clinicians have regarded neurotic depression as having nonorganic (i.e., psychosocial, environmental) causes. However, there has been divergence as to whether these causes are to be found in acute situational stress, precipitating situational reactions, or long-standing maladaptive personality features. The results provided here, in addition to studies already cited in the literature, lead to the conclusion that these alternative meanings, while they share a common view as to the psychosocial environmental origin of neurotic depression, are sufficiently diverse as not to be interchangeable or identical. Therefore, we recommend that in the search for etiological bases for alternative meanings to neurotic depression, relatively homogeneous groupings are required to test hypotheses about the antecedent causal relationship of the symptom pattern to postulated causes such as life stress, personality, or social change.

CONCLUSIONS

A test of the interrelationship of four of the different meanings (nonpsychotic, nonendogenous, nonincapacitating, situational) indicates only a modest degree of overlap. We therefore conclude that the value of using the term neurotic depression is limited.

The results of this study have influenced and supported the decision of the American Psychiatric Association's Task Force on Nomenclature and Statistics to drop the category of neurotic depression and instead to enable the clinician to characterize depressive disorders along the following dimensions, using a multiaxial approach: 1) course (episodic versus chronic) (Axis I); 2) presence or absence of bipolarity (Axis I); 3) severity (including psychotic or not) (Axis I); 4) personality pattern or disorder (Axis II); 5) presence of life stress (Axis IV); 6) highest level of adaptive functioning in the past year (Axis V).

This approach should enhance precise communication among clinicians and researchers and allow more

precise determinations of the relationship of subtypes of the heterogeneous group of disorders previously called neurotic depression to treatment outcome, genetics, and influence by life stress or other psychological factors.

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Secondary Depression: Familial, Clinical, and Research Perspectives

BY NANCY C. ANDREASEN, M.D., AND GEORGE WINOKUR, M.D.

The authors evaluate the clinical and research significance of the diagnosis of secondary depression by comparing 48 cases of primary and 26 cases of secondary depression. The patients with secondary depression have a higher familial prevalence of alcoholism, affective disorder, and drug abuse. The groups differ somewhat on a few sociodemographic, behavioral, and attitudinal variables but are similar in symptomatology, sex ratio, onset and duration of symptoms, treatment received, and response to treatment. These results suggest that the distinction between primary and secondary depression should be retained in research that examines neurochemistry or genetics. Primary and secondary depression appear to be identical from the perspective of clinical care. Management of these patients should emphasize the diagnosis of depression rather than antecedent diagnoses.

BECAUSE PERIODS of sadness or grief are ubiquitous in human experience and because sadness is often accompanied by such typical depressive symptoms as sleeplessness or loss of interest, the concept of depression can be overinclusive and heterogeneous. Researchers interested in studying the pathophysiology or pharmacology of depression need techniques for defining depression that maximize the homogeneity of the groups under investigation, for otherwise specific findings may remain undiscovered when data are pooled and averaged. The concept of secondary depression was originally developed as one method of narrowing for purposes of research the concept of depression.

As Woodruff pointed out in one of the earliest definitions of secondary depression, the use of the natural history of illnesses to clarify nosology has impeccable historical and theoretical precedents that reach back to Sydenham (1). Drawing on a distinction originally made by Munro (2), Woodruff proposed that the broad category of depressive disorders be broken down into two mutually exclusive groups, primary depression and secondary depression, on the basis of natural history. Primary depression was defined as a depressive syndrome occurring in a patient with no prior history of any other psychiatric illness; secondary depression was defined as a depressive syndrome occurring in a patient who had an antecedent disorder. This classification system was based on the recognition that depressive syndromes are frequent accompaniments or consequences of particular psychiatric disorders, such as alcoholism, anxiety disorder, or hysteria. Woodruff and his associates anticipated that the course and prognosis of secondary depression would resemble that of the antecedent disorder and that it might require a different type of treatment than primary depression. Because of the potential influences of the various antecedent diagnoses on course and treatment, secondary depression was thought to be a less "pure" disorder than primary depression.

Perhaps because secondary depression is probably heterogeneous and therefore more difficult to study, only a modest number of investigations of this disorder have been completed since its initial definition (3–8). These investigations have suggested a number of methodologic problems inherent in its study. Should it be defined as secondary only to psychiatric disorders, or should depressions occurring in the context of other medical illnesses such as cancer also be considered as secondary? The latter alternative produces a concept of secondary depression that may be so broad as to be meaningless; consequently, the disorder should probably be defined as secondary only to psychiatric disorders. When a patient initially identified as having a primary depression later develops alcoholism, should depressions developing subsequent to the alcoholism be considered primary or secondary? To change the diagnosis to secondary seems illogical, but investigators doing biochemical or pharmacological studies

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may wish to exclude such patients from their studies in order to be sure that the sample is as homogeneous as possible. Wood and associates have also suggested that a third category, primary complicated affective disorder, may be defined to describe such cases (5). Should investigations of secondary depression further subdivide patients on the basis of their antecedent diagnoses? Since the concept of secondary depression mixes patients who have very different antecedent diagnoses, such as antisocial personality and obsessive-compulsive disorder, perhaps it should be studied on the basis of several different subgroupings, at least whenever a large enough number of patients can be accumulated. Recently Weissman and associates (6) completed an investigation that compared secondary depression in alcoholism, drug addiction, and schizophrenia and found some differences in symptom patterns.

While the usefulness of the concept of secondary depression for research has become widely accepted and various definitional issues have slowly been resolving, the clinical significance of secondary depression has received very little attention. A category that has heuristic value for purposes of research may not have heuristic value clinically. Consequently, this paper will explore the clinical utility of the concept of secondary depression. Does it differ from primary depression in symptomatology, onset, duration, and other related variables? Should it be treated in terms of cross-sectional symptomatology, or should the underlying disorder be the focus of attention and treatment?

METHOD

A subsample of patients diagnosed as having unipolar major depressive disorder were selected from the patients examined in the four centers participating in the NIMH-Clinical Research Branch Collaborative Program on the Psychobiology of Depression (Harvard Medical School, Boston; University of Iowa College of Medicine, Iowa City; New York State Psychiatric Institute, New York; Washington University School of Medicine, St. Louis).

All patients were hospitalized and met screening criteria for a depressive or manic syndrome. A total of 150 index cases were examined by investigators from the four participating centers: 50 each at Boston and St. Louis, and 25 each at New York and Iowa City. In addition, investigators from New York and Iowa City interviewed all available first-degree relatives, a total of 88. Diagnoses of these probands were made by using the Schedule for Affective Disorders and Schizophrenia (SADS) (9) and the Research Diagnostic Criteria (RDC) (10). The first-degree relatives were interviewed with the SADS-L, a lifetime version of the SADS, and diagnosed through the RDC. All probands and available first-degree relatives were also interviewed concerning family history by investigators who used the Family History-Research Diagnostic Criteria (FH-RDC) (11). These instruments and criteria have excellent reliability.

In addition to these interviewing instruments, which provided information about course and symptoms, the patients were studied comprehensively in a variety of other ways. The Personal History for Depressive Disorders (PHDD) (G. Hogarty, unpublished data) provided a wide range of sociodemographic information, such as parental occupation, marital status, income, and education. The Symptom Checklist-90 (SCL-90) (12) was completed by each patient and provided a self-report inventory of symptomatology. These assessments were completed within the first week after admission. The SADS and RDC were updated at discharge so that new material collected since the initial interview could be incorporated, and data were also collected at this time concerning response to treatment. Data were analyzed with t test and chi-square programs from the Statistical Package for the Social Sciences (13).

RESULTS

A total of 83 patients received a diagnosis of unipolar major depressive disorder for either a current or past episode, but only 74 currently had unipolar major depressive disorder. Because we wished to study depressive phenomenology during the current episode, we included only those 74 in our investigation. Of these 74, 48 had a primary depression, and 26 had a secondary depression. Two patients whose only antecedent diagnosis was a nonpsychiatric medical illness were classified as primary. Patients who by history had a nondepressive diagnosis such as alcoholism interposed between an initial and a current depression were considered as secondary because of the possible unreliability of the depressive history. Among the patients with secondary depression, the majority of the antecedent diagnoses were personality and "acting out" disorders: Briquet's syndrome, 32%; alcoholism, 36%; drug abuse, 23%; and antisocial personality, 14%. Only a few patients had depressions secondary to disorders characterized by high levels of anxiety and internalization: panic disorder, 14%; phobic disorder, 9%; and obsessive-compulsive disorder, 5%.

The SADS interview may be subdivided into eight different scale scores on the basis of ratings on individual items (depressive mood and ideation, endogenous features, associated features, suicidal ideation and behavior, anxiety, manic syndrome, delusions/hallucinations, and formal thought disorder). Scores obtained by the patients with primary and secondary depression were strikingly similar. In order to further test the hypothesis that primary depression might have more endogenous features, the two groups were also compared on individual items from that scale (feelings of guilt, early morning awakening, loss of appetite, weight loss, poor concentration, worse in morning, loss of interest, and psychomotor retardation). The

two groups differed significantly at the .05 level (chi-square analysis, Yates's correction) only on weight loss (secondary depression higher) and on psychomotor retardation (primary depression higher). These results confirm the scale score data indicating that the two groups do not differ in any meaningful way in their amount of endogenous phenomenology.

The groups differed on a number of behavioral and attitudinal variables, based on the SADS interview and the SCL-90. Analyses were done with Student's t test. The patients with secondary depression tended to have more suicidal behavior (p=.04) but the attempts that the patients with primary depression made were more serious and life-threatening (p=.05). Unlike the data on endogenous phenomenology, these data imply that primary depression may indeed be more severe than secondary depression. The data from the SCL-90 indicate that patients with secondary depression complain of a number of self-report symptoms that probably reflect their underlying or antecedent condition. They report more somatization (p=.001), anger and hostility (p=.04), phobic anxiety (p=.01), trouble falling asleep (p=.006), and "psychoticism" (i.e., unusual or bizarre symptoms) (p=.002). This mixture of "neurotic" and nonneurotic complaints probably reflects the heterogeneity of the underlying conditions.

Table 1 summarizes family history and family study data concerning type and amount of psychiatric illness in the patients' first-degree relatives. We hypothesized that secondary depression should have a higher familial prevalence of alcoholism and personality disorders as Guze and associates (3) had observed previously. As table 1 indicates, this hypothesis was confirmed, and the two groups in fact differ rather strikingly in their familial history of illness. When the data are examined in terms of numbers and percentage of first-degree relatives who have psychiatric illness, patients with secondary depression have significantly more relatives with any diagnosis, with affective disorder, with alcoholism, and with drug abuse. Alternatively, the data may be examined in terms of the percentage of patients coming from families with a positive history of psychiatric illness, and in this instance 89% of the patients with secondary depression come from families with a more positive history for affective disorder and/or alcoholism compared with 56% of the patients with primary depression. There is a trend in the direction of patients with secondary depression having more affective disorder alone. If familial loading for psychiatric illness can be considered as an index of potential severity, then it is secondary depression that is more severe.

Table 2 compares the two groups on a number of sociodemographic and course variables. In general the two groups tend to differ in directions that are expected or that have been previously noted, but very few of these differences are statistically significant. In fact, only the difference in the father's education is statistically significant (p<.05, chi-square analysis). Some of the trends support differences noted in previous studies: patients with secondary depression are more often male, have an earlier age of onset, and have a higher divorce rate. Since those variables are frequently used to support the idea that the nature and course of secondary depression is more like that of the antecedent disorder than like that of primary depression, it is noteworthy that this investigation finds trends at most and no major statistically significant differences on these sociodemographic and course variables.

When the treatments that the two groups received and their response to treatment were examined, we found no statistically significant differences in the

TABLE 1
Psychiatric Illness in First-Degree Relatives of Patients with Primary and Secondary Depression

Diagnosis	Patients with Primary Depression N	%	Patients with Secondary Depression N	%	χ^2	p
First-degree relatives	262		131			
Any diagnosis	66	25	60	46	17.03	.001
Affective disorder	27	10	25	19	5.86	.05
Alcoholism	14	5	17	13	7.00	.01
Drug abuse	2	1	7	5	8.19	.005
Families	48		26			
Affective disorder and/or alcoholism	27	56	23	89	7.99	.005
Affective disorder only	12	25	12	46	3.44	.10
Alcoholism only	9	19	5	19	.002	n.s.

TABLE 2
Sociodemographic and Course Variables in Primary and Secondary Depression

Variable	Primary (N=48)	Secondary (N=26)
Father's education*		
College	22	11
High school	22	58
Grade school	56	31
Mother's education		
College	12	26
High school	48	58
Grade school	40	16
Proband's education		
College	42	54
High school	49	42
Grade school	9	4
Parents divorced/separated	15	15
Proband divorced/separated	22	35
Males	22	31
Onset before 40**	78.6	88.5
Recovering from current episode***	84.4	69.2

*χ^2=7.03, p<.03.
**Mean age at onset was 30 years for patients with primary depression and 27 years for those with secondary depression.
***Mean duration of current episode was 26.9±29.6 weeks for patients with primary depression and 37.9±39.8 for those with secondary depression.

treatments received. Since treatment was not a variable under controlled evaluation in this study, treatment was determined by the decision of the primary ward physician, not by any of the investigators conducting the collaborative studies. Consequently, we can conclude that the clinicians responsible for the care of these patients saw no differences in them sufficient to warrant significantly different treatment approaches, although in general the patients with primary depression received slightly more ECT, antidepressants, lithium, and minor tranquilizers.

A summary of the follow-up data obtained on these patients approximately two years after their inclusion in the study indicates that a total of 31 patients with primary and 13 with secondary depression were reached and interviewed directly. In addition, 3 of those with primary and 1 with secondary depression had died. The two groups differed significantly only in terms of the number on medication at the time of follow-up (70.9% of those with primary depression still on medication 1 year later compared with only 30.7% of the secondary depression patients, $p=.03$). Trends suggest that more patients with primary depression may be working and more with secondary depression make subsequent suicide attempts, but these trends do not reach statistical significance. Otherwise the two groups are strikingly similar.

DISCUSSION

The clinical and research significance of secondary depression is not a trivial issue. Secondary depression is a very common diagnosis both on inpatient services and in outclinics. Although reports vary, the frequency of secondary depression among patients with major depression ranges from 35% in this investigation to 49% in the study of Wood and associates (5). The frequency is probably even higher in some outclinics.

The distinction between primary and secondary depression was originally developed as a way of purifying the category of depressive disorders for the purpose of research. Secondary depression was of interest only for purposes of exclusion. When it was discussed from a clinical perspective, the antecedent diagnosis was assumed to be of more clinical importance as a predictor of prognosis and response to treatment than the depressive symptomatology.

This investigation raises the possibility that that assumption may be at least partially incorrect. On most measures primary and secondary depression are more similar than they are different. Earlier investigations that found differences in sex ratio and age of onset have not been replicated, and the two groups look alike in severity of symptomatology, amount of endogenous phenomenology, most sociodemographic variables, duration of symptoms, response to treatment, and course at follow-up. There is a marked difference in familial psychopathology, with secondary depression having substantially more than primary depression, including affective disorder as well as alcoholism and drug abuse. These familial data are open to a variety of interpretations, but from a clinical perspective they tend to suggest that secondary depression is potentially more severe and familial (but not necessarily genetic) than primary depression. This investigation also suggests that secondary depression tends to be more colorful and florid, with patients making more suicide attempts, showing more "psychotic" symptoms, and complaining more of phobias, anger, difficulty falling asleep, and somatic problems. These findings imply that the picture of secondary depression may be colored by its antecedent or underlying condition.

Concerning the appropriate clinical approach to secondary depression, the results of this investigation are somewhat equivocal. They suggest provisionally that it is perhaps best to conceptualize secondary depression as an acute illness superimposed on a more chronic one. The acute symptomatology, depression, should be regarded as the immediate problem, and often the management may be no different than if it were not complicated by another underlying chronic problem. This approach is not only suggested by our data but is also the one used by our medical colleagues in most other specialties. Having one illness usually does not protect a person from developing another. In fact, the reverse is usually true, and there are often recognized relationships between medical diagnoses. Often an acute condition is a well-recognized and feared complication of a more chronic one: subacute bacterial endocarditis as a complication of rheumatic heart disease, acute leukemia as a complication of mongolism, recurrent urinary tract infection as a complication of spina bifida, pneumonia as a complication of chronic lung disease, atelectasis as a complication of asthma. Depression developing in a person with alcoholism or obsessive-compulsive disorder is quite analogous. From a clinical perspective, the most sensible course at present may be to assume that the person has two conditions, that his management is therefore somewhat more complicated and difficult than if he had only one, and that he should perhaps be treated in one way for the acute condition and in another for the chronic one.

While a clinical perspective stresses the similarities between primary and secondary depression, a research perspective continues to stress the importance of maintaining the distinction. Analogies with other medical disorders also make it quite clear that illnesses may be phenomenologically similar or identical and yet have quite a different pathogenesis. Particularly in view of significant differences between primary and secondary depression in familial psychopathology, investigators interested in the genetics, pharmacology, neurophysiology, or neurochemistry of depression should continue to exclude patients with secondary depression from study or to study them separately in order to maximize homogeneity in the group under investigation.

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Personality Attributes and Affective Disorders

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To determine the personality characteristics associated with affective disorders the authors administered a battery of self-report personality inventories to a sample of hospitalized affective patients when their manifest symptoms had abated. Patients were instructed to answer according to their premorbid personalities. The personality characteristics assessed in the 73 depressive and 24 manic patients included neuroticism and extraversion from the Maudsley Personaltiy Inventory, obsessional pattern, hysterical pattern, and oral pattern from the Lazare-Klerman-Armor Personality Inventory, obsessional state and trait from the Leyton Obsessionality Inventory, and solidity, stability, and validity from the Marke-Nyman Temperament Survey. Depressive patients demonstrated more neuroticism, introversion, and obsessionality than manic patients or normal individuals. The manic patients differed from normal persons only on obsessionality.

THE ROLE of personality in depression and mania has been given considerable attention by the psychiatric community. In a recent review of psychoanalytic contributions Chodoff (1) concluded that undue interpersonal dependency and obsessionality often predispose to depression. Several newer approaches from cognitive and behavioral theory have also been applied to depression. Most notable have been works by Beck (2), Seligman (3), and Lewinsohn (4).

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This work was done as part of the NIMH-Clinical Research Branch Collaborative Program on the Psychobiology of Depression: Clinical Studies and was completed with the cooperation and participation of the Collaborative Program investigators: Martin M. Katz, Ph.D., and Gerald L. Klerman, M.D., Co-Chairmen; Robert M.A. Hirschfeld, M.D., Coordinator; Martin B. Keller, M.D., Robert W. Shapiro, M.D., and James Barrett, M.D. (Boston); Jan A. Fawcett, M.D., and William A. Scheftner, M.D. (Chicago); George Winokur, M.D., and Nancy C. Andreasen, M.D. (Iowa City); Robert L. Spitzer, M.D., and Jean Endicott, Ph.D. (New York); and Eli Robins, M.D., Paula J. Clayton, M.D., Theodore Reich, M.D., Amos Welner, M.D., and Jack Croghan, M.D. (St. Louis).

In contrast to these predispositional theoretical approaches, two alternate formulations about the relationship of personality and depression may be considered:

1. Certain personality features and certain psychiatric disorders may be manifestations of the same underlying process, be they genetic, developmental, familial, or other. That is, a personality trait may represent a subclinical expression of a psychiatric disorder (e.g., cyclothymia and bipolar disorder). If this trait has a familial or genetic basis, we would expect to find increased levels of the trait in relatives, both affected and nonaffected, as well as in patients.
2. Personality qualities may be altered by the experience of the affective disorder. Chronic diseases, either medical or psychiatric, often lead to changes in individual personality patterns. The extent to which this may occur in acute, chronic, and current depressions is at present unknown.

In spite of the considerable clinical and theoretical interest, surprisingly little controlled empirical research has been conducted in this area. Probably the major reason for this is methodologic difficulties, especially the lack of reliable and valid assessment procedures for diagnosis and for personality. Relatively few empirical studies have addressed the association of personality variables and depressive syndromes, and even fewer have dealt with mania. Interpretation of even these studies is difficult due to a lack of standardized diagnostic assessment tools and variation in personality variables.

A necessary first step in testing these or other formulations about the relationship between personality and depression is careful objective assessment of personality traits in sufficiently large samples of depressed patients. In this paper we describe a study addressing this issue: specifically, ascertainment of personality characteristics in a carefully diagnosed sample of patients with affective disorders.

PATIENT SAMPLE AND METHOD

Our sample included 119 depressed inpatients at four major university hospital centers (Boston, New York, St. Louis, and Iowa City) who were comprehensively assessed using the Schedule for Affective Disorders and Schizophrenia (SADS) (5) and the Research Diagnostic Criteria (RDC) (6) in addition to sociodemographic and other measures. The sample included 41 males and 78 females, had a mean age of

34, and was predominantly Caucasian. Seventy-three patients were diagnosed as having a major depressive disorder, 24 as having a manic or hypomanic disorder, and the rest as having various other diagnoses, including minor depression, schizophrenia, schizo-affective disorder, alcoholism, and drug abuse.

A research assistant administered a battery of self-report personality inventories to the patients near the time of hospital discharge when their manifest symptoms had largely abated. Because we were seeking estimates of premorbid personality levels, the research assistant explicitly instructed the patients to disregard their current state of illness and to answer the questions according to their usual self. The rationale for this procedure comes from independent studies by Kendall and DiScipio (7), Philip (8), and Foulds (9), which demonstrated that scores similar to those made during asymptomatic periods may be obtained from symptomatic depressed patients.

The personality inventories used were the Maudsley Personality Inventory, the Lazare-Klerman-Armor Personality Inventory, the Marke-Nyman Temperament Scale, and the Leyton Obsessional Inventory. The Maudsley Personality Inventory (MPI) (10) measures extraversion and neuroticism. The Lazare-Klerman-Armor Personality Inventory (11, 12) assesses three psychoanalytic personality patterns—oral, obsessional, and hysterical. The Marke-Nyman Temperament Inventory (MNT) (13) assesses three of the four Sjöbring personality factors: validity, stability, and solidity. Although relatively unknown in the United States, it has been used extensively in Europe. A version suitable for American subjects was developed by Barrett and Nyman (14). The Leyton Obsessional Inventory (15) measures two aspects of obsessionality: symptoms (as in obsessional neurosis) and trait (as in obsessional character).

RESULTS

The depressive patients differed substantially both from the manic patients and from the published American norms for each of the scales (see table 1). The depressive patients scored significantly higher on introversion, neuroticism, obsessionality, solidity, and stability and significantly lower on validity than the norms in the literature. The manic subjects differed from the norms only on obsessional state and obsessional trait.

In order to facilitate interpretation of these findings, a principal component factor analysis and rotation was performed on the 10 × 10 matrix of correlations of scale scores. Three factors emerged, accounting for 73% of the total variance. The factor-loading matrix was then subjected to a normal varimax rotation (see table 2). Examination of the resultant factor-loading matrix reveals that factor 1 is representative of the neuroticism, validity, orality, and obsessional state scales and might be deemed "nonspecific neuroticism and emotional weakness." Factor 2 represents solidity, hysterical pattern, extraversion, and stability and might be labeled "general sociability and suggestibility." The third factor clearly represents obsessionality. Scores were calculated for each of the depressive and manic patients. When the groups were compared using t tests, the depressive patients scored much higher than the manic patients on nonspecific neuroticism and emotional weakness ($p < .0001$), slightly lower on general sociability and suggestibility ($p < .07$), and very similar on obsessionality.

Although we had specifically instructed the patients to disregard the experience and effects of their current illness, the possibility remained of contamination of

TABLE 1
Mean Scores of Depressive and Manic Patients in Collaborative Program and Published Norms on Four Personality Inventories[a]

	Depressive Patients (N=73)		Manic Patients (N=24)		Published Norms	
Inventory	Mean	SD	Mean	SD	Mean	SD
Maudsley Personality Inventory						
Neuroticism	30.3[b,d]	10.6	19.7	14.0	20.7	10.7
Extraversion	21.2[b,d]	9.8	29.3	21.2	28.7	8.2
Marke-Nyman Temperament Survey						
Solidity	10.4[d]	3.4	9.2	4.0	8.8	3.2
Stability	8.5[b,c]	3.2	6.1	4.8	7.1	4.1
Validity	8.0[b,d]	4.1	11.8	4.2	11.8	4.2
Lazare-Klerman-Armor Personality Inventory						
Obsessional pattern	24.4	7.4	27.0[c]	6.7	23.0	7.3
Hysterical pattern	10.3	6.4	11.6	5.8	11.7	7.8
Oral pattern	20.0[b]	7.4	12.6[c]	7.4	20.0	7.2
Leyton Obsessionality Inventory						
State	20.9[b,d]	8.9	14.9[d]	9.7	10.3	6.3
Trait	10.1[d]	3.9	9.9[d]	3.8	5.1	3.6

[a]Norms for Maudsley Personality Inventory (10) based on 1,065 American students; Marke-Nyman Temperament Survey (14), 101 American normal individuals; Lazare-Klerman-Armor Personality Inventory (11), 90 American normal individuals; and Leyton Obsessionality Inventory (15), 101 normal individuals.
[b]Significantly different from manic patients ($p<.01$).
[c]Significantly different from norms ($p<.05$).
[d]Significantly different from norms ($p<.01$).

TABLE 2
Factor-Loading Matrix of Personality Variables (N=119)

	Factor Number		
Personality Variable	1	2	3
Neuroticism	0.931	0.019	−.006
Validity	−0.771	0.247	−0.020
Orality	0.766	−0.169	0.266
Obsessional symptom	0.739	0.031	0.414
Solidity	0.065	−0.795	0.230
Hysteria	0.197	0.794	0.260
Extraversion	−0.544	0.666	0.117
Stability	0.442	−0.659	0.039
Obsessionality	−0.069	0.035	0.893
Obsessional trait	0.266	−0.020	0.883

TABLE 3
Stability of Personality Variables Over Time in Depressive Patients (N=15)

Personality Variable	Index Score		Follow-Up Score		Pairwise	
	Mean	SD	Mean	SD	t	r
Extraversion	19.8	11.85	22.2	9.61	−.66	0.15
Neuroticism	28.6	15.83	22.33	16.18	1.56	0.53
Solidity	10.9	3.93	11.13	2.59	−.26	.32
Stability	8.27	3.88	8.13	3.91	.11	.30
Validity	8.47	5.55	9.8	5.05	−.93	.46
Obsessionality	24.2	7.45	24	7.94	.16	.81
Hysterical pattern	9.8	7.46	11	6.93	.77	.65
Orality	20.4	7.69	18.3	7.48	1.21	.55

the trait assessments by clinical state. So we readministered most of the personality battery two years later to those subjects who at index had suffered from either a major depressive disorder or manic disorder and who at follow-up were asymptomatic or back to their usual self. Of the 73 major depressive patients, 15 who were willing to complete the questionnaire met these criteria. The index episode personality scale scores of this subgroup of 15 were very similar to those of the whole major depressive group, which suggests that they are representative. The follow-up scores for seven of the eight measures were stable over this two-year period, with only the neuroticism score changing a great deal. Pairwise t tests and Pearson product-moment correlation coefficients provided statistical confirmation of this (see table 3).

Of the 24 manic patients, only 4 who met the criteria for the follow-up returned the questionnaire, so statistical analysis was not feasible. The result was some shifts in scores for these 4 patients; however, none of the follow-up scores approached those of the depressive patients.

DISCUSSION

Our empirical findings strongly support the notion that there are abnormalities in the personalities of patients with affective disorders. These findings agree with prior empirical work. In two studies of recovered hospitalized depression patients Kendall and DiScipio (7, 16) reported mean scale scores very similar to our study's scores on neuroticism, extraversion, obsessional state, and obsessional trait. Paykel and Prusoff (17) found nearly identical mean obsessional, hysterical, and oral factor scores in their study of 176 depressive patients. With the exception of the mean score on the stability scale, the mean scores on the Sjöbring dimensions of solidity and validity of the 62 recovered depressive patients in Coppen's study (18) were also very similar.

Further support for the validity of these findings is provided by comparing the intercorrelations among the scales in this study with those of other studies in the literature; the correlation coefficients between the prior studies and this one are nearly identical (16, 17, 19).

Overall, the results suggest that depressive patients are more likely to break down under stress, have less energy, are more insecure and sensitive, tend to worry more, are less socially adroit, are more needy, and are more obsessional than normal individuals. Manic patients, on the other hand, have much more normal personality profiles, at least as far as the assessed dimensions indicate. However, they are significantly more obsessional than normal individuals.

Our findings are also consistent with the major psychological theories of depression. Chodoff (1) concluded that depression-prone people are inordinately and almost exclusively dependent on narcissistic supplies derived directly or indirectly from other people for maintenance of their self-esteem. Their frustration tolerance is low, and they may employ various techniques—submissive, manipulative, coercive, piteous, demanding, or placating—to maintain their desperately needed relationships with external or internalized objects that provide these supplies.

In the cognitive approach put forth by Beck (2, 20) depressive patients are seen as possessing an enduring cognitive set consisting of negative attitudes and beliefs regarding the self, the world, and the future. This set causes the depression-prone person to distort his or her interpretations of experience so as to see and expect defeat, frustration, and deprivation and to regard himself or herself as inadequate, undesirable, and unworthy. Such people are highly sensitive to situations that reinforce these negative self-attitudes.

Another cognitive theory is that of learned helplessness, which was proposed by Seligman (21). He noted that if dogs are unable to prevent or alter an aversive stimulus (e.g., an electric shock), they soon elicit behavior that is strikingly similar to that of human depressive individuals, including withdrawal, apathy, and reduction in goal-directed behavior. Once conditioned, this behavior is very resistant to change, even when opportunities to avoid the aversive stimulus become available. This observation led to the development of the learned helplessness theory of depression. Seligman notes that six of the major symptoms of learned helplessness are parallel to those found in depression: 1) lowered initiation of voluntary responses, 2) negative cognitive set, 3) time course, 4) lowered aggression, 5) loss of appetite, and 6) physiological changes.

The behavioral approach to depression emphasizes the deficient instrumental behaviors of the depressive individual. Lewinsohn (4), for example, has shown that instrumental behaviors such as social skill (i.e., the ability to emit behaviors that are positively reinforced by others) are significantly less developed in depressive patients than in others. He postulates specifically that a reduction in the rate of positive behavioral reinforcements is the cause of depression. Low self-esteem is a consequence of the lack of such reinforcements. The interaction of impaired instrumental be-

havior and a low rate of positive reinforcements tends to perpetuate low self-esteem and dysphoria. Thus, the depressive individual is less able to influence others to receive positive attention from them.

The excessive dependency on others described in psychoanalytic theory, the negative attitudes and learned helplessness of cognitive theory, and the deficient instrumental behaviors described in behavior theory are all consistent with the profile emerging from this study. That is, the depressive is introverted, lacking in self-confidence, unassertive, dependent, pessimistic, and perceives himself or herself as inadequate.

In order to examine the utility of these different approaches, one must determine what causes the changes noted in depression. The psychoanalytic approach puts primary emphasis on the satisfaction of instinctual drives and postulates specifically that abnormalities exist in the depressive person's drive system. The cognitive theorists propose that the abnormal cognitive set derived from early experience is the fundamental problem and that affective changes are secondary. Obviously, the behavioral approach considers behavioral deficits as the source of the affective and cognitive changes.

There are fewer theories about mania and bipolar illness, but one that is quite consistent with the reported results is that proposed in the mid-1950s by a group of Washington psychoanalysts, including Mabel Blake Cohen, Robert Cohen, and Frieda Fromm-Reichmann (22). In essence, they proposed that various childhood experiences led to an adult character in manic individuals that was extraverted and outward-directed, in addition to well adjusted. However, close scrutiny revealed a few extremely dependent relationships, in which there was a demand for love without reciprocity that stemmed from a fear of abandonment due to internal emptiness.

Our results are consistent with this hypothesis, but our methods are insufficient to test the full extent of this theory. Several studies (23–26) done in the general time frame of the seminar did tend to support these findings. More recently, Perris (27) found that bipolar depressive persons had more normal personality profiles than unipolar depressive individuals.

We have reported here on the personality characteristics of manic and major depressive patients without further subtyping diagnoses. With the depressive disorders in particular, there is a large amount of genetic, clinical, and biochemical evidence that depressive disorders are heterogeneous. Therefore, it would be expected that the personality profiles of various depressive disorders would differ. Documentation of such differences would provide further evidence for the validity of the heterogeneity theory of affective disorders. Insufficient cell sizes have prevented definitive analysis of possible differences among depressive subtypes, but future studies are planned with cell sizes sufficiently large to allow such analyses.

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EDITORIALS

The APA Model Law on Confidentiality

THE MODEL LAW ON CONFIDENTIALITY, prepared jointly by the APA Task Force on Confidentiality of Children's and Adolescents' Clinical Records and the APA Committee on Confidentiality and published elsewhere in this issue, is designed to serve as a prototype for potential enactment in the individual states. The model is broadly drawn; it applies to all medical, psychiatric, and social service records. Although some provisions may be considered controversial, each jurisdiction can decide for itself which are desirable and/or enactable given the local legislative and professional ambience.

The issues of privacy, confidentiality, and the privilege of communications between physician and patient are in the public eye because of the impact of Watergate, the Ellsberg case, the report of the Privacy Protection Study Commission, the proliferation of linked data banks, and the encroachments on privacy by insurance carriers. Several independent groups have fashioned model laws on confidentiality. We believe the APA Model Law is at present most sophisticated from the point of view of our profession.

A law on confidentiality has a much broader reach than does a law on privilege; the latter applies only to doctor-patient privilege in judicial, administrative, and legislative proceedings. Also, most privilege laws have so many exceptions as to be of limited value. A law on confidentiality, on the other hand, must consider the constitutional right to privacy, professional codes of ethics, confidentiality of therapist-patient communications, and rules and regulations regarding hospital records and other health care and social service information. The responsibilities of computer-bank technicians, researchers, insurance companies, and employers must be articulated to protect the privacy of the individual and yet recognize the valid requirements of society vis-à-vis scientific advance and business technology.

The APA Model Law on Confidentiality is a highly technical document and will require expert legal counsel to "translate" some of its provisions. For example, Section 2 on "Definitions" provides the basis for confidentiality to a degree that becomes apparent only as one studies carefully the remainder of the law. Section 3 provides for informed consent in such a way that blanket consent forms cannot be used by insurance carriers; also, patients may revoke previous consent, and unauthorized redisclosures are prohibited. The insurance industry objects to these provisions on the basis of cost, but the public increasingly demands such protection. Several reviewers of the Model Law have objected to the 12-year age of consent, believing this to be too low; others point out that age 7 is stipulated in federal regulations concerning human experimentation. Again, each jurisdiction can decide the specific age of consent according to a local consensus.

Section 4, "Disclosures Without Authorization," is a particularly subtle section providing confidentiality guidelines in situations of training, audit, accreditation, billing, medical emergency, dangerousness, and litigation. Much case law and practical experience has been encompassed in drawing an optimal line between the needs for confidentiality and the legitimate needs of a complex society for information. Paragraph 4 (c) allows breaches of confidentiality for reporting child abuse and also to protect a patient or an intended victim from a dangerous act at

the therapist's sole discretion. This is much different from the "duty to warn" interpreted to have been mandated by the *Tarasoff* decision.

Paragraph 4 (e), "Patient/Client—Litigant Exception," protects communications between a therapist and patient in litigation. Provision is made for testimony by an examining clinician rather than the therapist unless there are unusual circumstances; similarly, the content of the therapist-patient communications is protected unless a compelling need for disclosure is proven. In contrast to most current laws, the balance is changed toward protecting patient-therapist communication. Provision is also made to allow a clinician to defend himself in case of a malpractice action. Similarly, disclosures from a court-ordered psychiatric examination are limited to the patient's clinical condition, thus precluding using the professional examination for a police function.

Of particular current relevance is Section 7, "Prescriptions," which provides that prescriptions constitute confidential information and are thus subject to the Act, but that state and federal drug regulations and laws must be obeyed.

Provisos for confidentiality in a research context have been resisted by epidemiologists and statisticians. The Model Law draws the line at removing patient-identifiable confidential material from the facility providing service. Most statistical and epidemiological research can be carried out through nonidentifiable data; research access to on-site identifiable data must be pre-reviewed by an appropriate research review committee.

Penalties for unauthorized disclosures (Sections 10 and 11) are intended to underline the responsibility of professional and clerical personnel to recognize their responsibilities to maintain the confidentiality of patient records. There is some controversy over whether punitive as well as civil penalties are in order. The APA committees believe that punitive damages for willful or grossly negligent violations of confidentiality are reasonable, but, again, local jurisdictions can modify these stipulations according to their own judgment. Hospital administrators will view provisions for damages with apprehension, but in our information-hungry society the balance of forces must emphasize the importance of privacy, and all personnel must be educated as to their responsibilities to protect the interests and privacy of the patient. The Denver State's Attorney demonstrated that the Factual Services Bureau fraudulently obtained sensitive medical information from hospital and private office charts by impersonating medical personnel. Punitive penalties would help prevent such criminal activity.

Another innovative and perhaps controversial section (Section 12) deals with access by patients to their clinical records. Current federal and state legislation as well as case law provides for access by consumers to data from which decisions modifying their lives may be made by others, such as credit agencies. However, when a physician determines that disclosure of clinical data would be detrimental to the patient, a hierarchy of procedures is established by which a patient may contest that determination. A "clinical mediator" clinician may be appointed by the patient to review the material; if he agrees with the original clinician that the information should not be disclosed, the persistent patient may go to court for a hearing and, if necessary, an *in camera* review of the data.

Another precaution regarding such disclosures is provided in Section 14, "Personal Notes." The concept of a work record of an attorney provides that a lawyer's personal notes regarding a client's case are not "discoverable" in the interest of establishing a circumstance under which a citizen may be free to disclose his troubles to a lawyer in his democratic self-interest. This concept is extended to the work record of a clinician, so that sensitive third-party information can be protected, as well as his own speculations, impressions, and research data. Thus a chilling factor to his research and clinical interests is avoided. This is a pioneer concept and will have to stand the test of time.

Section 15 provides for confidentiality of insurance records. There have been many instances of breach of privacy by employers and/or clerical personnel. Provision is made for bypassing the employer's personnel office and direct communication with the insurance companies. Section 16 prohibits unauthorized redisclosure by the insurance companies to other agencies such as the Medical Information Bureau. The insurance industry may well resist the enactment of these two

sections and in most states may prevail, as they did this past year in Illinois when a Confidentiality Act containing most of the provisions of the APA Model Law was enacted. It is hoped that implementation of the recommendations of the Privacy Protection Study Commission by Congress will neutralize this aspect of insurance applications and claims.

Sections 17 and 18 formulate guidelines for state, county, local, and federal agencies in performing audits, inspections, and investigations. There have been many instances in which auditors and police agencies have demanded access to sensitive records, thereby destroying the effectiveness of psychiatric and drug-abuse clinics. The principle has been established that funding per se does not entitle complete access to identifiable clinical records. Government agents must become educated to their responsibility to protect the privacy of citizens, including those who receive agency largesse.

Similar problems exist with data processing. Linkages of various data banks allow the profiling of private citizens to a surprising extent through use of Social Security or driver's license numbers. Paragraph 17 (c) has been designed to help counter such unauthorized invasions of privacy. At present most government agencies will resist such attempts at regulation, but, again, it is a matter of persistent education of the consumer, the profession, and the bureau.

In some states the APA Model Law will represent such a radical advance that it will not be enactable at present. As already noted, in Illinois most of the model has been enacted, even though it was necessary to omit the section dealing with insurance matters because of the effectiveness of the insurance industry lobby in order to save the remainder of the bill. Similarly, it was necessary to provide access to the state's attorneys for investigative purposes, but with the proviso that unauthorized redisclosure is prohibited. It is also of interest that similar legislation was recently introduced into Congress as S 3450 under the joint sponsorship of Senators Javits, Muskie, and Ribicoff.

Because of the public's interest in preserving privacy, it has been predicted that in ten years all states will have privacy laws. Psychiatrists, because they are particularly sensitive to confidentiality as a prerequisite to the effectiveness of their work, are among the leaders in effectuating such legislation. The APA Model Law incorporates our expertise. Most lawyers will be opposed to many of its provisions. Just as we require confidentiality for our work, lawyers require complete discovery of information. Our professional interests are antithetical but are subject to education, negotiation, legislation, and litigation. It is in the interest of society, our patients, and our profession that confidentiality laws be enacted in each state. The Model Law can serve as a prototype lending itself to local modifications. The APA Committee is available for consultation.

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A Note of Appreciation

"WHEN IT COMES to the need for praise," wrote a recent reviewer in the *Times Literary Supplement,* "authors. . .like it applied not with a trowel but a crane." The Editor's own introspections long ago convinced him of the truth of that dictum, and it is perhaps one of the more painful aspects of his job that, as the ultimate arbiter of manuscripts submitted to the *Journal,* he must disappoint so many aspiring writers. The burden of his task is, however, immeasurably lightened not only by the collective wisdom of his Managing Editor, his Associate Editors, and his Editorial Staff, but by the help of the multitude of expert reviewers throughout the country who selflessly give of their knowledge and time. The latter, indeed, work silently behind the scenes, demanding no praise, requiring no recognition, asking no recompense other than the satisfaction of helping to maintain the *Journal's* scientific standards. "Mute inglorious Miltons" they would remain were it not possible once a year for the Editor and his staff to recognize them by name in these pages. And so, as the new year dawns, we give heartfelt thanks for their invaluable contributions.

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1978 has seen the editorship change hands, and the Editor would like to express his deep appreciation to the Editor Emeritus, the Managing Editor, the Associate Editors, and the *Journal* staff for making the transition easy and pleasurable. The entire Editorial Staff, furthermore, must acknowledge its considerable indebtedness to our Medical Director, Dr. Melvin Sabshin, for his unfailing help and understanding. Our sincere thanks for their continued support and interest go also to Drs. Lee Gurel, Donald W. Hammersley, Carolyn Robinowitz, Jeanne Spurlock, Jack White, and Henry H. Work; to Mses. Nancy Frey and Jean Jones; and to Mssrs. Herbert M. Gant, Raymond M. Glasscote, Charles B. Kurta, and Robert L. Robinson.

Finally, we extend our warmest wishes to all our colleagues for a joyous, successful, and peaceful 1979.

J.C.N.

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BRIEF COMMUNICATIONS

Factors Related to Tardive Dyskinesia

BY GUY CHOUINARD, M.D., M.SC.(PHARMACOL.), LAWRENCE ANNABLE, DIP.STAT., ANDRÉE ROSS-CHOUINARD, M.D., AND JOANNIS N. NESTOROS, M.D.

The authors found a 31% incidence of tardive dyskinesia among 261 schizophrenic outpatients treated with neuroleptics. Multiple linear logistic regression analysis revealed a higher incidence of tardive dyskinesia among elderly patients, those with longer records of hospitalization, those for whom neuroleptic medication had little therapeutic effect, and those treated with fluphenazine. Patients manifesting tardive dyskinesia tended to have fewer parkinsonian symptoms than those without the disorder, especially when tremors and akathisia were excluded from consideration. Multiple linear regression analysis indicated that brain-damaged patients and male patients were more susceptible to severe forms of the disorder, even though these factors were not implicated in its initial appearance.

TARDIVE DYSKINESIA is a hyperkinetic syndrome of extrapyramidal nature associated with neuroleptic drug use (1–3). It is characterized by involuntary repetitive and purposeless movements varying in localization and form but commonly involving the mouth, lips, tongue, and jaw (bucco-linguo-masticatory dyskinesia) and at times by choreoathetoid movements of the neck, trunk, or limbs. These movements are usually intensified by emotional tension or by voluntary movements of other muscle groups. They are reduced in intensity by sleep or drowsiness. Neuroleptic drugs that are causing the syndrome can temporarily reduce the dyskinesia, and the abnormal movements may become more severe or appear for the first time after drug withdrawal or dose reduction (4). Antiparkinsonian drugs fail to reduce the dyskinetic movements and can aggravate or uncover the tardive dyskinesia (5). In many patients the syndrome is irreversible; in others it can sometimes be reversed by discontinuing the neuroleptics (6). When the condition is life-threatening, as in rare cases of dyskinesias of the esophagus or diaphragm, piperazine-like neuroleptics (fluphenazine or haloperidol) can be given and anticholinergic drugs (antiparkinsonian or others) avoided.

Recent studies have shown that tardive dyskinesia occurs with alarming frequency among patients treated with neuroleptics (7, 8). Fann and associates (7) found that 36% of a sample of 204 patients treated with neuroleptics manifested the syndrome. At the present time, there is no treatment of tardive dyskinesia that is effective over a long period of time; most efforts are directed toward its prevention.

The Allan Memorial Institute maintains a special follow-up clinic for the long-term maintenance treatment of schizophrenic outpatients. The present study was designed to evaluate the incidence and severity of tardive dyskinesia among 261 patients from this population and to determine the relationship of the syndrome to various other factors. Whereas most previous surveys of tardive dyskinesia were conducted in long-term wards of mental hospitals, this study dealt with a population of schizophrenic outpatients.

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METHOD

Patients are accepted in the special follow-up clinic after the primary hospital diagnosis of schizophrenia is confirmed by the psychiatrist in charge of the clinic. The diagnostic criteria used are similar to those of the National Institute of Mental Health–Psychopharmacology Service Center Collaborative Study (9). Schizophrenic patients who are not likely to need long-term pharmacotherapy are not accepted in the clinic. During the two years before this study pharmacotherapy was under strict control, and the following principles were applied: the minimum therapeutic dose was given; a single neuroleptic was prescribed whenever possible; drugs were administered in a single daily dose or twice-daily regimen; procyclidine (Kemadrin) was the only antiparkinsonian drug used; attempts were made to withdraw the antiparkinsonian after three months of treatment; fluphenazine enanthate, an injectable long-acting drug, was given to patients who were resistant to other neuroleptics, those who could not be relied upon to take medication, and those with frequent relapses; fluphenazine hydrochloride was the only piperazine-type drug given orally; polypharmacy was avoided and antidepressant drugs, minor tranquilizers, and hypnotics were not prescribed; and, finally, medication was reviewed each time the patient came to the clinic.

The patients were examined by a neurologist who had not seen them before in random order on the day they came for their regular visit to the clinic. All patients were examined except for a small percentage (less than 5%) who repeatedly failed to keep appointments. The neurologist completed a structured medical questionnaire,[1] carried out a complete neurological examination, and rated each patient for the presence and severity of tardive dyskinesia and other extrapyramidal symptoms. The presence of tardive dyskinesias was assessed according to a standard procedure that included the following routine neurological tests: 1) the patient's spontaneous behavior was observed while seated, standing, or walking, 2) since abnormal movements are increased by voluntary movements of other muscle groups, the oral-facial region was observed while the patient carried out the pronation-supination tests of both hands as fast as possible and performed rapid alternate movements of both wrists and the finger-nose-finger test, 3) the patient was asked to walk without shoes so that any choreoathetoid movements of the limbs could be noted, and 4) the patient was asked to copy a spiral with both hands and to sign his or her name (since the test is performed under emotional tension, this procedure may activate dyskinetic movements and thus sometimes uncover covert dyskinesias). In doubtful cases, the patient was asked to open his or her mouth while performing the pronation-supination and alternate movement tests so that the tongue could be observed. Questionable cases of tardive dyskinesia were not considered to be positive.

Dyskinetic movements were rated according to frequency and amplitude on a 4-point scale (0–3) as follows: 0=absent, 1=mild but clearly present, 2=moderate, and 3=severe. Abnormal movements in the lingual, masticatory, and facial-labial areas and the trunk and extremities were rated separately.

Other extrapyramidal symptoms were also rated on a modified version of a specially designed extrapyramidal rating scale (10). The following items were evaluated: expressive automatic movements (facial mask and speech), bradykinesia, rigidity of limbs, gait and posture, tremor (of limbs, head, chin, and tongue), akathisia, increased salivation, and dystonia. Both spontaneous tremors and those manifested when the patient extended both arms forward with palms down and eyes closed or performed the spiral test and handwriting were evaluated. Each item was rated on a 6-point scale ranging from normal to extremely severe.

In the same visit the patient was seen by one of three psychiatrists who rated the therapeutic response to treatment on a 5-point scale (11) and overall severity of illness on a 7-point scale (11). The neurologist and psychiatrists did not have access to each others' findings.

RESULTS

The study population consisted of 261 schizophrenic outpatients, 138 male and 123 female, ranging in age from 19 to 67 years. Table 1 shows the mean and standard deviation for the age, length of neuroleptic treatment, neuroleptic dose (converted to chlorpromazine units [12]), clinical global impression (CGI) of overall severity of illness, and CGI therapeutic effect for male and female patients separately and for both sexes combined. Table 2 shows the percentages of patients with tardive dyskinesia and parkinsonian symptoms, those being treated with antiparkinsonian medication and fluphenazine, and those with a history of ECT treatment, insulin treatment, brain damage, alcoholism, antidepressant treatment, and hospitalization.

Three kinds of statistical analysis were performed on the data. In each analysis the variable for tardive dyskinesia was regressed against the following variables: age, number of years treated with neuroleptics, length of previous psychiatric hospitalizations, current dosage of neuroleptics (converted to chlorpromazine units), current fluphenazine treatment, total score for parkinsonian symptoms, antiparkinsonian dosage, number of ECT treatments, number of insulin treatments, presence of brain damage, alcoholism, history of antidepressant treatment, CGI overall severity of illness, and CGI therapeutic effect.

[1]The medical questionnaire covered the following items: 1) family history of mental disease, epilepsy, abnormal movements or tics, or any neurological disease, 2) history of previous admission for or investigation of neurological disorder, brain surgery, head trauma (with or without skull fracture), meningitis, or coma, and history or presence of abnormal movements, tics, epilepsy, or venereal disease, 3) abnormal laboratory results (VDRL or EEG), 4) history of loss of consciousness, headache, visual trouble (decreased vision), loss of balance or difficulty walking, or difficulty swallowing or talking, and 5) history of alcoholism or drug abuse (LSD, barbiturates, etc.).

TABLE 1
Characteristics of 138 Male Patients and 123 Female Patients in Study Population

Characteristic	Males Mean	Males SD	Females Mean	Females SD	Total Mean	Total SD
Age (years)	38.3	11.0	44.2	11.1	41.1	11.4
Years of neuroleptic treatment	8.8	5.7	10.7	5.8	9.7	5.8
Neuroleptic dose (chlorpromazine units)*	425.0		300.0		400.0	
CGI (in general)	4.6	0.9	4.5	0.9	4.5	0.9
CGI (therapeutic effect)	1.6	0.9	1.7	0.9	1.6	0.9

*Median dose given because the distribution of neuroleptic dosages was skewed (range=0–5500 chlorpromazine units/day). Neuroleptic medications were converted to chlorpromazine units according to the equivalency given by Davis and Cole (12). Fluphenazine enanthate was converted to chlorpromazine units on the basis of an equivalency of a dose of 25 mg I.M. every 2 weeks to 300 mg chlorpromazine daily.

TABLE 2
History and Presence of Tardive Dyskinesia or Parkinsonian Symptoms in 138 Male Patients and 123 Female Patients in Study Population

Factor	Males (%)	Females (%)	Total (%)
Tardive dyskinesia	29.0	32.5	30.7
Parkinsonian symptoms	71.0	59.3	65.5
Antiparkinsonian treatment	60.9	56.9	59.0
Fluphenazine treatment	47.8	30.9	39.9
ECT treatment	47.8	59.3	53.3
Insulin treatment	4.5	8.8	6.5
Brain damage	24.6	17.1	21.1
History of alcoholism	17.4	3.3	10.7
History of antidepressant treatment	23.2	31.7	27.2
Previously hospitalized	86.2	90.2	88.1

TABLE 3
Variables Significantly Related to Presence of Tardive Dyskinesia According to Stepwise Logistic Regression Analysis

Variable	Significance
Age	p<.001
Days in hospital	p=.03
CGI therapeutic effect	p=.05
Fluphenazine treatment	p=.03
Parkinsonism total score (negatively related)	p=.06*

*p=.03 when score for tremors is excluded from parkinsonism total score; p=.02 when scores for tremors and akathisia are excluded from parkinsonism total score.

Variables Predicting Incidence of Tardive Dyskinesia

Patients were coded 1 or 0 according to whether tardive dyskinesia was present or not; a regression analysis using the linear logistic model (13) related other variables to the probability of the patient's manifesting tardive dyskinesia. This analysis was carried out on all 261 patients by a computerized stepwise inclusion procedure (14), which first determines the single most important characteristic in predicting the incidence of tardive dyskinesia, then the second most important variable given that the first has been included in the equation, and so on. Table 3 shows the five variables that, when entered sequentially, made statistically significant ($p < .05$) contributions to the regression relationship. The variable most related to the incidence of tardive dyskinesia was age, which was highly significant ($p<.001$). Then followed length of time spent in a psychiatric hospital, score for CGI therapeutic effect (a high score indicates little improvement, i.e., there tended to be a greater incidence of tardive dyskinesia among patients with little therapeutic improvement), fluphenazine treatment, and, finally, the total score for parkinsonian symptoms, which was related inversely to tardive dyskinesia (i.e., patients with fewer parkinsonian symptoms tended to have a higher incidence of tardive dyskinesia). When the score for tremors or the scores for both tremors and akathisia are excluded from the total score for parkinsonian symptoms, the inverse relationship between tardive dyskinesia and parkinsonian symptoms becomes more pronounced (see table 3). A test of goodness of fit of the model showed the fit to be satisfactory ($\chi^2=1.6$, df=4, p=.8).

TABLE 4
Variables Contributing Significantly to Multiple Regression Relationship with Total Score for Tardive Dyskinesia,* As Entered by Stepwise Inclusion

Variable	Significance	Multiple Correlation Coefficient	Partial Correlation Coefficient
All patients included (N=261)			
Age	p<.001	.29	.29
CGI therapeutic effect	p=.003	.34	.19
Parkinsonism total score (negatively related)	p=.026	.37	−.14
Fluphenazine treatment	p=.013	.39	.15
Days in hospital	p=.050	.41	.12
Sex (male)	p=.049	.43	.12
Only tardive dyskinesia patients (N=80)			
Brain damage	p=.003	.33	.33
CGI therapeutic effect	p=.045	.39	.23
Age	p=.089	.43	.19
Parkinsonism total score (negatively related)	p=.079	.47	−.20
Sex (male)	p=.077	.50	.20

*Scores for tremors and akathisia are excluded from total score for parkinsonian symptoms.

Variables Predicting Severity of Tardive Dyskinesia

This analysis related the severity of tardive dyskinesia, as measured by the total score, to the other variables by the usual multiple linear regression model. A first analysis was carried out on all 261 patients by a computer program using a stepwise inclusion procedure (15). Table 4 shows the six variables that, when entered sequentially, were significantly related to the total score for tardive dyskinesia. The same five variables found to be related to the incidence of tardive dyskinesia were also related to the severity of the disorder, although there is some change in their relative importance. Again, the inverse relationship between tardive dyskinesia and parkinsonian symptoms was more pronounced when the score for tremors and aka-

thisia were excluded from the parkinsonism total score. In addition, the sex of the patient enters the relation, with male patients tending to show more severe forms of the condition than female patients. The six variables included in the equation account for 18.5% of the total variation in the scores for tardive dyskinesia among the 261 patients.

Finally, we repeated this analysis including only those 80 patients who had tardive dyskinesia (table 4). This analysis revealed that among the patients with tardive dyskinesia, those with previous brain damage tended to have more severe forms of the disorder.

DISCUSSION

Recent surveys show a higher incidence of tardive dyskinesia than was first reported. In this study, tardive dyskinesia was found to be present in 31% of a population of 261 schizophrenic outpatients treated by neuroleptics; this percentage is similar to that reported by Fann and associates (7). Age was clearly the most important contributory factor; older patients were more susceptible to tardive dyskinesia. It has also been our clinical experience that the older the patient is when first started on neuroleptics, the sooner he or she is likely to develop the syndrome. This would indicate a need for caution when prescribing neuroleptics to the elderly patient. A 1977 study (16) suggested biochemical changes in the striatum with aging, and it might be hypothesized that these changes could favor the appearance of tardive dyskinesia.

Among patients of the same age, those for whom neuroleptic treatment was producing little therapeutic effect were more likely to present severe forms of tardive dyskinesia. When linked with the finding on age, this suggests that in some cases it may be beneficial to withdraw neuroleptics from older patients who are responding poorly to treatment.

Crane (2, 4) has reported that severe tardive dyskinesia is seldom observed in patients with severe parkinsonism, and vice-versa. Our findings support this, especially when tremors and akathisia are not included among the other parkinsonian symptoms. These two symptoms were excluded because our clinical experience has shown that a patient with tremors or akathisia may manifest tardive dyskinesia, whereas a patient with akinesia or rigidity seldom does. There tended to be a high incidence of tardive dyskinesia among patients being treated with fluphenazine (oral or enanthate). Although fluphenazine was the only piperazine drug given to these patients, one might expect this finding would also apply to the other piperazine-like drugs, which are responsible for a higher incidence of parkinsonian symptoms. It is possible that the hyperkinetic parkinsonian symptoms of tremor and akathisia are precursors of hyperkinetic symptoms of dyskinesia. Thus, these symptoms could be expected to coexist with dyskinetic symptoms in patients shifting from one syndrome to the other. In contrast, the hypokinetic parkinsonian symptoms of rigidity and akinesia could reduce or cover the expression of dyskinesia.

Tardive dyskinesia was more prevalent among patients who had spent longer periods of time in psychiatric hospitals (independent of their age). Since this variable appeared to be more important than the length of neuroleptic treatment, it may reflect the effect of regular high-dose neuroleptic administration in hospitals. Similarly, the finding that patients responding poorly to neuroleptics had a higher incidence of tardive dyskinesia might be related to past exposure to high neuroleptic doses. This would indicate that use of neuroleptics at higher than normal dosage should be limited to the most severe cases. However, the dose of neuroleptic currently received by the patient was not found to enter significantly into the regression relationship. This is not unexpected, since high doses of neuroleptics are known to mask tardive dyskinesia.

Previous brain damage tended to be present in the more severe cases of tardive dyskinesia but was not found to be implicated in the appearance of the disorder. The overall incidence of tardive dyskinesia among brain-damaged patients was not higher than among patients with no brain damage. Since the most severe forms of dyskinesia are incapacitating, a history of brain damage would appear to be a relative contraindication to neuroleptic treatment. In the same way, male patients also appeared to be more susceptible to severe forms of the disorder. Because men are often less responsive to neuroleptics than women, a possible explanation of their increased susceptibility to severe tardive dyskinesia is the use of higher neuroleptic dosage. Other factors, such as ECT, insulin treatment, antiparkinsonian medication at the time of examination (which in the clinic is always adjusted so as not to aggravate the tardive dyskinesia), and history of tricyclic antidepressant treatment were not found to be related to tardive dyskinesia in this population of schizophrenic outpatients.

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ADDENDUM

Most patients in this study treated with fluphenazine were receiving fluphenazine enanthate I.M. and were rated for tardive dyskinesia when they came to the clinic for their biweekly injections. Consequently, they were evaluated at a time when their fluphenazine blood levels were at their lowest, and dyskinetic movements that are normally masked at higher fluphenazine levels may thus have been exposed. This may have been responsible for the higher incidence of tardive dyskinesia we found among patients treated with fluphenzine.

Hypnotic Treatment of Smoking: The Single-Treatment Method Revisited

BY BERNARD BERKOWITZ, M.D., ANITA ROSS-TOWNSEND, PH.D., AND ROBERT KOHBERGER, PH.D.

The authors replicated Spiegel's single-treatment method by treating 40 self-referred patients in a one-hour hypnosis session for smoking. Their results, a 6-month total abstinence rate of about 25%, were comparable with Spiegel's experience. They recommend future studies that include nontreatment control groups to validate the effects of the hypnotic treatment method of smoking.

JOHNSTON AND DONOGHUE (1), in a 1971 review of the literature on hypnosis in the treatment of smoking, noted the difficulties involved in replicating results: "The hypnosis-smoking literature . . . is primarily anecdotal and rarely in the form of a controlled investigation. . . . Most articles claim many successes and, although they admit to some failures, it is never entirely clear who fails, and when, and why failures occur" (1). They concluded that "Most authors claim success, but their procedures cannot be reproduced." A review of reports since that time shows this still to be true, with the exception of the work of Spiegel (2, 3). His reports of a single-treatment method not only describe in detail the method used but also analyze the results of a 6-month follow-up with 615 patients. Our paper is an effort to address the deficiencies noted by Johnston and Donoghue by replicating Spiegel's method.

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TREATMENT

Spiegel has stated that his earlier, open-ended approach to treatment encouraged the patient to delay confrontation with the decision to terminate smoking (3). He thought that if the procedure were going to work, it would do so right away, so he moved to the single-treatment approach. Briefly, Spiegel's single-treatment method consists of taking a brief clinical and smoking history, the application of his Hypnotic Induction Profile, and the induction of trance and confrontation with three basic points: 1) for your body smoking is a poison; 2) you need your body to live; and 3) you owe your body this respect and protection. Three points are taught to the patient so that he can use them as a self-hypnotic exercise 10 times daily. Positively oriented suggestions are given by the therapist, who emphasizes the individual's own responsibility for his success. The method is described in detail elsewhere (2, 3).

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The treatment was applied in a private psychiatric setting to 40 consecutive patients who came voluntarily after calling for an appointment. Patients were generally referred by word of mouth, although a few were referred by their physicians. There were 20 men and 20 women ranging in age from 22 to 63 years of age (mean=39) who had been smoking for from 5 to 40 years (mean=23). We followed Spiegel's method as faithfully as possible. One of us (B.B.) had seen Spiegel demonstrate the method, had been treated for smoking by this method by Spiegel, and had read Spiegel's procedural descriptions. This investigator did all of the actual treatment.

Although the patients were generally seen only once, we encouraged patients to return for subsequent visits without charge should they fail (a departure from Spiegel's method). Seven patients took advantage of this offer, but none succeeded in terminating smoking.

A research assistant unknown to the patients interviewed them by telephone an average of 64.5 weeks after treatment (range=13 to 104 weeks). This included at least 38 weeks for successes and 13 weeks for failures. We obtained information on outcome and the smoking-free interval (time of complete abstinence) for all patients. Generally, patients could recall dates of failure with good precision, but we calculated the midpoint of the interval based on 4 weeks per month for those patients who gave vague answers. Successful outcome was defined as total abstinence from smoking from the point of treatment until follow-up. Failure was defined as any resumption in smoking from the point of treatment to the point of follow-up; decrease in base-rate smoking short of abstinence was counted as failure.

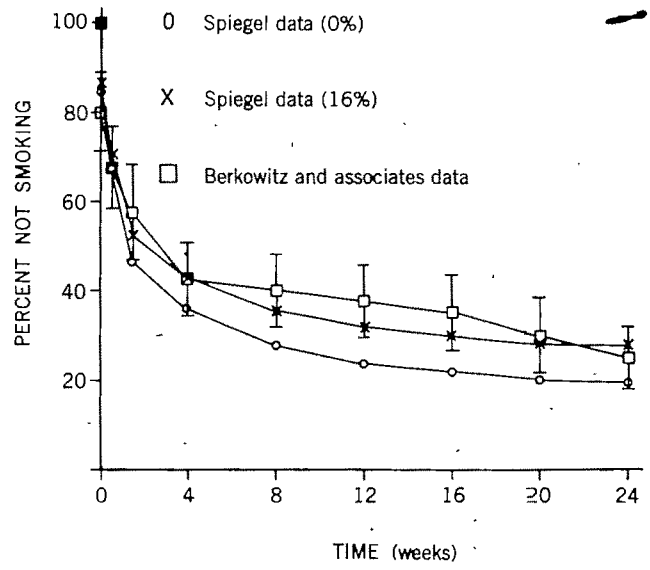
DATA ANALYSIS

The percentage of patients not smoking at various times and its associated standard error was estimated by the method of Cutler and Ederer (4). In order to compare our data with Spiegel's, calculations were made only to 24 weeks. Two methods were used to estimate the number of follow-up nonrespondents in Spiegel's data who are not smoking. Spiegel's approach was to assume that all nonrespondents have resumed smoking. This will only give a lower bound to the percentage not smoking because it is likely that some of the nonrespondents are not smoking. Kanzler and associates (5) in a follow-up study of a smoking treatment found that 75% of those responding to a mail questionnaire were not smoking while 27% of nonrespondents were not smoking when they were later contacted by telephone. This suggests that the nonsmoking percentage of nonrespondents to a mail questionnaire is about 1/3 of the nonsmoking percentage of respondents. Applying this estimate to Spiegel's data yields an estimate of 16% for the nonrespondents. Standard errors were not calculated for Spiegel's data because estimates were used.

TABLE 1
Comparison of Results of Treatment Outcomes

	Spiegel (N=615)		Berkowitz and Associates (N=40)	
Subjects' Response	N	%	N	%
Stopped smoking (6 months or longer)	121	19.7	10	25.0
Resumed smoking (failures)				
Immediately	29	19.3	8	26.7
Less than 1 week	32	21.3	5	16.7
1 to 2 weeks	39	26.0	4	13.3
2 to 6 weeks	19	12.7	6	20.0
6 to 10 weeks	16	10.7	1	3.3
10 to 14 weeks	8	5.3	1	3.3
14 to 18 weeks	3	2.0	1	3.3
18 to 22 weeks	3	2.0	2	6 7
22 to 26 weeks	1	.7	2	6.7
No response to follow-up	344	55.9	0	0
Failures plus nonrespondents	494	80.3	30	75.0

FIGURE 1
Success Curves for Berkowitz and Associates Data and Spiegel's Data



RESULTS

Table 1 presents Spiegel's data (2) and our data. The shapes of the success curves (figure 1) for our data and Spiegel's data closely resemble each other. Spiegel's data with the assumption that 16% of the nonrespondents were not smoking fall within 1 standard deviation of every point of the sample curve. Spiegel's data with the assumption that 0% of the nonrespondents were not smoking fall within 2 standard deviations of the sample data, and the data for up to 8 weeks and at 24 weeks fall within 1 standard deviation. The 6-month success rate for the sample data, Spiegel (16%), and Spiegel (0%) are 25%, 28.2%, and 19.7%, respectively. These are not statistically different.

DISCUSSION

Various claims have been made for different treatment methods. Our results, although modest at the 6-month interval, reproduce Spiegel's experience and do so over time; as such, they may represent a stable basis for methodologic improvements.

Future studies that include nontreatment control groups are needed to validate the effects of the hypnotic treatment method. Spiegel's single-treatment method can be replicated and meets with a 6-month success rate of complete abstinence of about 25%.

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MMPI Diagnosis of Black Psychiatric Patients

BY VICTOR R. ADEBIMPE, M.D., JOSEPH GIGANDET, M.A., AND ELLIOT HARRIS

Black-white differences in MMPI norms may lead to significant misclassification of black psychiatric patients, which can compound diagnostic errors from other sources. The authors suggest that clinicians who use MMPI results in evaluations of black patients interpret test reports carefully, keeping in mind the possibility of spuriously elevated scores and paying careful attention to areas of potential "false positives."

CONTROVERSIES GENERATED by intelligence tests have led to the realization that psychological testing is not always free of cultural bias (1). However, few psychiatrists are fully aware of the possibility that diagnostic errors can result from the use of other types of tests with black patients. The purpose of this article is to bring to the attention of psychiatrists evidence that the Minnesota Multiphasic Personality Inventory (MMPI) may not be entirely suitable for American blacks (2–4) and to suggest ways of dealing with this problem when clinical decisions must be made.

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MMPI COMPARISONS: NORMAL AND PSYCHIATRIC POPULATIONS

In order to determine the MMPI scales in which normal blacks have been reported to score different baselines from their white counterparts, we reviewed the literature on studies comparing high school students, patients in a prenatal clinic, and nonpatient adults (5–11). As shown in table 1, blacks scored significantly higher on the Validity (F), Schizophrenia (Sc), and Hypomania (Ma) scales. The literature on psychiatric patients was also reviewed (12–15), and table 2 shows that the trends seen in table 1 are also found in this population.

DISCUSSION

Differences in MMPI norms for various ethnic, cultural, and national groups are well-documented (16). In most cases, however, profile patterns that are unique to the group are not associated with erroneous psychiatric diagnoses. American blacks appear to be an exception. For example, the majority of a sample of normal rural blacks were labeled schizophrenic by this instrument (17). This pattern is also found in psychiatric populations. Gynther (2) reported a significant difference in the rates of misdiagnosis, with black patients being inaccurately diagnosed more often than white patients. Sixty-six percent of his black psychotic patients were incorrectly classified compared with

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TABLE 1
Comparisons of MMPI Scale Scores for Blacks and Whites: Normal Populations*

Study	Sample	Scale ?	L	F	K	Hs	D	Hy	Pd	Mf	Pa	Pt	Sc	Ma	Si
Ball (5)	9th grade students in Kentucky, blacks low status, whites high status														
	Females			B	W			W					B		B
	Males					B									
Hokanson and Calden (6)	Male tuberculosis patients, similar age, occupation, status		B	B					W	B			B	B	
McDonald and Gynther (7)**	High school seniors in segregated schools, low-SES blacks		B	B		B	B							B	
McDonald and Gynther (8)	High school seniors controlled for race, sex, SES		B	B			B			B			B	B	
Harrison and Kass (9)***	Boston Hospital prenatal clinic, predominantly lower class	B		B		B							B	B	
Baughman and Dahlstrom (10)	8th grade students in North Carolina			B			B						B		
Erdberg (11)	Low and middle class adults			B				W	B	W	B		B	B	

*"B" indicates black group mean significantly higher than white, "W" indicates white group mean significantly higher than black ($p \leq .05$ for all comparisons). SES=socioeconomic status.
**Black-white score differential markedly stronger for females.
***Female subjects only.

TABLE 2
Comparisons of MMPI Scale Scores for Blacks and Whites: Psychiatric Populations*

Study	Sample	Scale ?	L	F	K	Hs	D	Hy	Pd	Mf	Pa	Pt	Sc	Ma	Si
Miller and associates (12)	VA mental hygiene clinic, controlled for age, employment status, education		B			B				W			B**	B	
Miller and associates (13)	VA mental hygiene clinic, controlled for age, employment status, education		B			B				W			B**	B	
Costello and associates (3)	Medical center, psychiatric patients matched for age, sex, SES, duration of illness			B	W	B				B			B	B	
Costello and associates (4)***	Medical center, psychiatric patients matched for age, sex, SES, hospital status			B											
Davis and associates (14)	51 black, 54 white, diagnosed schizophrenic, blacks had fewer years of formal education			B					B				B		
Genther and Graham (15)	State hospital, Cleveland, psychiatric patients, significantly lower SES for blacks		B										B	B	

*"B" indicates black group mean significantly higher than white, "W" indicates white group mean significantly higher ($p \leq .05$ except where indicated).
**$p \leq .10$.
***Black-white score differential markedly stronger for females.

44% of the whites. These findings have been replicated and are essentially unchanged by the use of a variety of interpretive formulas (18).

A number of attempts have been made to explain the differences on the basis of social class. Evidence has been produced to show that black patients do not score differently from white patients if the groups are similar in mean age, years of education, and occupation (3, 4, 14, 19). Such studies are useful in showing the cause of the problem but are less relevant to its effects on a group of patients who are over-represented in the social classes at greatest risk. Whatever the causes of black-white differences in MMPI profiles, their most probable effect in clinical situations is significant misdiagnosis among black psychiatric patients.

In general, MMPI diagnoses correlate rather poorly with psychiatric diagnoses (20). Since the MMPI report usually supplements other data, it would seem that blatant disagreement between such reports and clinical diagnoses should lead diagnosticians to rely more on their clinical judgment when dealing with black patients. Unfortunately, clinical judgment may be just as erroneous as MMPI reports. Simon and as-

sociates (21) found a tendency to overdiagnose schizophrenia and to underdiagnose affective illness in black patients admitted to nine psychiatric hospitals in New York State. Welner and associates (22) have also reported the difficulty of accurate psychiatric diagnosis in blacks. In a study of "undiagnosed" psychiatric patients, systematic data collection and discussion of each case by two experienced clinicians still resulted in a significantly higher rate of diagnostic error in black patients.

It is likely, therefore, that uncritical acceptance of MMPI reports on blacks may result in the "confirmation" of one set of errors by another. This situation may be related to the consistently higher rates of schizophrenia reported for hospitalized blacks (23–25).

Clearly, the MMPI should be used with caution in the diagnosis of psychiatric illness in black Americans. Clinical psychologists should attempt to make interpretations that take into consideration the possibility of spuriously elevated scales. There should be a greater awareness of this problem among psychiatrists, so that reports suggesting psychopathology in "false positive" areas will be given closer scrutiny, especially when they are at variance with other clinical information.

When *DSM-III* comes into use, it may be safest to use the MMPI as little as possible for diagnostic evaluations in blacks. However, it remains to be shown that the new manual is free of the potential for diagnostic errors based on cultural differences.

Even if the MMPI is used less often in psychiatric evaluations, it will probably continue to be used for personality assessment in nonmedical settings. Therefore, further study of ways to minimize interpretive errors in blacks appears essential.

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Behavior Therapy as a Test of Psychoanalytic Theory

BY R. JULIAN HAFNER, M.D., M.PHIL.

The author describes 4 patients in whom radically new symptoms appeared after intensive behavior therapy. He suggests that fresh symptoms are most likely to develop in a small minority of patients who are unable to learn more adaptive psychological defense mechanisms during or after behavior therapy and that psychoanalytic theory may help predict the nature of such symptoms.

IN A SYSTEMATIC and comprehensive review of attempts to test psychoanalytic theory by experimentation, Eysenck and Wilson (1) found justification for Eysenck's earlier statement that "there is no evidence at all for psychoanalytic theory" (2). They added, "The fact that Freudian theory explains everything is its greatest weakness, because at the same time it predicts nothing."

An important aspect of Eysenck's sustained attack on psychoanalytic theory is his claim that fresh symptom emergence or "symptom substitution" is rarely if ever a consequence of behavioral treatments based on learning theory (3). Behavior therapists appear to unanimously agree with this (4–7).

Therapists who use or systematically observe the use of behavioral techniques while retaining an eclectic approach to psychotherapy have found otherwise (8, 9). Crisp (8) advocated behavior therapy as a means of testing psychodynamic theory but found a major practical problem—the inability of psychodynamically oriented psychiatrists to agree on their predictions of the outcome of behavior therapy in specific cases. I kept Crisp's suggestion in mind during my involvement in the behavioral treatment of 83 patients, 67 of whom suffered from agoraphobia, 11 from chronic obsessional neuroses, and 5 from specific phobias. The basic methods used in the treatment of the agoraphobics are detailed elsewhere (10), and the remaining patients were treated with the techniques of exposure in vivo and response prevention outlined by Marks (11). Of these 83 patients, the following 4 changed their behavior in an unexpected and substantial way soon after treatment.

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The author wishes to thank Dr. L.K.G. Hsu, who treated the patient described in case 4.

CASE REPORTS

Case 1. Ms. A, a widow of 50 with a 25-year-old son, described the sudden onset of her symptoms. She was in a crowded store 1 year after her husband's accidental death by drowning (some 9 years before her referral): "I was looking at cardigans when my vision started to get a bit hazy and everything started going round. I felt faint and hot, my heart pounded, I hung on to the counter and said, 'I'm not going to let myself go, I don't want to black out.' " She managed to make her way home unaided but subsequently found that similar unpleasant sensations stopped her from leaving home alone, although she could travel up to 400 yards when accompanied. Numerous and repeated medical investigations over 2 years revealed no physical illness, and she failed to respond to a range of psychiatric treatments. Throughout the 8 years of her agoraphobia she remained socially isolated, seeing very little of her elderly parents or son, whom she was unable to visit and who appeared to avoid her, mainly because of her constant complaints about her health. She relied for social contact and support on a succession of mental health workers, on whom she invariably and rapidly became extremely dependent.

She responded to 16 hours (spread over 3 weeks) of graduated real life exposure to traveling outside her home. Particular attention was paid to her fears of falling or blacking out, which she largely overcame. However, as the postural problems receded, she developed nausea, occasional vomiting, and other gastric symptoms that threatened to halt her progress. Nonetheless, after treatment she was able to travel alone on buses and trains for the first time in 8 years. The death of her cat about 4 months after treatment caused her profound distress and was associated with a marked increase in vomiting. This stopped her going out, since she found that she would almost invariably vomit within 100 yards of her home. She then embarked on a series of medical investigations for her vomiting, all of which proved negative. A year after treatment, she was more housebound than she had been before treatment, but vomiting and fear of vomiting had almost totally replaced the postural symptoms as reasons for her inability to venture outside her home alone.

Case 2. Ms. B, a 48-year-old married woman with 3 adolescent children, had suffered for 12 years from a profound fear of dogs and obsessive-compulsive symptoms related to the need to maintain excessive cleanliness of herself and her surroundings. Her mother and sister also displayed severe obsessional symptoms, although neither had received formal psychiatric treatment. Her therapy included 1) graded exposure in vivo to situations where she was likely to encoun-

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ter dogs and 2) marital therapy aimed mainly at improving the couple's sexual relationship, which had been virtually nonexistent for 4 years. Her total failure to respond, and the failure of a range of psychiatric treatments over the previous 4 years, led to her having a stereotaxic limbic leucotomy after 10 weeks. Subsequently, her fear of dogs was reduced enough for her to agree to a program of graduated exposure in vivo to a dog in the same room, with prevention of her washing and decontamination rituals. The marital therapy continued. After 3 sessions with a dog she was able (with her left hand) to touch it—the first time this had occurred for 12 years—but within 3 hours she developed a paralysis of her left arm, the nature of which was incompatible with any neurological deficit and therefore assumed to be hysterical. She was unable to move her left arm for 10 days and normal function did not fully return for 5 weeks. During the period of her partial paralysis, her fear of dogs virtually disappeared but returned in attenuated form on her recovery. The paralysis temporarily stopped the sexual aspect of marital therapy, which had been progressing very slowly and with great difficulty for both partners.

Case 3. Ms. C, a 44-year-old woman, married for 24 years and with a 21-year-old son, had been severely agoraphobic for 7 years prior to referral. She was virtually unable to leave her home unaccompanied because of weakness and trembling in her legs and fears that she would fall or faint and make a public spectacle of herself. After 15 hours of intensive real life exposure with a group of agoraphobics, she was able to travel alone to a local town and felt more confident socially than she had for many years. Three weeks after treatment, her husband deserted her without warning and went to live with a much younger woman who apparently closely resembled his mother at the same age. After the initial shock, Ms. C appeared to adjust to the situation, but within 6 weeks of being deserted she developed a florid psychosis, with delusions such as "my lungs have collapsed, my body is dead, I have bacteria all over me and my home, I smell all over. . . ." On admission to the hospital she kept exposing her breasts and genital area in order to demonstrate the bacteria. She said, "I have taken all the oxygen from the house; I can't walk out of the house because my lungs are gone. My husband is a murderer . . . all the bacteria have gone to my son's brain." Later she became convinced she was dying of lung cancer and/or stomach gangrene. She remained seriously disturbed for 7 months and was then discharged. Shortly thereafter she was readmitted to a surgical ward for treatment of bilateral calcaneal fractures sustained when she jumped from a first floor window of her home "to escape the smell of gangrene." She returned home 3 months later free of delusions and recovered from her injuries.

Case 4. Mr. D, a 24-year-old bachelor, was referred for therapy when he fainted at a party after hearing an account of a friend's minor operation. He had had a severe needle phobia since he was 8, when he fainted after an antitetanus injection. Subsequently he had been unable to go to the dentist, to discuss needles and hospitals, or to watch TV programs concerning such matters. His father and 3 older brothers also had phobias of needles, knives, and dentists, and the brother who had sought treatment had subsequently become massively obese.

Mr. D was given 5 hours of graded exposure in vivo to needles and medical equipment and environments, culminating in his own venipuncture. He then declared himself virtually free of his fears and within 6 weeks visited the dentist for the first time in 16 years, remaining fairly calm throughout his appointment.

Within days after treatment he began to experience panic attacks and dizziness before leaving home, abnormal anxiety in many social situations, and a fear of public transport, which he avoided if possible. On days when a train journey was unavoidable, he experienced distressing diarrhea and a fear of losing bowel control. Four months after treatment, his fears of public transportation were still troublesome, but tranquilizers helped him to cope. His fear of needles and hospitals had not returned.

DISCUSSION

Blanchard and Hersen (12) suggested that symptom substitution or return following behavior therapy is likely to occur only when the patient lacks the social or interpersonal skills to cope without the secondary gain derived from the symptoms. It seems important to distinguish between the precise nature of new symptoms and the use to which they are put: it is difficult to see how new or previous learning can account for the fairly sudden appearance of radically different symptoms after treatment, but it may well explain how the symptoms are handled by the patient and in particular the way they are used to try to influence others.

If learning cannot readily account for the precise nature of the new symptoms in these 4 cases, can psychoanalytic theory? Central themes in psychoanalytic explanations of phobic behavior (13) are the repression of threatening impulses or feelings and their displacement onto an external object, which then becomes fear-provoking and is avoided if possible. Interpersonal theorists (13) emphasize the unconscious need of the phobic person to be protected by their counter-phobic companion from acting on forbidden impulses.

In case 2, touching the dog may have reduced the patient's capacity to displace forbidden or unacceptable impulses onto it, placing a greater emphasis on the defense mechanism of repression. Some of the "badness" contained symbolically within the dog may have been felt by the patient to enter the left hand, leading her to attempt to somehow dissociate the hand from the rest of her body. Paralysis of the left hand and arm had the additional (secondary) gain of temporarily stopping the highly challenging sex therapy. These three relatively separate factors may have combined to generate the hysterical paralysis.

In case 3, the loss of the patient's counter-phobic companion of 24 years (her husband) may have left her feeling unable to control or contain her unacceptable aggressive and sexual feelings and impulses. Only through psychotic mechanisms could these impulses be both endured and expressed, e.g., her sexual acting-out—ostensibly because of the bacteria but in reality allowing the overt and grossly inappropriate expression of sexuality.

Case 4 is perhaps most readily explicable in psychoanalytic terms. The original fear of needles and hospi-

tals contained within it the patient's displaced (and denied) aggressive impulses. Removal of the fear left Mr. D having to contain these impulses within himself. His fear of losing control of aggressive impulses was reflected in his sharply increased social anxiety, panic attacks, and fear of loss of bowel control. His brother may have reacted to the same problem after treatment by trying, through massive weight gain, to become substantial enough to contain his aggressive impulses.

Although psychoanalytic theory helps explain aspects of the behavior of 3 of these 4 patients after treatment, can it explain why such phenomena were observed in only 4 out of 83 cases? If the inappropriate use of psychological defense mechanisms underlies phobic behavior, then the successful removal of phobias should merely expose this—unless during and after treatment the patient learns more adaptive and appropriate defenses. I have shown elsewhere (14) that the successful treatment of agoraphobia in married women appears to require changes in the personality structure of both patients and their spouses that reflect a more normal use of defense mechanisms. It may be that the emergence of radically new symptoms after behavior therapy is most likely to occur in that small proportion of patients who are unable to achieve a more appropriate use of defense mechanisms after symptom removal. The precise nature of new symptoms may be importantly determined by the way in which defense mechanisms are employed. Such a view is not necessarily incompatible with the notion that phobic and obsessional symptoms are learned: the inappropriate use of defense mechanisms may be one aspect of such learning. If behavior therapists were more willing to acknowledge that fresh symptom emergence might occur after behavior therapy, they could apply to the phenomenom their powers of objective and systematic observation. It may then appear that psychoanalytic theory is both testable and verifiable and that in the course of such testing a fruitful integration of psychoanalytic and behavioristic concepts occurs.

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Emergency Room Patients in Concurrent Therapy: A Neglected Clinical Phenomenon

BY FREDERIC KASS, M.D., TOKSOZ B. KARASU, M.D., AND TIMOTHY WALSH, M.D.

The authors found that more than one-third of emergency room patients were participating in concurrent psychiatric treatment. Exploration of the relationship between patient and therapist proved to be a valuable tool in formulating consultation and in ensuring continuity of care.

THE TRADITIONAL emergency room consultation has included an evaluation of the patient's important social and familial relationships. For patients who are participating in psychiatric treatment, the therapeutic relationship deserves the same careful clinical attention.

We have observed that many patients who visit our psychiatric emergency room are involved in treatment. The psychiatric residents who staff our service have difficulty assessing thoroughly the treatment relationship and offering appropriate consultation. Our impression is that residents tend either to deny difficulties which may exist in the treatment or to participate with the patient in an unproductive blaming of the patient's therapist. The literature, including a recent comprehensive review, has not addressed this phenomenon (1).

The present study was designed to determine the percentage of emergency room patients who are already involved in psychiatric treatment and to develop a rationale for consultation.

METHOD

This study was conducted in the psychiatric emergency room of the Bronx Municipal Hospital Center, Albert Einstein College of Medicine, which has been described previously (2). We interviewed 100 consecutive patients during random tours of emergency room service. In addition to the routine psychiatric evaluation, patients were asked questions designed to elicit information about concurrent therapy, expectations from treatment, and feelings toward therapists.

Patients were considered to be involved in concurrent psychiatric treatment if they had regular appointments with a therapist whom they had seen within the previous 6 weeks. High-frequency therapy was defined as one or more sessions a week and low-frequency treatment as less than one session a week. Patients who were participating in intake evaluation or had been seeing a new therapist for less than one month were categorized as beginning treatment.

RESULTS

Of 100 patients interviewed, 36 were found to be in concurrent psychiatric treatment. There were no significant differences in age, sex, or diagnostic distribution between patients in treatment and those not in treatment.

Patients in High-Frequency Therapy

The largest subgroup of emergency room patients in concurrent treatment (N=18) was involved in high-frequency therapy. These patients described their treatment as psychotherapy, with a main focus on discussion and understanding of their problems. There was evidence that many of these patients had strong feelings toward their therapists.

In the following case, the therapist's countertransference may have been a factor in the failure to deal with the patient's strong negative feelings.

Case vignette. A 22-year-old woman came to the emergency room after cutting her wrist. Subsequent to a hospitalization 5 months earlier, she had attended twice-weekly sessions with a psychiatric resident. The patient criticized her therapist, particularly for forgetting a recent appointment. In this context, she stated that her suicidal gesture was proof of the therapist's inadequacy. When contacted by telephone, the resident stated in a pejorative tone, "She is a very sick girl, clearly a schizophrenic, and the wrist cutting is a manipulation." Although he seemed unaware of his frustration and

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annoyance, the therapist's behavior and manner indicated considerable anger and unresolved countertransference.

Patients in Low-Frequency Treatment

Patients in low-frequency treatment (N=9) were chronic schizophrenics who usually came to the emergency room because of problems related to medication. The condition of several patients was deteriorating, apparently because their therapists had confused akinesia with depression or akathisia with anxiety. Recently, several authors have pointed out the high frequency of undetected side effects in patients who are taking psychotropic drugs (3, 4), and Van Putten (5) has suggested that these side effects are a major cause of drug noncompliance. Most of these patients described a withdrawn and socially isolated existence. They may have selected low-frequency treatment to minimize personal contact and prevent activation of conflicts over closeness. In this context, the patient's visit to the emergency room allowed him or her to solve problems without making special requests of the therapist.

Patients Beginning Treatment

Patients who were beginning treatment (N=9) usually acknowledged difficulty in communicating needs to their new therapists. Such patients came to the emergency room in a search for care and attention, often expressed symptomatically in the form of self-destructive behavior. Interviews with the therapists suggested that they may have been uncomfortable with the urgency of the patients' needs. Lazare (6) has noted that some therapists fail to empathize with new patients because of their own difficulty in experiencing the magnitude of the patients' problems (6).

COMMENT

We recommend that the standard emergency room interview include questions about the existence of concurrent treatment, the nature of that treatment, and the patient's current feelings toward his therapist. Direct questioning is indicated because patients may be initially reluctant to discuss treatment and frequently are unaware of significant transference feelings.

Because each case is unique, it is impossible to recommend a single approach to emergency consultation. A patient's visit to the emergency room during a psychotherapeutic crisis may represent acting out of the transference in an effort to alleviate painful feelings. In these cases emergency consultation can be initiated with the patient—contacting the therapist immediately could perpetuate the patient's defenses against autonomous initiation of communication within treatment. The patient's feelings about the therapist can be explored in a nonjudgmental manner. The conflicts underlying the patient's inability to express feelings in the sessions can also be identified.

When the patient has suicidal impulses and there is evidence of a therapist's negative countertransference (as in the case vignette) the therapist should be contacted. Failure to address the therapeutic impasse may foster the continuation of self-destructive impulses and behavior.

In cases where the concurrent treatment more approximates therapeutic management than psychotherapy, an attempt can be made to contact all individuals in the patient's milieu, including the therapist, and to gather information in order to make appropriate recommendations. For example, when the patient is experiencing medication side effects, these can be made known to the therapist. In our experience patients' therapists appreciated our suggestions and found the exchange of information valuable.

In conclusion, more attention to emergency room patients' involvement in concurrent treatment will contribute significantly to the well-being of these individuals and ensure an effective continuity of care. Further research in this area is indicated, particularly with regard to possible negative effects of treatment. Investigation of patients in concurrent treatment will provide a unique window[1] into the psychotherapeutic process.

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[1]The concept of a "window" into the psychotherapeutic process was provided by Richard Druss, M.D.

The Fiscal Crisis in New York City: Effects on the Mental Health Care of Minority Populations

BY PEDRO RUIZ, M.D.

The author describes the effect that severe budget cuts accompanied by guidelines stipulating that direct services be given priority over primary preventive services had on a community mental health center in New York City. He focuses his discussion on the effect these guidelines had on the mental health care services provided the minority populations served by the center. Before the budget cuts, 1 out of every 4 staff members was a nonprofessional indigenous worker. After the cutback, 2 out of every 3 of the people laid off were nonprofessionals. The author provides a number of recommendations to help government agencies plan budget reductions without sacrificing primary prevention efforts.

IN THE CURRENT DECADE the United States has gone through the worst urban crisis ever experienced by any country in the Western hemisphere. The promises of the "melting pot" that most large cities represented to different migrant groups no longer exist. Among the factors that contributed to the rise in urban populations are the following: the black rural migration to the large cities, the attraction that prospering industries offered the unskilled worker, the social advantages obtained by large labor movements in big cities, and the socioeconomic support extended to the poor and disadvantaged in urban areas. However, accompanying these developments were substandard and insufficient housing, a rise in the incidence of crime and delinquency, a weakening of the educational system, mass transit problems, difficulties in communication systems, mass unemployment, and deterioration of health care. In this paper I would like to focus on the impact that the fiscal crisis of New York City had on the mental health care systems for minority populations living in this metropolis.

As is well known, health costs have escalated during the last few years to such proportions that illness can represent a financial catastrophe for any family. Modern technical developments, high labor costs, expensive medical and paramedical training, and increases in malpractice insurance costs have contributed to this situation. Although we have been reading and hearing much about government debates regarding a national health insurance system, it is still far from being a reality.

In 1975 and 1976 the City of New York regrettably achieved notoriety as the prototype of a city in crisis with the emergence of serious financial insolvency. Among other services the city's health care delivery system was profoundly affected by severe budget cuts. I would like to analyze what I consider to be a disaster in health services by depicting the impact of the financial crisis on a large comprehensive mental health center located in a deprived ghetto area of New York City—the Southeast Bronx.

THE PROGRAM

In the 1960s community psychiatry ascended to the center of interest for American psychiatry (1). The emphasis on community psychiatry led to the development of many innovative approaches to the treatment of the mentally ill. In 1963 the federal government launched its "bold new approach," and Congress passed the Community Mental Health Centers Act setting specific guidelines for the operation of mental health centers. As a result of this piece of federal legislation, the Albert Einstein College of Medicine obtained a grant for the operation of the Lincoln Community Mental Health Center, a comprehensive mental health center in the southeast area of the Borough of the Bronx in New York City.

Since its inception in 1965, the Lincoln CMHC has been a vivid example of the prevailing social model in the delivery of human services emanating from the socially oriented movements that were a part of the "War on Poverty" (2). As a result of this, one of the center's major emphases was on the primary prevention of mental illness. The emphasis on primary prevention had already been emerging in the last decade in a number of different settings ranging from solo to group practices and including neighborhood health centers and teaching hospitals (3). In another framework, public health professionals were simultaneously stressing the provision of mental health services as a coordinated part of primary health care (4). The emphasis on primary prevention went hand in hand with the attempt to provide secondary and tertiary pre-

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vention programs as part of the armamentarium of service delivery. Along these lines, a major innovative feature in the service delivery of the Lincoln CMHC was using culturally sensitized and community-related paraprofessional staff. This kind of staff use has been given much recognition in the general medical literature (5) as well as in psychiatric literature. In developing outreach programs in consultation, education, self-help, and community action, the center attempted basically to foster the development of a more competent community (6).

NEW YORK CITY'S FINANCIAL CRISIS

Starting in the spring of 1975 the Lincoln CMHC was confronted with serious budget reductions as part of the local government's attempt to deal with its fiscal situation. Thus, with little warning and without opportunity to plan, the center was notified in June 1975 of a budget reduction of approximately $750,000 from an annual budget of close to $4 million. This budget cut had to become effective within one month—on July 1, 1975. In June 1976 another reduction of about $500,000 was implemented, thus reducing the center's annual budget to approximately $2.6 million by July 1, 1976. These two large budget cuts within a period of one year were accompanied by guidelines from the New York City Department of Mental Health and Mental Retardation. These guidelines gave priority to direct patient care, thus protecting "collectible" services covered by such third-party insurers as Medicaid and Medicare. These actions taken by the local government not only forced the reduction and elimination of essential services geared toward primary prevention (e.g., the consultation services to community agencies and community organization services) but also protected hospital-based programs throughout the city at the expense of community-based programs in poverty areas.

It is well known that hospital-based providers are more capable of maximizing third-party collections. This circumstance might well account for the fact that the Medicare and Medicaid systems in the city have become very expensive operations. An average New York City welfare family of four receives $4,500 annually for all living expenses other than medical expenses. The same family costs the city $9,000 a year in medical services (7). It should be noted that the guidelines regarding service priorities were put into effect without local community input. These guidelines were based exclusively on the opinion prevailing in the city's mental health bureaucracy.

DISCUSSION

Since the inception of the Lincoln CMHC, every effort was made by its administration to plan the staffing patterns as well as staff recruitment and selection in such a way that the program's overall philosophy and goals of primary prevention would be enhanced and a social model of service delivery would be emphasized. Table 1 shows the center's staff breakdown before the budget reductions and consequent layoffs. With full staffing, all community-oriented program goals were easily met. The percentages shown in table 1 are comparable to the population percentages in the catchment area served by the program, which according to the 1970 census report were 57% Hispanic, 33% black, 7% white, and 3% other.

Table 1 also shows that 1 out of every 4 staff members was a nonprofessional indigenous worker. These staff members represented a natural tie with local community groups and agencies because they were recruited primarily from the neighborhood itself and thus brought relevant sociocultural characteristics to the services offered. They also facilitated client identification with the program because these workers were predominantly black and Puerto Rican. It is also worth noting in table 1 that 27% of the professionals in the program were of minority background, thus reinforcing the community-geared and social model character of the program.

Table 2 shows the positions eliminated as a result of the budget cuts during the period from July 1, 1975, to July 1, 1976. It can be seen in this table that 2 out of every 3 individuals laid off were nonprofessionals. As a result of the priority guidelines put forth by the local government bureaucracy, the impact on minority employees terminated from the program was tremendous. There were unforeseen repercussions on the socioeconomic life of this disadvantaged neighborhood.

As a result of the budget cuts the center's administration had to reduce and subsequently to close its active consultation-education department, which employed 3 professionals and 12 nonprofessionals. The consultee community organizations left to fend for themselves included churches, settlement houses, youth and senior citizen's service organizations, and two elementary schools. The city bureaucracy's guidelines accordingly succeeded in effecting the elimination of all of the center's primary prevention activities while they protected direct services. In fact, there was a 15% increase in the individual and group therapy sessions, which were conducted for the most part by professionals.

It is my belief that the program goals, which were imposed on the center, will undoubtedly lead in the near future to an increase in the number of patients institutionalized. A financial saving for the city will thus bring damage to the patients, their families, and their hopes for reintegration into society. Once again, a severe blow has been administered to an impoverished minority community. In short, the city's approach in dealing with the fiscal crisis was to produce an immediate increase in revenue to alleviate its financial crisis. The result may be a frightful situation in terms of patient care, which will undoubtedly lead to an intensification of the fiscal crisis in the long run.

TABLE 1
Program Staff Breakdown as of July 1, 1975

	Blacks		Hispanics		Whites		Orientals		All Minorities*		Total	
Staff	N	%	N	%	N	%	N	%	N	%	N	%
Professionals												
Physicians	1	.5	13	8	3	2	4	2	14	8	21	12
Psychologists	1	.5	4	2	6	3.5	0		5	3	11	6.5
Social workers	3	2	6	3.5	2	1	0		9	5	11	6.5
Nurses	3	2	7	4	0		0		10	6	10	6
Other**	2	1	6	3.5	7	4	0		8	5	15	9
Total	10	6	36	21	18	10.5	4	2	46	27	68	40
Nonprofessionals												
Indigenous workers	21	12	23	13	1	.5	0		44	25	45	26
Clericals	15	9	21	12	1	.5	0		36	21	37	22
Service and maintenance	5	3	12	7	0		0		17	10	17	10
Other***	0		3	2	1	.5	0		3	2	4	2
Total	41	24	59	35	3	1.5	0		100	58	103	60
Total	51	30	95	56	21	12	4	2	146	85	171	100

*These figures exclude Orientals.
**Administrators, vocational counselors, art therapists, and sociologists.
***Case aides and pharmacy aides.

TABLE 2
Positions Eliminated After Budget Cuts from July 1, 1975, to July 1, 1976

	Blacks		Hispanics		Whites		Orientals		All Minorities*		Total	
Staff	N	%	N	%	N	%	N	%	N	%	N	%
Professionals												
Physicians	0		1	2	0		1	2	1	2	2	3
Psychologists	1	2	2	3	0		0		3	4.5	3	5
Social workers	1	2	2	3	1	2	0		3	4.5	4	6
Nurses	0		5	7.5	0		0		5	8	5	8
Other**	1	2	3	4.5	2	3	0		4	6	6	9
Total	3	6	13	20	3	5	1	2	16	25	20	31
Nonprofessionals												
Indigenous workers	11	16	14	22	1	1.5	0		25	38	26	40
Clericals	2	3	7	10.5	1	1.5	0		9	14	10	15
Service and maintenance	2	3	7	10.5	0		0		9	14	9	14
Other***	0		0		0		0		0		0	
Total	15	22	28	43	2	3			43	66	45	69
Total	18	28	41	63	5	8	1	2	59	91	65	100

*These figures exclude Orientals.
**Administrators, vocational counselors, art therapists, and sociologists.
***Case aides and pharmacy aides.

RECOMMENDATIONS

In my opinion, New York should have pursued different and less destructive approaches to deal with its financial crisis. These would include the following:

1. Needed expensive services, such as court programs, services for the mentally retarded, addictive services, and adolescent programs, should have been regionalized. Regionalization could have been done on a borough-wide if not city-wide basis, disregarding political considerations and focused on the needs for services in a geographic area. Regionalization would have eliminated unnecessary duplication of services and alleviated the fiscal crisis without curtailing essential programs.

2. Administrative control should have been centralized. This would have helped to reduce overhead and administrative costs. Centralization could also have been done without regard to political considerations, which are still regrettably prevalent in the city bureaucracy. Furthermore, centralization could have been directed toward program effectiveness and could have emphasized the protection of public services, thus benefiting the poor and the needy.

3. Consortium models should have been used for service delivery. This would have protected a larger number of agencies in the network of health care delivery. Undoubtedly, local community groups would have benefited by the use of consortium models. Without such models, large private or quasiprivate corporations will take control of health care services.

4. Planning for services should have been comprehensive, taking into consideration both long- and short-range plans that are intrinsically related rather

than just short-term plans. This would certainly have avoided the introduction of measures that would tend to create financial problems later.

5. The city-wide philosophy and goals for mental health care should have been redefined. One hopes that, as a result, comprehensiveness of services and continuity of care would emerge stronger than they were and would provide special support for primary prevention approaches. To pursue this goal, local and state governments would have had to collaborate in every aspect of mental health care delivery.

6. In addition to each agency's local community board, comprising civic leaders and consumers, some ways of community representation on a borough-wide basis should have been developed. It is well known that local community input, although vocal, inevitably gets lost in the maze of the city's bureaucracy. With government decentralization at a borough-wide level and community input centralized at the same level, perhaps the consumers would have a better opportunity to influence their own destiny.

7. The catchment areas should have been redefined, not according to political structures or to the providers' geographical controls but according to the natural locus of needs for services.

8. Unification of services should have been attempted. There should have been fewer providers delivering more services. These changes need to be backed by appropriate legislation at the federal, state, and local government levels.

9. Monitoring and evaluation of programs should have been done by independent third parties in order to avoid conflicts of interests.

These recommendations are especially important because the fiscal crisis of urban America is here to stay. Furthermore, even without a fiscal crisis, every attempt should be made to maximize program effectiveness and cost control for the benefit of the taxpayers.

CONCLUSIONS

The example of this large urban mental health center was presented in an attempt to demonstrate the impact that the New York City fiscal crisis had upon the mental health care delivery system, particularly on minority populations. I feel that the experience of the Lincoln CMHC is a good example of the way the city's mental health bureaucracy dealt with its fiscal problems. I hope that my observations will help government agencies in planning budget reductions while avoiding catastrophic consequences on the primary prevention aspects of health care. Politically, it is probably easier to sacrifice urban groups because they are composed of minorities, mostly blacks and Hispanics. Americans often blame societal problems on the big cities, forgetting that progress and the great strides in health care have also come from them. Urban communities like the Southeast Bronx represent not only a local crisis but also a national promise.

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Mood and Affect: A Semantic Confusion

BY HOWARD OWENS, M.D., AND JERROLD S. MAXMEN, M.D.

Although semantic clarity is a cornerstone of scientific psychiatry, psychiatrists often use technical words with the false assumption that their meanings are clear. The common use of the terms "mood" and "affect" illustrates this point. The authors attempt to show how unclear definitions of these words have led to confusion and how they could be more clearly understood by careful semantic and phenomenologic analysis.

> The merely descriptive literature of the emotions is one of the most tedious parts of psychology. And not only is it tedious, but you feel that its subdivisions are to a great extent either fictitious or unimportant, and that its pretences to accuracy are a sham.
>
> —William James (1)

A REVIEW of psychiatric texts and dictionaries reveals wide variations and contradictions in the definitions and uses of the terms "mood" and "affect." There are two common but unrelated ways of distinguishing the terms. Some writers emphasize the duration of an emotional state, regarding affect as a person's immediate condition and mood as a prolonged state or summation of numerous instances of affect (2–5). For example, Linn stated that "when an affective state is sustained for a considerable period, one speaks of a mood" (3, p. 808). However, does an affect need to exist for seconds, hours, days, or weeks before it evolves into a mood? Moreover, saying that affect and mood are transitory and persistent states, respectively, conflicts with popular usage. It is not uncommon to remark, "Right now, I'm in an angry mood."

The other approach depends on a distinction between subjective and objective emotional phenomena. Kolb (4) defined affect as "subjectively experienced." By contrast, other psychiatrists emphasize the subjective nature of mood and regard affect as, by definition, observable. For example, Hinsie and Campbell (6) stated that mood is "experienced internally," while affect refers to "external manifestations of the subject's feeling." English and English (7) likewise regard mood as "internal" and define affect as "a feeling state . . . accompanied by noticeable bodily activity." The latter definitions reflect the commonly held opinion that affect refers to observed reactions and mood pertains to verbal reports about one's "inner" state. However, in actual practice, clinicians sometimes conclude entirely by observation that a patient's mood is depressed (e.g., from the patient's knitted brow, slow movements, glum facies, sighs, etc.).

Another illustration of how psychiatric usage often fails to follow any of these standard distinctions is provided in Rapaport's description of practitioners' habitual use of the word affect to refer to phenomena that are both subjective and objective, temporary and sustained:

> We call affect not only the infant's rage . . . and the adult's anger accompanied by the corresponding expressive movements and other physiological concomitants, but also the subjective feelings of those well-controlled adults who show little or no affect expression, as well as the anger of over-controlling compulsive persons who just "know" that they "could be" or "should be" angry . . . And we must take notice of such affects as anxiety, guilt, elation, depression, which—in contrast to the momentary affects so far mentioned—may take pathological chronic forms; even more important, they may take characterological chronic forms, as in anxious people, gay people, gloomy people, bashful people, etc. (8, p. 478)

TWO ALTERNATIVE APPROACHES

Plutchik (9) maintained that because descriptions of the subjective aspects of feeling states lack reliability, they cannot provide a general basis for a theory of emotion. Instead, his theory of feeling states stresses their adaptive functions and the relationships among different types of emotions in terms of their intensity, similarity, and polarity. Although his ideas are useful in conceptualizing emotions from an adaptive perspective, they fail to clarify more fundamental problems, such as the meanings of and distinctions between mood and affect.

In order to address these issues, Ketai (5) reclassi-

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fied mood and affect into seven subcategories. His definitions primarily emphasize temporal distinctions: affect means an "immediate emotional experience," and mood constitutes a feeling state lasting over a "specified period of time." He also suggested that affects have greater intensity and flexibility than moods. Further, he considers both mood and affect to have subjective and objective components; "affective sensation" refers to a patient's subjective reports, while "affective display" means the overt behaviors associated with an emotion. He emphasized the need to describe the qualities of affects, such as their range (variability), intensity (strength or depth), and appropriateness (to current situations and contents of speech). Although Ketai's classification system clarifies certain issues, it tends to rehash previously cited definitions. More importantly, it avoids fundamental semantic and phenomenologic issues that must be addressed if one seeks a fuller understanding of the terms mood and affect.

TOWARD SEMANTIC CLARITY

Hayakawa (10) pointed out that defining words by other words at the same level of abstraction (e.g., saying that affect is an emotional experience) leads nowhere; the best definitions are those which are referable to lower levels of abstraction, i.e., closer to concrete phenomena. The problem with most traditional psychiatric distinctions between mood and affect is that they are based on concepts of emotion which are not readily translatable into concrete clinical phenomena.

Psychiatrists have confused the meaning of terms like mood and affect because they have misconstrued the process by which we perceive and describe emotions. Mood and affect have been regarded as concrete, observable phenomena, when in fact both terms are abstractions, i.e., they are based on inferences from observable phenomena. In examining a patient the psychiatrist makes observations that can be classified in various ways—physical signs (pallor, tremor), facial expressions (grimaces, smiles), motor activity (fidgeting), verbal statements ("I feel awful"), and other utterances (groans, sighs)—all of which are objective. Based on these observations, the psychiatrist proceeds to make inferences about affect and mood. The process of inference is necessary because affect and mood are not directly observable.

Schafer (11) argues that psychiatrists have reified concepts like affect. With regard to the concept of anger he writes,

> We have the illusion that we are referring to something that may be designated by a concrete noun, that is, to a unit that can be pointed to, like a claw, or a fang, or a fecal mass. But that is not what anger is. The anger of common usage is really an abstract noun; it is a rubric for a set of referents, and it is the referents rather than anger that can be pointed to." (11, pp 424–425)

Thus a psychiatrist can point to physiological events like pallor or tremor or to specific statements that a patient makes, but he cannot observe anger itself. The statement "He shook his fist" reports an observation. If we say, "He was angry," we have already made an inference. If the patient says, "I'm angry," then it is he who has made an inference, which he is reporting. The patient is not observing his own anger directly any more than we are.

The attempt to construe mood and affect as subjective or internal merely conceals a popular misconception. Schafer (11) points out that the mind is not localized in space, and affects cannot be "inside" anything in any sense but a metaphorical one. That an affect can, of course, be private does not mean it must be construed as "internal." Private thoughts are those which are not shared, including those which are unconscious. If a patient's affect is kept private, then clearly no one else knows about it. Any conclusion we reach about a patient's affect is necessarily based on what he has shared or exhibited so that it is no longer private.

Schafer acknowledged a debt to Ryle (12), whose analysis of the mind devoted considerable attention to the concept of mood. Ryle demonstrated that philosophers and psychologists have often conceived of mental operations as chains of cause and effect events analogous to physical causes and effects. According to this view there is supposed to be an internal mental cause, such as mood, which is not directly observable but which produces overt reactions and behavior. In fact, no one's mind operates this way. "When we describe people as exercising qualities of mind," writes Ryle, "we are not referring to occult episodes of which their overt acts and utterances are effects; we are referring to those overt acts and utterances themselves" (p. 25). When a person identifies his own mood, he does so by recognizing concrete bodily feelings (such as fatigue, heaviness, or sighs) and particular thoughts (that he is inept, worthless, stupid). There is no such thing as purely "internal" mood that exists in isolation from such feelings or thoughts. Similarly, we can only tell if another person is depressed by observing whether he acts or sounds depressed; we cannot examine any hidden mental events "behind" his expressions. It was for this reason that James (1) went so far as to identify emotion with the perception of bodily changes: "If we fancy some strong emotion," he wrote, "and then try to abstract from our consciousness of it all the feelings of its bodily symptoms, we find we have nothing left behind. . . . A cold and neutral state of intellectual perception is all that remains" (1, p. 451). A mood does not produce bodily feelings but rather is an inference from them.

Ryle (12) clarified this distinction by pointing out that a mood is nothing more than a disposition to act or react in a certain way. A disposition (such as intelligence or depression) is not an event (such as solving a problem or crying). The relation of a disposition to its exercise in behavior is not causal. A disposition

is not witnessable; only its exercise in specific actions can be witnessed. Recognizing a disposition entails a prediction. If we say that a man is depressed, we are saying that he is likely to speak morosely, perhaps to cry, move sluggishly, etc., and we base this conclusion on some previous patterns of such behavior.

Similarly, in speaking of an unconscious affect that manifests itself neurotically or characterologically, the clinician is making both inferences and predictions. The conclusion that a patient is unconscious of his depression is an inference derived from observations of or interactions with him. Statements about unconscious affect are often erroneously taken to be cause and effect statements. For example, an adolescent girl who runs away from home is described as acting out feelings of depression of which she is unaware. Rather than saying that depression caused her to run away, it would be more precise to frame a prediction about her affect on the basis of her manifest behavior. The clinician might then say that if the patient stops running away, she is likely to feel depressed. Just as a conscious affect may be inferred from sobs and moans, so a potential affect may be inferred from neurotic acting out.

Thus, the difference between mood and affect does not lie in the former being subjective and the latter being objective. Mood and affect can both refer to a person's disposition to react emotionally in certain specific ways. Consequently, to describe a particular mood or affect entails a prediction about how a person will act, based on previous patterns of behavior. Both mood and affect are abstractions, and both are inferred from concrete phenomena. The overlap between the two concepts is considerable. Perhaps the main distinction between them is that inferences about mood stem from present observations and past events (and therefore belong in a patient's history rather than as part of his mental status examination), while inferences about affect usually pertain only to current observations. We have dignified this distinction largely because these two terms are so thoroughly entrenched in our clinical vocabulary.[1] Nevertheless, we would suggest that emotional states could be described quite adequately without resorting to this distinction.

To include mood and affect side by side in a mental status examination amounts to a category mistake because it implies that there are two separate sets of observations. In fact, there is only one set: any observable event or bodily feeling from which the clinician makes an inference about affect could also be incorporated into a conclusion about mood. The converse, however, may not be true. Because statements about mood are usually derived from observations of past as well as present phenomena, all referents to mood do not apply to affect. All observations about current emotional expression can be included in a single category. Whether we call this category "affect" or some other word (e.g., "feeling tone") the important point is to recognize the distinction between observations and inferences when describing emotion.

We believe that there can be greater clarity in reporting a psychiatric case if this distinction is underscored: inferences about emotional states should not be reported without the concrete observations that led to these inferences. Just as a good clinician offers examples that justify the statement that a patient is delusional (e.g., the patient's stated belief, "I'm Jesus Christ"), so should he describe the events (e.g., crying and frowning) that justify his conclusion that a patient is depressed. When such observations do not accompany inferences about emotional states, the reliability of the inference is open to question. Moreover, because the range, duration, appropriateness, and intensity of these phenomena (5, 9) are also observable, statements about these qualities will further enhance the reliability of one's description. The patient himself can be introduced to this way of thinking by reporting specific events and feelings to explain a conclusion. The observations of both clinician and patient can be reported together, and inferences can then be made from the whole body of observations rather than being mixed in with them. Implementing these guidelines will reduce the current semantic confusion about describing emotions.

[1]It was primarily for this reason that, after some debate, the Task Force on Nomenclature and Statistics preferred the term "affective disorders" instead of "mood disorders" for the proposed *DSM-III*. Because we view mood as a disposition persisting over time, and because the Task Force itself defines affective disorders as being "characterized by a primary disturbance of mood" with the term "mood" referring to a "prolonged emotion" (13), it might be more accurate to call this category "mood disorders" in the final version of *DSM-III*.

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CLINICAL AND RESEARCH REPORTS

This section contains 1) new research findings, including preliminary data from pilot studies, either clinical or laboratory; 2) worthwhile replication studies; 3) case reports that describe a truly new syndrome or cast new light on established ones; and 4) case reports that indicate a new therapeutic procedure of potential value or call attention to adverse effects of drugs or previously unreported complications of therapeutic interventions. Program descriptions and literature reviews cannot be printed in this section. Criteria for format are listed in "Information for Contributors" in each issue; papers that do not adhere to these criteria will be returned to the author.

Noradrenergic Hyperactivity in Opiate Withdrawal Supported by Clonidine Reversal of Opiate Withdrawal

BY MARK S. GOLD, M.D., D. EUGENE REDMOND, JR., M.D., AND HERBERT D. KLEBER, M.D.

The widespread use and abuse of opiates and their unique effects on mood have generated considerable interest and study by diverse health and research scientists. However, the studies and the literature have failed to produce a unifying theory of opiate action on neurotransmitter systems or to lead to new nonopiate medical treatments for opiate withdrawal. We previously hypothesized that the locus coeruleus or other brain regions inhibited by both opiate and alpha-adrenergic receptor stimulation are responsible for opiate withdrawal (1). We proposed this hypothesis after observing that specific behaviors elicited in primates by dangerous situations, locus coeruleus stimulation, and drugs like piperoxane, which activate the locus coeruleus, were strikingly similar to the behaviors and physiologic changes associated with opiate withdrawal. We concluded that locus coeruleus stimulation or piperoxane administration could be useful as a model for the signs and symptoms of opiate withdrawal, so that drugs which block the specific behaviors elicited by these provocative procedures, like clonidine (2, 3), and which inhibit the function of the locus coeruleus may be useful in the medical treatment of opiate withdrawal and naturally occurring anxiety states. Preclinical studies have suggested that the pontine noradrenergic nucleus locus coeruleus is a brain alarm modulator (2–5) which may be essential to the human emotions and behaviors relieved by opiates and evoked in opiate withdrawal (1). These data and our evidence of clonidine's efficacy as an effective nonopiate treatment for opiate withdrawal support the hypothesized noradrenergic hyperactivity as the neurobiological substrate for opiate withdrawal (1).

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Method

We administered 5 μg/kg of clonidine to 6 male opiate addicts after discontinuation of chronic methadone treatment (doses of 15–50 mg/day) and to 6 heroin addicts who exhibited signs and symptoms of opiate withdrawal. The subjects had been addicted to opiates for 2–10 years. Those subjects who were being maintained on methadone had been taking methadone for 6–60 months. All subjects gave informed consent to participate in the study, which required a 2-day phased withdrawal from methadone before admission to the research unit (for the methadone subjects) and at least 36 hours with no opiate administration. All subjects had objective signs of opiate withdrawal and urine specimens that showed residual opiates. Baseline observations for the presence or absence of objective withdrawal signs (e.g., rhinorrhea, lacrimation, increased blood pressure) were made every 30 minutes by a research nurse, beginning at 8 a.m. while the subjects were at bed rest. The 12 subjects were also asked to complete self-rated analogue scales for nervousness and tension. At 11 a.m. and 1 p.m. subjects received placebo or 5 μg/kg of clonidine orally in matching vehicles. All trials were double-blind, with patient, nurse, and physician unaware of the order of administration. All subjects completed the entire 3-hour baseline and two consecutive 2-hour blind trials.

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Results

As expected, the number and severity of opiate withdrawal signs and symptoms increased during the baseline period in all 12 patients. In both the heroin withdrawal and methadone withdrawal groups, clonidine administration produced a rapid, prolonged, and significant reduction of opiate withdrawal signs and symptoms. Methadone withdrawal signs were reduced from a mean just before clonidine administration of 13.7±1.2 (SEM) to 0.3±0.3 90 minutes after administration (paired t test, $p<.01$) and heroin withdrawal signs and symptoms decreased from a preclonidine mean of 8.1±1.1 to 0.8±0.6 (paired t test, $p<.01$). All subjects also reported a dramatic relief of subjective distress. Patients who had reported they were "anxious, tense, or felt impending doom or death" denied these feelings at 120 minutes after clonidine administration. Nervousness and tension ratings on self-rating analogue scales were significantly decreased from a pretreatment mean of 54±3 (70 was the highest score) to 25.3±1.5 at 120 minutes. All 12 patients stated before clonidine administration that they were in withdrawal or "kicking" but said they were not kicking 120 minutes after administration. There were no reports of euphoria or "feeling high" after clonidine. Placebo had no significant effects. Five methadone withdrawal patients were offered clonidine alone on discharge and all chose this over a return to methadone. All heroin withdrawal patients were offered clonidine alone to be taken on an outpatient basis for 1 week; again, all chose this option.

All outpatients received 5 μg/kg of clonidine orally b.i.d. for 1 week. Outpatients were rated for abstinence signs daily for the week of outpatient clonidine treatment and for 1 additional week of follow-up. The only consistent complaint in both groups was occasional sluggishness and a sleep continuity disturbance. There were no signs of an exacerbation of opiate withdrawal, and 10 patients were clonidine-free and opiate-free at the 2-week follow-up. All of these patients had made previous unsuccessful attempts to withdraw from opiates. Although it is too early to evaluate the effects of a brief clonidine treatment on permanent abstinence, these data strongly support preliminary evidence for the efficacy of clonidine in the acute treatment of opiate withdrawal and as an alternative detoxification strategy (1).

Discussion

This trial was undertaken with specific hypotheses as to the mechanisms responsible for opiate withdrawal, based on our studies of the major noradrenergic nucleus locus coeruleus (LC). Striking similarities were noted between the effects of electrical or pharmacologic activation of this nucleus and those noted after opiate withdrawal. Both morphine and clonidine blocked the effects of electrical stimulation and pharmacologic activation of the LC in primates (2, 5). Although clonidine and the opiates blocked the effects of electrical stimulation and pharmacologic activation of LC, decreased neuronal activity in the LC, and decreased norepinephrine (NE) release (6–8), clonidine appears to exert its effects through nonopiate alpha-2-adrenergic receptors (6). It also has been recently demonstrated by Simon[1] that clonidine does not have any significant affinity for the opiate receptor. These data suggest that the opiate withdrawal syndrome is due to increased NE neuronal activity in areas such as the LC which are regulated both by alpha-2-adrenergic and opiate receptors. These data also support the notion that clonidine is an effective nonopiate treatment which replaces opiate-induced inhibition by augmenting alpha-2-mediated inhibition of noradrenergic activity. Our findings suggest that changes take place in the sensitivity of the alpha-2 receptors that are thought to regulate neuronal activity of the NE nuclei by powerful recurrent collateral inhibition (6). Since neuronal activity of these NE systems would be chronically inhibited by exogenous opiate administration, these receptors might also become subsensitive to the nonopiate neurotransmitter that normally inhibits their activity. Pharmacologic agents such as clonidine, which specifically inhibit noradrenergic neuronal activity via these alpha-2 receptors, would be expected to ameliorate withdrawal symptoms and possibly normalize receptor sensitivities. Although the exact neural mechanism mediating opiate withdrawal symptomatology remains to be defined, the absence of opiates in the dependent individual could readily result in a loss of opiate inhibition in brain areas like the LC and subsequent large increases in noradrenergic neuronal activity and NE release. Our hypothesis of noradrenergic hyperactivity as the neurobiological substrate mediating opiate withdrawal signs and symptoms can explain the effects of clonidine and the partial effects of other nonopiate medications on opiate withdrawal symptomatology (9, 10) by their inhibitory action on NE function.

This preliminary study supports an augmented noradrenergic system, released from opiate inhibition, mediating opiate withdrawal. It also suggests that clonidine may be a more definitive treatment for opiate withdrawal signs and symptoms produced by the discontinuation of either methadone or heroin than any treatment now available. The data indicate that opiate withdrawal may be a model for naturally occurring anxiety or panic states in man and suggest anxiolytic activity for clonidine.

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Endocrine Testing in Tardive Dyskinesias: Preliminary Report

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Tardive dyskinesia is a buccal-lingual-masticatory triad associated frequently with abnormal movements of the extremities and occasionally of the trunk. Long-term administration of antipsychotic medication is often implicated as the causative factor, although other medications are also believed to be predisposing and/or causative factors. Some of the dyskinesias are preceded by ECT and brain damage (such as cerebrovascular accident); others seem to occur spontaneously even in the absence of a drug history. The dyskinesias occur more frequently in women and in older patients than in men or younger patients (1-3).

Recently, Davis and associates (4) and Alpert and associates (5) reported a high incidence of affective disorders in patients with tardive dyskinesia. Our data (3) showed that 17 of 18 consecutive patients with tardive dyskinesia had primary affective disorders and that responses of dyskinesia and depression to treatment paralleled each other. These findings led us to hypothesize that primary affective disorders are a major predisposing factor for the development of tardive dyskinesia and that patients with tardive dyskinesia have neuroendocrine abnormalities similar to those of patients with primary affective disorders (6). To test this hypothesis, we measured the levels of 24-hour urinary free cortisol (UFC) and blood thyroid-stimulating hormone (TSH) in consecutive patients with tardive dyskinesia and compared these levels with those found in controls. In patients with primary affective disorders, 24-hour UFC levels were elevated (6) and the response of TSH to thyrotropin-releasing hormone (TRH) stimulation was reduced (7). Further studies have indicated that the high level of glucocorticoids may blunt this response (8); thus, there may be an inverse relationship between the level of corticosteroids and that of TSH.

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Method

In 18 consecutive patients with tardive dyskinesia (diagnosed by one psychiatrist and one neurologist) who were not receiving antidepressant medication, the values for 24-hour UFC and blood TSH were determined. Five patients who were tested before August 1977 did not have serum TSH determinations.

TSH levels were determined by a double-antibody radioimmunoassay procedure. TSH standard 68/38 was obtained from the National Institute for Biological Standards and Control, Mill Hill, England; UFC was determined by a method modified from that of Murphy (9) by a double-extraction technique. The standard was obtained from Sigma Chemical Company, St. Louis. The mean level in 100 adult control patients was 66 μg/24 hours, with an upper limit (2 standard deviations above the mean) of 108 μg.

Results

Values for UFC (see table 1) ranged from 81 to 630 μg/24 hours, with a mean of 198 (SD=147). The difference between our study group and the adult control group was statistically significant ($p<.01$, one-tailed Student's t test). Of the 18 patients, 13 (72%) had elevated UFC levels (>108), and 2 (11%) had upper normal levels of UFC. Of these 15 patients, all but 2 (pa-

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TABLE 1
Urinary Free Cortisol and Thyroid-Stimulating Hormone in Patients with Tardive Dyskinesias

Patient	Sex	Age (years)	UFC (μg/24 hours)	TSH (μU/ml)
1	F	70	81	—
2	F	67	216	—
3	M	67	110	—
4	M	35	630	—
5	F	61	175	—
6	F	33	206	4.1
7	M	64	212	1.4
8	M	66	159	1.0
9	F	49	197	2.6
10	M	39	500	2.6
11	M	40	102	2.4
12	M	58	102	8.2
13	M	39	110	1.0
14	F	62	83	6.9
15	F	68	184	2.1
16	F	67	89	4.4
17	F	74	127	3.7
18	M	63	281	1.5
Normal			≤108	0–15

tients 4 and 6) fulfilled at least four of the depressive symptoms in the Research Diagnostic Criteria (RDC) (10), indicating at least "probable" depression, although only 9 of the 13 (69%) claimed a dysphoric mood. Patient 4 fulfilled the RDC criteria for mania; patients 6, 11, and 13 had delusional ideations. None of these 15 patients met RDC criteria for schizophrenia. However, all of these patients had been receiving some type of antipsychotic medication before the onset of the tardive dyskinesia.

Of the 3 patients with normal UFC levels, 2 (patients 14 and 16) had no drug history before the onset of the tardive dyskinesia. Patient 14 had suffered a cerebrovascular accident before the onset of the dyskinesia and was given haloperidol to control the dyskinesia. The patient denied feelings of depression, but she fulfilled the other RDC criteria for depression with severe withdrawal. Except for some tooth extractions, patient 16 had no significant history before the onset of the tardive dyskinesia. According to RDC, she was depressed. The third patient (patient 1), who was severely depressed and had nihilistic delusions, was receiving antipsychotic medication.

UFC and TSH levels were determined after treatment for depression for 4 patients. Patients 5 and 21 were in remission and had UFC levels of 42 and 52 μg/24 hours and TSH levels of 9.1 and 17.9 μU/ml, respectively. Patients 8 and 12, who were in partial remission, had UFC levels of 99 and 128 and TSH levels of 2.8 and less than 1, respectively.

TSH values tended to be inversely related to the cortisol levels ($p<.12$, Fisher's exact test); in our laboratory, TSH levels of 0–3 are considered to be essentially zero. Most patients with TSH levels greater than 3.0 had UFC levels less than 108.

Discussion

Carroll and associates (6) have reported elevated UFC levels in primary affective disorders but not in schizophrenia. Our present data showed that 13 of the 18 patients (72%) had elevated UFC levels, which supports our hypothesis that the endocrine dysfunction in these patients is similar to that in patients with primary affective disorders. Further, patients with low TSH levels tended to have elevated UFC levels, which suggests an inverse relationship. None of these patients fulfilled RDC for schizophrenia at the time of our evaluation. Instead, all of the patients with elevated UFC levels, except the manic and paranoid patients, fulfilled four to six of the eight RDC depressive symptoms, although many denied a dysphoric mood.

Repeat UFC and TSH determinations further supported our hypothesis. Two patients in remission had normal levels of both UFC and TSH, and 2 patients in partial remission had elevated UFC levels and low TSH levels.

Although our study is a preliminary one, the findings indicate that a subgroup of patients with tardive dyskinesia have endocrine dysfunction similar to that seen in primary affective disorders. To confirm our hypothesis, further testing using dexamethasone suppression and TRH stimulation, along with determination of UFC levels, seems to be indicated. Meanwhile, physicians may benefit by reevaluating their use of antipsychotic medications for patients who have symptoms of primary affective disorders.

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Diazepam Withdrawal Syndrome: A Case with Psychosis, Seizure, and Coma

BY MARK L. DE BARD, M.D.

Since its introduction into clinical use in 1963, diazepam has been considered nonaddicting. However, for years the literature has been sprinkled with reports to the contrary. Phenobarbital was also initially thought to be safe and free of addictive properties until accumulated clinical experience over many years proved otherwise (1). Amphetamine, introduced clinically in 1935, was not realized to be addictive until about 1958.

Numerous authors have warned of the existence of a diazepam withdrawal syndrome (1–3). At least 8 cases of acute organic brain syndrome thought to be secondary to withdrawal of diazepam have been described (4, 5). Thirteen cases of grand mal withdrawal seizures have been reported (3–9). One case of prolonged coma following diazepam withdrawal was found (10).

I will describe a case that combined an acute organic brain syndrome and prolonged coma in three separate instances, along with a grand mal seizure in one instance, in a patient who could not be withdrawn from diazepam.

Case Report

The patient, a 56-year-old man, was seen in the emergency room for a suicide gesture. The mental status exam showed him to be much more apathetic, withdrawn, and depressed than he had seemed during an examination 2 weeks earlier, and he had obvious suicidal ideation. His medications included diazepam, 10 mg q.i.d.

The patient gave no personal or family history of epilepsy or seizures. He used alcohol only socially, and this was confirmed by his relatives for the 3 weeks that he stayed with them before his admission.

The patient was admitted to the psychiatric unit with the diagnosis of involutional depression. All prehospital medications were continued with the exception of diazepam, and imipramine was started. He improved over the next week. However, 8 days after admission, he became despondent and preoccupied, with staring behavior. He complained of numbness and became very unsteady on his feet.

Over the next 2 days, the patient experienced visual hallucinations and disorientation. He became very restless, started perspiring, and suddenly had a grand mal seizure. A complete seizure workup was normal. He remained deeply comatose for the next 6 hours. His previous physician was contacted and the following history was obtained.

The patient had been taking diazepam, 80 mg/day, from 1969 through 1973. In 1973 he had an elective medical hospitalization. The diazepam was discontinued during this hospitalization, but no other medications were changed. He was not given tricyclic antidepressants then, and he had never had a seizure to that point.

Ten days after that admission the patient reportedly developed seizures and lapsed into coma for an unknown length of time. A neurosurgeon gave him intravenous diazepam. Over the next 24 hours the patient's sensorium cleared and he recovered completely. He was discharged with the diagnosis of acute brain syndrome secondary to diazepam withdrawal and was maintained on diazepam, 10 mg q.i.d.

Based on this information, the patient was given diazepam, 5 mg I.M. Several hours later he was sufficiently alert to take diazepam orally, 5 mg every 8 hours. Over the next 24 hours he began eating and returned to a normal mental status.

During the next 9 days the diazepam dosage was gradually decreased and then stopped. Imipramine was restarted. Ten days later, he began to show the same withdrawal symptoms and became comatose, with frequent jerking movements.

He was given diazepam, 5 mg I.M. The twitching stopped, but he remained deeply comatose, with flaccid extremities and no response to pain. He was given a second dose of diazepam, 5 mg I.M. Two hours later he had awakened enough to request some food. He improved over the next 18 hours and again attained a normal mental status. Diazepam was given in a maintenance dose of 5 mg q.i.d., and he was discharged 5 weeks after admission. He has been maintained on diazepam, and follow-up for 11 months showed no further problems.

Comment

The occurrence of a diazepam withdrawal seizure is not without precedent. Such convulsions have occurred 2–12 days after discontinuation of the medication, with an average of 8 days.

The persistence of coma is very unusual; the only similar case I am aware of occurred after high-dose therapy for tetanus (10). The occurrence of coma three times and the dramatic resolution within hours of diazepam administration seem to confirm this as withdrawal coma. This would seem to be a very important effect that has not received much medical attention.

One might say this patient's mental changes resulted from acute psychotic depression or even from the anticholinergic effects of imipramine. However, the temporal relationships do not favor these interpretations, and imipramine was not given the first time the patient underwent withdrawal. The recurrence of the same mental symptoms three times 8–10 days after discontinuation of diazepam firmly supports the diagnosis of acute organic brain syndrome secondary to drug withdrawal.

Discussion

It has been almost 15 years since the introduction of diazepam, and its addictive potential is just beginning to be realized. A physical dependence seems to devel-

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op in some patients who use the drug on a chronic basis (probably a minimum of 2 weeks). More severe withdrawal symptoms seem to be associated with a longer period of use and higher doses, but there is marked individual variation.

For many years it has been said that the "anxiety state" many people experience after they stop diazepam is just a return to their premedication state (3, 6). Maletzky and Klotter (3) question the feasibility of such a distinction between physiologic dependence and the recurrence of patients' original symptoms. Certainly, the more severe symptoms of withdrawal that I have described are no longer difficult to identify.

In conclusion, to prevent the development of even minor physical dependence, one should refrain from outpatient prescription of diazepam for longer than a month in any individual patient. Similarly, one should not abruptly discontinue diazepam treatment for patients who have been on long-term and/or high-dose therapy.

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Panic Attacks: Diagnostic Evaluations of 17 Patients

BY STEPHEN F. PARISER, M.D., BRUCE A. JONES, M.D., EMIL R. PINTA, M.D., ELIZABETH A. YOUNG, M.D., AND MARY E. FONTANA, M.D.

There are growing numbers of reports on anxiety-panic syndromes and their relationship to cardiovascular disorders such as hyperdynamic beta-adrenergic states and mitral valve prolapse syndromes (MVPS) (1–3). We recently reported on a case involving an association between panic disorders and MVPS (1). Panic attacks have also been noted to occur in affective disorders (depression) (4).

Treatment of panic attacks has challenged the medical community. Tricyclic antidepressants, monoamine oxidase (MAO) inhibitors, beta-blocking agents, and psychotherapy have all been reported effective to some degree in treating patients with panic attacks (5–7). Recent changes in psychiatric nosology have encouraged specific treatment in specific syndromes by way of exclusion-inclusion criteria. The draft third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (8) incorporates a variety of syndromes, referred to as anxiety disorders, all associated with one or more types of anxiety symptoms. Proper use of these diagnostic categories requires that the physician inquire as to the presence or absence of panic attacks. This is important because there is a need to differentiate patients with different forms of anxiety-related symptoms.

Anxiety symptoms are related to autonomic nervous system activity and to cognitive-affective functioning. Anxiety symptoms (panic or other symptoms) can be signs of any number of disorders. Our work focuses on patients whose chief complaint is panic attacks. We raise questions about the etiology of panic attacks, their relationship to cardiac and psychiatric disorders, and their treatment.

Method and Subjects

The subjects were adult patients who consulted one of the psychiatrist authors (S.F.P., B.A.J., E.R.P., or E.A.Y.) during a 6-month period with a chief complaint of panic attack symptoms. There were 24 patients, 11 men and 13 women, who had panic attacks diagnosed by the Research Diagnostic Criteria (9). Their ages ranged from 21 to 53 years. All were interviewed by a psychiatrist. Because of the nature of their symptoms, cardiology evaluation, echocardiogram, and thyroid studies (T_3, T_4, and FTI) were requested. Seven patients did not complete the full evaluation. We will report here on the 17 patients who completed the

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psychiatric evaluation, echocardiograms, and cardiology evaluations. Twelve of these 17 patients completed the thyroid studies.

Results

The RDC diagnoses made by psychiatric interview in these 17 patients were panic disorder (N=10) and major depression (N=5) (see table 1). No patient met RDC for schizophrenia, schizo-affective disorders, mania, hypomania, or Briquet's disorder. Depression was particularly common in women with panic attacks. Eight of 9 women but only 2 of 8 men met RDC for major depression at the time of the interview.

Cardiology evaluation showed that 6 patients had definite MVPS by cardiac auscultation and echocardiogram. Two other patients had positive echocardiograms and cardiac auscultatory findings that suggested but did not confirm MVPS. No patient had abnormal thyroid studies.

Comment

This clinical investigation suggests that the panic attack is a symptom complex with a variety of underlying etiologies. Panic attacks may be associated with affective illness, particularly in women, and with cardiovascular syndromes such as MVPS in addition to panic disorder. Our results suggest that clinicians should consider these diagnoses in patients with panic attacks. The exact relationship between MVPS, affective disorders, and panic attacks is controversial, but there may be common involvement of the autonomic nervous system. A relevant exclusion clause in the RDC for panic disorders eliminates patients who have other disorders that can account for panic symptoms. Therefore, if MVPS is a cause of panic attacks, only 6 of the 17 patients would qualify for the diagnosis of panic disorder, because 4 patients who met other RDC for panic disorder also had definite MVPS. The relationship between MVPS and panic attacks needs clarification. This study suggests that as the diagnosis of other disorders such as MVPS and affective illness is aggressively pursued, fewer patients may be diagnosed as having RDC-defined panic disorder. The diagnosis of associated disorders may permit better choice of pharmacologic treatment and better understanding of the specific pathophysiology of the syndromes.

TABLE 1
Psychiatric Diagnoses and MVPS in Patients with Panic Attacks

	All Patients			Patients with MVPS		
Psychiatric Diagnosis	Male	Female	Total	Male	Female	Total
Panic disorder	4	1	5	0	1	1
Major depression	2	8	10	1	2	3
No psychiatric diagnosis	2	0	2	2	0	2

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Childhood Cross-Gender Behavior and Subsequent Sexual Preference

BY RICHARD GREEN, M.D.

Are there childhood behavioral predictors of adolescent and adult homosexual orientation? Until the past decade there were essentially no data to support a positive response to this question. The answer is beginning to emerge more clearly as a result of both retrospective and prospective research. I have been primarily engaged in the latter.

Relevant retrospective reports are limited in number but clearly provocative. Whitam (1) studied 107 exclusively homosexual males and compared them to exclusively heterosexual males. He found that 47% of the homosexuals recalled being more interested in doll-play than other boys their age, 44% recalled liking to

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cross-dress more, and 42% recalled preferring girls' games and having a female peer group. In contrast, such behaviors were recalled by less than 1% of the heterosexuals.

Before the Whitam report appeared, a psychiatric investigation by Saghir and Robins (2) provided considerable support for a childhood behavioral pattern associated with both male and female homosexuality. Of the males studied, 67% of the 89 homosexuals versus 3% of the heterosexuals reported that they had played mostly with girls and were considered "sissy" or effeminate during boyhood. There was a significantly higher proportion of "tomboys" in the prehomosexual female sample (N=56) both in childhood (70% versus 16%) and adolescence (35% versus 0%), with lack of interest in doll-play discriminating prehomosexual from preheterosexual "tomboys."

In the late 1950s in collaboration with Money I initiated a pilot study of a small sample of behaviorally "feminine" boys (3, 4). Five years ago, I reassessed the few subjects still available from the sample and found them to be primarily homosexual. Because of the small number in the original sample (N=11) and the 55% attrition rate, I reflected on this outcome in a conjectural section of a book (5) but did not consider the data appropriate for a scientific report. More recently, Money and Russo (6) reinterviewed 5 of these persons and confirm that this sample remains homosexually oriented.

In collaboration with Stoller, I began a comprehensive longitudinal study of psychosexual development in two initially disparate male samples (7, 8). One sample consisted of boys who, when initially evaluated, preferred the dress, toys, activities, and companionship of girls, typically role-played as females, and stated their wish to be girls. The 60 boys ranged in age from 4 to 11 years. Fifty were matched with same-age boys from demographically comparable families who, at the time of initial evaluation, showed conventionally masculine behaviors. The extensive disparity in these boys' behaviors and the demographic similarities of the sample have been reported elsewhere (5, 8).

Some of these boys are now in early to mid-adolescence. More of the previously "feminine" boys are in this age group because the contrast group was generated later and thus is younger. This is the initial preliminary report describing the emerging sexuality of the older members of the "feminine-boy" sample.

Data on Previously "Feminine" Boys

These preliminary data on the "feminine-boy" sample will be divided into four areas: masturbation fantasies, reported erectile response to heterosexual stimuli, reported erectile response to homosexual stimuli, and interpersonal genital experiences.

For the 11 boys who have masturbated, none has exclusively homosexual associated fantasies, 4 have both homosexual and heterosexual fantasies, and 4 have exclusively heterosexual fantasies. Three boys report no fantasies. One boy who does not masturbate has nocturnal emissions that are activated exclusively by homosexual imagery, and another who does not masturbate has emissions activated by heterosexual imagery. Thus, 50% of the boys who have fantasies that accompany masturbation or nocturnal emissions respond to homosexual fantasies.

Twelve boys have viewed photos of nude males: 3 report that they usually get erections when viewing such a stimulus, 5 sometimes get erections, and 4 do not get erections. Of the 18 boys who have viewed photos of nude females, 6 usually get erections, 9 sometimes, 1 rarely, and 2 never. Thus, 3 of these 18 boys typically do not respond with erections to these heterosexual stimuli.

Of the 9 boys who have engaged in intercourse or heavy genital petting, 1 has had such experiences only with males, 5 with both males and females, and 3 with females only. Thus, two-thirds of this group is homosexually or bisexually oriented at present.

It is clear that feminine boyhood behavior, as defined by clothing, toy, peer group, activity, and role-playing preferences, does not consistently predict later homosexual orientation. However, from preliminary data, it does appear to load in favor of such an outcome in some persons.

"Tomboys"

Next, a word about the developmental course of "tomboyism." Certainly, "tomboyism" is more common than sissiness and more often converts to more conventional feminine behavior in adolescence. However, at least one study (2) suggests that "tomboy" behavior may be associated with later feminine homosexual orientation.

I am conducting a large-scale developmental study of "tomboys" (9). Previously, I initiated a small pilot study (5), without a control group, similar to the pilot study I did with Money. The sample consisted of 4 girls who insisted on wearing boys' clothes, were very interested in participating in sports, rejected doll-play, had a male peer group, and stated they wished they were boys. Their degree of "tomboyism" seemed greater than is typical for children commonly termed "tomboys." Recently, follow-up data have been collected on these 4 subjects, who are now midteenage or young adult.

One is living as a man, has had a hysterectomy, and is receiving exogenous androgen. Two are varsity athletes; one is behaviorally asexual, and the other has recently "discovered boys." Both are described by their parents as "still walking and sitting like boys" and avoiding cosmetics and conventionally feminine attire. The fourth is described as conventionally feminine and very interested in dating boys. Thus the range is considerable and, as with the Green and Money series, these data from a small sample with no controls must await more comprehensive follow-up with large samples.

Discussion

Our data so far indicate a moderate amount of homosexual fantasy, arousability, and overt behavior in a sample of boys who were previously "feminine." However, of considerable importance is the current variation in sexual orientation in these boys. One mid-adolescent is a "drag queen" and has had an extensive series of same-sex partners. One is genuinely bisexual, with an equal distribution of erotic fantasies and overt behaviors involving males and females. One prepubertal boy reports erections in response to fantasies or pictures of nude males but not females, although he has had no interpersonal genital experience. Others report an exclusively heterosexual orientation.

This variation in outcome is noteworthy. Clearly all boys who show behavioral patterns considered "feminine" are not prehomosexual. Parents of boys whose behavior does not fit the conventional stereotype of "masculinity" should not be rendered anxious by the belief that their children are prehomosexual. I have elsewhere (10) reviewed data supporting the conclusion that homosexuality is not a mental illness and does not predispose to poor social or psychological adjustment. However, many parents do become grief-stricken if they believe their children are or will become homosexual. Thus I feel it important to stress the markedly atypical early childhood behavior of the sample described here, and the considerable variability in their later sexual behaviors.

Our research task in the coming years goes beyond collecting sexual history on the remainder of the sample and extending the data on the subjects reported here. The major challenge is examining the early life behavioral and psychological test data on the sample, features of their parents, and developmental life events leading to adolescence and young adulthood. We hope to discriminate among those previously "feminine" boys who emerge with a primarily homosexual, bisexual, or heterosexual orientation.

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Serum Prolactin and Clinical State During Neuroleptic Treatment and Withdrawal

BY THOMAS P. LAUGHREN, M.D., WALTER ARMIN BROWN, M.D., AND BRADFORD W. WILLIAMS

Serum prolactin is elevated both acutely (1) and chronically (2) by neuroleptic drugs. The prolactin-elevating effect of neuroleptics correlates well with their antischizophrenic potency (1) and appears to correlate better with neuroleptic-induced sedation and EEG changes than does neuroleptic plasma level or dose (3). Clinical improvement during neuroleptic treatment does not appear to correlate well with prolactin elevation in most patients (4). This finding may be explained by the narrow range of sensitivity of the prolactin-elevating response to neuroleptics. An alternate approach to examining the relationship between prolactin concentration and clinical state is to monitor patients during withdrawal from neuroleptic treatment. The present report describes the relationship between serum prolactin and psychological state during chronic treatment with thioridazine and haloperidol, withdrawal from treatment, and return to treatment.

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Method

Informed consent was obtained from 11 male psychiatric outpatients aged 21–59 with diagnoses of

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TABLE 1
Serum Prolactin (ng/ml) and Change in POMS Scores During Thioridazine, Haloperidol, and Placebo Treatment

Patient	Dose* (mg)	Duration (years)	Chronic Treatment		Placebo Treatment			Acute Treatment		
			Mean	SE	Mean	SE	Change in POMS**	Mean	SE	Change in POMS***
1	20	1	3.8	0.1	1.0	0	+ 58	6.3	0.4	− 75
2	300	7	3.2	0.4	1.9	0.5	+900	7.1	2.1	− 55
3	200	14	5.8	2.1	3.7	1.9	+ 95	10.3	1.3	− 52
4	50	3/4	10.5	1.4	4.7	0.5	− 14	10.9	2.0	− 21
5	15	3 1/2	10.5	0.5	5.1	0.8	+ 4	17.1	2.2	− 43
6	75	1/3	10.6	0.6	5.5	1.4	+ 22	13.3	0.8	+ 2
7	15	3 1/2	45.5	14.4	6.9	1.5	− 22	48.3	17.2	− 17
8	300	6	13.0	0.9	7.2	0.2	−107	14.0	1.2	+386
9	75	1/2	12.4	0.7	8.1	1.1	+ 13	9.5	0.6	− 9
10	300	10	9.2	0.4	8.1	0.4	− 12	10.2	0.6	+ 25
11	400	2	23.0	3.2	9.5	2.4	− 45	18.6	2.8	− 38

*Patients 1, 5, and 7 were taking haloperidol; the remaining patients were taking thioridazine.
**Percent change in mean POMS scores from chronic to placebo period.
***Percent change in mean POMS scores from placebo to acute period.

chronic schizophrenia (N=8), hysterical neurosis (N=1), and alcoholism (N=1). Eight patients had been taking thioridazine in the range of 50–400 mg/day (mean=212 mg/day) for a period of 4 months to 14 years (mean=5.1 years). Three patients had been taking haloperidol in a range of 15–20 mg/day (mean=17 mg/day) for a period of 1–3.5 years (mean=2.7 years). Patients were taking no other medication. They continued their usual dose of medication for a 2-week baseline period (chronic), were given matched placebos for 4 weeks (placebo), and returned to their baseline dose for 4 weeks (acute). Four patients had shortened placebo periods because of the development of symptoms. A blood sample was obtained once weekly at 8:00 a.m. from patients who had fasted overnight and had been awake for at least 1 hour. The Brief Psychiatric Rating Scale (BPRS) and the Profile of Mood States (POMS) were completed for each patient.

Serum prolactin was measured by double antibody radioimmunoassay. All samples from the same subject were measured in duplicate in the same assay. Both the interassay and intraassay coefficients of variation were less than 7%. Laboratory personnel were blind to the study protocol.

Results

The mean serum prolactin concentrations (see table 1) were elevated during chronic (13.2 ng/ml) and acute (14.8 ng/ml) treatment and differed significantly from the placebo prolactin concentration (5.8 ng/ml, $F=6.8$, $p<.01$). The patients with the lowest prolactin concentrations chronically (less than 6 ng/ml) (patients 1, 2, and 3) showed the greatest relative clinical worsening during the placebo period (more than 50% increase in POMS scores, $p=.006$, Fisher exact probability test; more than 20% increase in BPRS scores, $p=.024$). These patients had BPRS scores similar to the other patients during the chronic treatment period. All 3 had schizophrenic diagnoses. During placebo, patient 1 developed delusional thoughts and bizarre behavior and patient 2 developed auditory hallucinations, insomnia, and marked anxiety. Although patient 3 did not show clear psychotic symptoms during placebo, he did become very anxious, sleepless, and more withdrawn. These patients also had the lowest prolactin concentrations during the placebo period (less than 4 ng/ml) and the greatest relative improvement during acute treatment (more than 50% decrease in POMS scores, $p=.006$). Patient age, drug, dose, and duration of treatment were not related to prolactin concentration or change in clinical state.

Nine of the 11 patients had lower mean serum prolactin concentrations during chronic than acute treatment ($p<.05$, sign test). The differences in prolactin concentration ranged from 4%–54% lower chronically than acutely. The same patients (patients 1, 2, and 3) showing the greatest clinical change during placebo and acute treatment also showed the greatest difference in prolactin concentration chronically compared to acutely (more than 39% lower chronically, $p=.024$).

Discussion

This study confirms earlier findings that male patients taking neuroleptics acutely or chronically have significantly higher serum prolactin concentrations than during drug-free states. The lower prolactin concentrations seen chronically compared with acutely in some patients are suggestive of at least partial tolerance to the prolactin-elevating effect. An earlier study (2) reported lower prolactin concentrations in female patients taking neuroleptics chronically (5 years or more) as compared with female patients taking equivalent doses acutely (2–4 weeks).

Other groups have not found tolerance effects to prolactin elevation, but these studies either examined prolactin concentrations over shorter time periods (1, 4) or did not maintain neuroleptic doses at fixed levels (4, 5). Our finding of tolerance to the prolactin-elevating effect of neuroleptics must be considered tentative until more data relevant to this effect are collected. Tolerance to the biochemical and behavioral effects of

neuroleptics in the basal ganglia has been demonstrated in both animal (6) and human (7) studies.

The patients with the lowest prolactin concentrations during the chronic and placebo periods also showed the greatest relative clinical worsening after drug withdrawal and the greatest relative clinical improvement after drug resumption. These patients also showed the greatest apparent tolerance to prolactin elevation. Possibly these patients represent a subgroup of schizophrenic patients with an overactive and labile dopaminergic system, which is reflected in generally lower serum prolactin concentrations and the development of partial tolerance to prolactin elevation. The increasingly ineffective dopaminergic blockade occurring in these patients could explain their greater vulnerability to relapse when completely withdrawn from neuroleptics. Alternatively, the patients showing apparent tolerance may not have been taking their medications regularly during chronic treatment and were more vulnerable to drug withdrawal on this basis. In either case, lower prolactin concentrations predicted greater susceptibility to clinical change with either drug withdrawal or treatment. Prolactin concentrations should be further explored as possibly useful predictors of clinical response following drug withdrawal.

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Use of Bethanechol Chloride with Phenothiazines: A Case Report

BY DANIEL S.P. SCHUBERT, M.D., PH.D.

Everett (1) reported the use of bethanechol chloride to treat dry mouth in patients taking tricyclic antidepressants. I have found this combination useful in several patients. Because dry mouth is also mentioned by patients taking phenothiazines, particularly in combination with antiparkinsonian agents, I thought bethanechol chloride might also counteract the anticholinergic effects in these cases. The case that I will describe is one in which the patient's most troubling symptom was blurred vision produced by the anticholinergic effects of the drugs.

Case Report

The patient, a 47-year-old man with chronic schizophrenia and a history of multiple hospitalizations, seemed relatively clear between episodes and was taking fluphenazine, 5 mg p.o., and benztropine mesylate, 2 mg. When he was hospitalized he was given 20 mg of fluphenazine and 8 mg of benztropine mesylate p.o., but because of his periodic noncompliance, fluphenazine decanoate, 25 mg I.M., was added. The patient began to complain of blurred vision as his psychosis receded. Because of my favorable experience with bethanechol chloride in treating dry mouth in patients given tricyclic antidepressants and because the anticholinergic effect seemed to account for both the blurred vision and dry mouth (2), I prescribed bethanechol chloride, 25 mg. t.i.d. No other change in medication was made at this time. Within 2 days he was able to read small print again. He reported that the dry mouth, which had not troubled him as much as the blurred vision, also remitted, although more slowly. He was continued on the three medications after he was discharged and continued to report that he could read well and had fewer symptoms of decreased saliva output.

Discussion

This case suggests that the anticholinergic side effects of phenothiazines, particularly when these drugs are combined with antiparkinsonian medications, may be effectively treated by the cholinergic bethanechol chloride. Blurred vision is a particularly distressing symptom to many patients who are taking antipsychotics with or without antiparkinsonian agents and will lead many of them to consider discontinuing the medication. I suggest that the use of bethanechol chlorid

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with tricyclic antidepressants be extended to other psychiatric medications that have pronounced, troublesome anticholinergic effects.

Caution should be exercised in the use of bethanechol, since it can produce side effects, including epigastric distress, abdominal cramps, diarrhea, and a sensation of tightness in the urinary bladder area (2). Contraindications are listed as asthma, hyperthyroidism, coronary insufficiency, and peptic ulcer (2).

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Amantadine-Induced Psychosis in a Geriatric Patient with Renal Disease

BY RICHARD L. BORISON, M.D., PH.D.

It has been widely demonstrated that agents which potentiate central dopaminergic mechanisms, such as amphetamine, are capable of producing schizophreniform psychoses (1). Similarly, drugs that ultimately act by the direct stimulation of central dopamine receptors and drugs that are most commonly used in Parkinson's disease, such as L-dopa or bromocriptine, are also capable of evoking psychotic states as relatively common side effects (2). However, drugs with less potent effects on dopamine systems may under particular circumstances also result in toxic psychotic states, as illustrated in the following case history.

Case Report

The patient was a 70-year-old woman who was admitted to a general hospital for evaluation of a sinus bradycardia. She had a history of hypertension, chronic congestive heart failure, adult onset diabetes mellitus, and chronic renal failure going back more than 10 years. There was no previous history of psychiatric illness. The patient appeared well accommodated to her illnesses: she lived alone, cared adequately for herself, and was actively engaged socially in her church organization. The patient's medications on admission were digitalis (.25 mg daily), α-methyldopa (250 mg q.i.d.), clonidine (.1 mg/day), and 20 units of NPH insulin daily.

Physical examination revealed grade II retinopathy on fundoscopy. There was no thyroidomegaly, bilateral carotid bruits were auscultated, and jugular venous distention of 4 cm was noted. The lungs were clear to auscultation and percussion. Cardiovascular examination showed a blood pressure of 160/80 mm, pulse of 56 beats/minute, and a nonradiating apical grade II/VI systolic ejection murmur. Neurological examination showed no sensory or cranial nerve deficits. The patient had a bilateral resting and intention tremor of the hands; however, there were no changes in muscle tone or pathological reflexes present. On mental status examination the patient was alert, cooperative, and fully oriented. There was no evidence of a mood or thought disorder.

Pertinent laboratory data showed that the patient was in electrolyte balance, thyroid function and blood gases were within the normal range, hemoglobin was 12.3 g, creatinine was 2.9 mg/dl, BUN was 38 mg/dl, 24-hour creatinine clearance was 16 ml/minute, and digoxin level on admission was .77 ng/ml.

The patient was continued on the medications that she was receiving at home and was also given amantadine (100 mg t.i.d.) for suspected Parkinson's disease. Within 48 hours of admission, the patient began reporting that she saw children in her room who were taunting and mimicking her and that she could see her late husband. The patient required restraints to keep her in bed because she repeatedly tried to leave to look under her bed, where she believed she could find her husband making love to another woman. These visual hallucinations were present throughout the day but occurred more frequently at night. During this time the patient remained fully oriented to person, place, and time and showed no impairment in ability to perform such tests of cognitive function as serial sevens. After 2 days of hallucinatory phenomena, the patient's amantadine was discontinued, and within the subsequent 36 hours all hallucinations ceased. The remainder of the patient's hospitalization was unremarkable.

Discussion

Amantadine was originally used as an antiviral agent but was later discovered to be efficacious in mild Parkinson's disease (3). The current belief is that amantadine's mechanism of action is either to release dopamine from storage granules or to block its presynaptic reuptake (4). Although a weak dopaminergic agent that rarely produces changes in mental status (5), it has been shown to have psychotogenic properties when used in a group of elderly patients with renal disease (6). It would thus appear that in the case presented, amantadine, which has a much prolonged half-life in geriatric patients (7), reached toxic levels in the presence of chronic renal disease, which then resulted in the patient's hallucinatory activity.

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Given that the use of weak-acting dopaminergic agents is now advocated in the treatment of early Parkinson's disease (8) and that amantadine has also been advocated for the treatment of neuroleptic-induced extrapyramidal side effects (9), this drug should be used only after careful clinical evaluation of renal function, particularly in the case of the geriatric patient.

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Sudden Death in a Patient Taking Haloperidol

BY RICHARD KETAI, M.D., JOHN MATTHEWS, M.D., AND JOHN J. MOZDZEN, JR., M.D.

To date, haloperidol has not been associated with sudden death, although this outcome has been suggested for phenothiazines. Deaths associated with phenothiazines have been attributed to hyperpyrexia (1), vasodilation and hypotension (2), and aspiration and asphyxia (3). Cardiac toxicity (4, 5) and generalized inhibitory effects on multiple brain regulatory centers have been hypothesized but have not been documented as causes of death in patients taking phenothiazines. We are reporting a case of sudden death in a young woman who received rapid tranquilization with increasing doses of haloperidol and for whom no obvious cause of death was found.

Case Report

The patient, a 35-year-old married woman with 3 children, had had no previous psychiatric problems or significant medical history. She was admitted to the acute inpatient service following a 2-month history of gradually increasing outgoing behavior, atypical of her usual reserved style, culminating 3 days before hospitalization in overtly psychotic behavior. This consisted of insomnia, religious preoccupation, agitation, and talkativeness. Two days before admission she received 200 mg of I.M. chlorpromazine in her local hospital emergency room with no effect.

At admission the patient was agitated and suspicious, unable to sit still, and had rapid speech and flight of ideas that were largely incoherent and illogical. Her affect was anxious and labile, and she was preoccupied with God and dying. Although she recognized family members, she was so disorganized and uncooperative with mental status testing that recent memory and orientation could not be accurately assessed.

Physical examination revealed no abnormalities except hyperreflexic knee jerks. She would not cooperate with fundoscopic examination or with blood drawing for laboratory screening despite repeated attempts by medical staff.

A course of rapid tranquilization with haloperidol was begun using a liquid concentrate. On the first day the patient received 10 mg every half hour up to 50 mg; on the second day of hospitalization she received 70 mg. Because she did not respond, progressive tranquilization was planned and she was given 140 mg over the next 12 hours. She had a mild dystonic reaction that responded quickly to 2 mg of benzotropine p.o., which was continued at 1 mg b.i.d. Her mental status did not change and she slept only 4 hours that night. On the fourth day of hospitalization she was as excitable as she had been at admission. She took 20 mg of haloperidol each hour for 4 hours and went to sleep. Her vital signs were normal at this time. She was checked every 15 minutes and appeared to be sleeping comfortably. Two hours after the patient fell asleep the nurse observed that she looked cyanotic. Her respiration and pulse had suddenly stopped. Immediate attempts at resuscitation by the cardiac arrest team were unsuccessful.

Autopsy findings revealed a 3-cm adenocarcinoma of the colon but no evidence of metastases. There was nonspecific mild edema of the lungs, brain, and liver; this was thought to be due to the cardiopulmonary resuscitation. Urinalysis showed that the only drugs present were haloperidol and benzotropine.

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Discussion

Neuroleptic-related death is a controversial topic because sudden deaths, even in unmedicated psychiatric patients, are sometimes unexplained. Peele and Von Loetzen (6) pointed out the existence of fatal "Bell's mania" or "lethal catatonia" before the medication era. Available investigative techniques for infectious and metabolic disturbances at that time, however, may not have been sufficient to detect an organic cause of psychosis and death. Even today, sudden unexplainable deaths occasionally occur in supposedly healthy people, e.g., the recently reported "sick sinus syndrome" (7). Patients with acute psychoses of undetermined origin could hypothetically be susceptible to sudden death reflecting neurologic dysfunction with secondary cardiac or respiratory pacing abnormalities.

Although most reviews of sudden death in phenothiazine-treated patients turn up negative autopsy findings (8) and although undetected neurologic or other unknown organic factors may be the cause of death, it is important to report sudden deaths in patients on various medications even if no cause and effect relationship is apparent. To date we are aware of no published cases related to haloperidol alone.

The manufacturer of haloperidol has abstracted cases of 12 fatalities in patients on this drug. Ten of these patients had implicated medical problems and/or were simultaneously taking other psychotropic medications, usually in large doses. Only 2 of the patients were taking haloperidol alone, a 19-year-old man who received 35 mg of haloperidol in 1 day and a 21-year-old man who received 25 mg over 4 days. Their deaths were not otherwise explained.

Our patient was being treated by rapid tranquilization in doses higher than were usually found necessary in the protocol developed by Anderson and associates (9). Daily dosages greater than 100 mg are sometimes required for refractory psychotic patients, according to the *Physician's Desk Reference*. Our experience has been doses as high as 150 mg are sometimes required to bring a psychotic episode under control, although ordinarily doses of 20–40 mg/day suffice. This patient showed no effect from 200 mg of I.M. chlorpromazine before hospitalization and did not become the slightest bit sedated on 140 mg of haloperidol the day before she died. The persistence of the psychosis and complete refractoriness to relatively high doses of medication may suggest an occult neurologic malfunction not involving dopamine pathways that would have been blocked by these drugs. Although we have no explanation for this death, there is the possibility that an idiosyncratic response to haloperidol may have occurred.

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Civil Commitment and the Rights of the Unborn

BY PAUL H. SOLOFF, M.D., STEVEN JEWELL, M.D., AND LOREN H. ROTH, M.D., M.P.H.

Clinical management of the pregnant psychotic patient is complicated by a potential conflict of interest between the treatment needs of the mother and the risks of such treatment to the unborn child. When the patient's psychosis involves a denial of pregnancy through the third trimester and poses a threat to the life of the unborn child, her right to refuse treatment comes into direct conflict with the best interests of the unborn child, which poses a difficult medicolegal problem for the physician. Although the psychiatric literature contains few guidelines for the clinical care of psychotic patients during pregnancy, recent developments in the law obligate the physician to consider the right of the unborn child "to be well born" (1, 2). In this report, we describe the resolution of this dilemma through the civil commitment of the mother for the duration of pregnancy, an unusual clinical maneuver involving a unique interpretation of commitment statutes.

Case Report

The patient, a 20-year-old single woman, was admitted to the hospital on an involuntary emergency commitment following a physical attack upon her mother during an argument. A diagnosis of schizophrenia, established during a similar admission 2 years earlier, was confirmed by the presence of a formal thought disorder and bizarre delusions in a clear sensorium. Of greatest concern was her psychotic denial of a very visible 7-month pregnancy. The patient maintained she was filled with "gas" and spent long hours in the bathroom trying to relieve her distension. She had joined a health club to lose the weight but had been expelled for diving onto her obviously pregnant abdomen from the diving board. For the 2 years before admission, she had lived at home with her mother, father, and 18-month-old daughter and had been easily managed without medication, despite her persistent psychosis. Although tolerant of the psychosis, her parents were unwilling to accept the denial of pregnancy and neglect of prenatal care. Their attempts to convince her of the pregnancy precipitated arguments that ultimately prompted her combativeness and admission to hospital.

The patient settled into the ward quite comfortably and was cooperative and easily directable. The ease of management raised the question of the appropriateness of initiating drug therapy during pregnancy. Weighing the small but present dangers of medication to the fetus in the third trimester (e.g., neonatal respiratory distress, extrapyramidal dysfunction, and transient neurologic symptoms[3]) against the ease of management in a well-structured environment, we decided to seek her commitment until delivery without active treatment of her thought disorder. The following grounds were presented in support of our request:

1. Involuntary hospitalization was needed to prevent harm to the unborn child through the mother's actions.
2. Confinement of the mother until delivery was the least restrictive alternative that could guarantee monitoring of the pregnancy and ensure a medically safe delivery for both mother and child.

Commitment was requested over the mother's objections in order to protect the child directly and the mother indirectly. Commitment was granted, extended, and sustained in a formal appeal before the Allegheny County Court of Common Pleas. From the 38th week of gestation, the patient was examined every 4 hours for the start of labor. The possibility of inducing labor to ensure a medically controlled delivery was considered. However, this would have required further adversary hearings to prove the patient incompetent and appoint a guardian. The more conservative approach of frequent examination was urged by obstetrical consultants. On her 81st hospital day, the patient requested sanitary napkins from the staff, stating she had finally resumed her menstrual periods. She was found to have ruptured membranes and was in active labor. She was rushed to a nearby women's hospital and delivered a healthy male child within the hour. The newborn infant, finally a person within his own right, was taken into the custody of the Child Welfare Department, initiating yet another round of adversary hearings in which the mother, recognizing the reality of the birth, sought custody of the child she had denied for 9 months.

The patient was returned to the psychiatric hospital following postpartum care and was started on perphenazine, 24–64 mg/day for 1 month, with no remission of her thought disorder. Three months after discharge, she has shown no clinical response to perphenazine, maintained at 32 mg/day.

Discussion

Commitment in this case was granted to afford protection to an unborn child over the protest of the mother. In so doing, the court upheld the welfare of the unborn as if it were already a "person-in-being," with rights to protection under the broad principle of *parens patriae* (the state's interest in protecting persons unable to protect themselves).

Despite the rigorous intent of Pennsylvania's commitment statutes, which require 1) evidence of danger to self or others in the preceding 30 days, and 2) a high probability of recurrence of danger in the next 30 days if treatment is refused, judicial interpretation has proven flexible in the face of clinical or social necessity. A review of the legal status of the unborn suggests that this case represents an interpretation of the commitment statute that is both unique and consonant with the growing medical and social concern for protection of the unborn.

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The Rights of the Unborn

The unborn child has been treated with great inconsistency in the different branches of law (4). In *Roe v. Wade* (5), the Supreme Court upheld the mother's "right to privacy"—to obtain an abortion—over the state's interest in preserving the life of the fetus during the first 6 months of pregnancy. Justice Blackmun held that the word "person," as used in the 14th Amendment, did not include the unborn. However, the same decision recognized the interest of the state in the unborn child after the sixth month, maintaining a basis for protection under the doctrine of *parens patriae*.

Under this broad doctrine, conflicts of interest between mother and unborn child have been heard in cases involving the religious objections of the mother to medical treatment for the child. In *Hoener v. Bertinato* (6), the court awarded custody of a child in utero to a welfare department for the purpose of administering a needed blood transfusion at the time of birth over the religious objections of both parents. Similarly, in *Raleigh Fitkin-Paul Morgan Memorial v. Anderson* (7), the court compelled a Jehovah's witness to submit to blood transfusion to protect the life of an unborn child. The child's right to life was placed above the parent's right to free practice of religion.

Reviewing the fate of the unborn child in mothers addicted to opiates, Densen-Gerber and associates advocated the use of civil commitment for treatment and detoxification of the mother, recognizing the unborn child as a person-in-being with a "right to be well born" (8). Commitment of the pregnant psychotic patient described here represents an affirmation of this principle by the court and a clinical use of the statute to safeguard both mother and child to term.

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The Effect of Lithium Treatment on Thyroid Function in Patients with Primary Affective Disorder

BY JUN TAE CHO, M.D., STANLEY BONE, M.D., DAVID L. DUNNER, M.D., EDWARD COLT, M.D., AND RONALD R. FIEVE, M.D.

The purpose of this study was to investigate the incidence of clinically significant thyroid gland dysfunction among a group of affectively ill patients who were attending a lithium clinic. Isolated cases of goiter during treatment with lithium carbonate were first reported by Danish and Swedish investigators in 1967, and the thyroid effects of lithium have been reviewed extensively by Shopsin (1). Three major thyroid effects of lithium treatment have been noted: asymptomatic thyroid gland enlargement, clinically significant hypothyroidism, and a few cases of thyrotoxicosis. Although these effects of lithium on the thyroid gland have long been demonstrated, the incidence of lithium-related thyroid dysfunction has not been systematically reported from large lithium treatment centers.

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Method

The study was conducted in the Lithium Clinic of the New York State Psychiatric Institute. All patients had primary affective disorder (2) and were further classified into bipolar and unipolar subtypes (3, 4). Patients who had four or more affective episodes yearly were separately noted as rapid cyclers (5). All subjects participated in the study on a voluntary basis.

Patients were routinely asked about their use of medication, including lithium, other psychiatric medi-

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cation, and drugs for medical indications. All patients who were taking supplemental thyroid hormone were studied regarding the medical indication for use of thyroid medication, duration of thyroid treatment, and history of thyroid disease before initiation of lithium treatment. These questions were also asked of a comparison group of patients who were evaluated before lithium treatment or who were relatives of index patients and had a diagnosis of primary affective disorder but were not taking lithium.

Results

The clinic sample consisted of 195 patients (80 men and 115 women). Twelve of these patients (1 man and 11 women) had thyroid disease and were taking thyroid medication in addition to lithium. Five patients (1 man and 4 women) had started thyroid treatment before they were given lithium. Thus, 7 women (6.4% of the lithium-treated women) developed clinically significant hypothyroidism after they started taking lithium.

Further investigation revealed that 5 of these 7 postlithium thyroid patients were rapid cyclers. Only 16 of the 115 women attending the clinic were rapid cyclers, so the incidence of postlithium thyroid medication use was significantly higher among rapid-cycling women than non-rapid-cycling women (31.2% versus 2.1%, $\chi^2=7.64$, $p<.01$). Excluding rapid cyclers, thyroid medication was used by 4 women before lithium treatment and by 2 other women subsequent to treatment.

The comparison group consisted of 39 women and 23 men who had primary affective disorder and no history of lithium treatment. None of the men and 2 of the women were taking thyroid. Of the 3 women who were rapid cyclers, none was taking thyroid. Thus, the incidence of thyroid use among these non-rapid-cycling women in the comparison group (5.6%) is quite similar to that among non-rapid-cycling women patients before lithium treatment (5.1%).

No cases of thyrotoxicosis subsequent to lithium treatment were reported by these patients. All instances of hypothyroidism had been documented by laboratory studies of thyroid function.

Discussion

Our data suggest that there may be a subgroup of patients—women with high frequencies of affective episodes—who are particularly prone to develop hypothyroidism while they are taking lithium carbonate. Other patients do not appear to have increased incidences of hypothyroidism secondary to lithium of sufficient clinical significance to warrant treatment with additional thyroid medication. It appears that about 5% of affectively ill women are taking thyroid before lithium treatment is started and that a few additional instances of hypothyroidism occur during ensuing years of lithium treatment. A third finding from our study is that the thyroid dysfunction was primarily limited to women.

The data from this study are presented because they represent a large number of patients and they may be useful background material for other clinicians as well as investigators from other large lithium research centers. We are particularly interested in data from other centers regarding the incidence of thyroid medication use among lithium-treated rapid-cycling women. Confirmation of our findings may lead to fruitful research regarding the difficulties in treating such patients (5).

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Orofacial Movement Disorder: Tic or Tardive Dyskinesia?

BY BENNETT L. LEVENTHAL, M.D., AND MEREDITH ALDEN, PH.D.

Orofacial dyskinesias are associated with a variety of physical and psychological conditions. Such cases present the clinician with the potential for an expansive differential diagnosis. For patients who are taking psychotropic medications, much concern focuses on tardive dyskinesia (1-3). However, it is essential that patients with movement disorders be assessed carefully for possible etiologies other than tardive dyskinesia before treatment is initiated.

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Case Report

A 48-year-old divorced woman had worked as a reservations clerk before her admission to a nearby psychiatric

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hospital in September 1977. At admission she had masked facies, a "drugged appearance," fine tremor, parkinsonian gait, and vegetative signs of depression that had been progressing since the sudden, unexplained departure of her boyfriend of several years. Evaluation revealed a normal EEG and CT scan and a full-scale I.Q. of 83 on the Wechsler Adult Intelligence Scale. The patient denied recent alcohol or drug use. She was diagnosed as having depressive neurosis, passive-dependent personality, and parkinsonism. During her hospitalization she was treated with imipramine, benztropine, and thioridazine. After returning from a 10-day stay at home, she was found to have an elevated tricyclic antidepressant plasma level (imipramine plus desipramine = 540 ng/ml). She failed to make significant clinical improvement and was transferred to Duke University Medical Center in late November 1977. All medications were discontinued and neurologic evaluation was negative except for a diffusely abnormal EEG. The patient had no parkinsonian symptoms but appeared to be severely depressed and had orofacial movements characterized by darting tongue movements, lip-pursing, chin quivering, and bucco-mandibular masticatory movements. Based on her age and history, the patient was given presumptive diagnoses of tardive dyskinesia and depression and was started on 600 mg/day of deanol and 6 g/day of L-tryptophan for the respective problems. By mid-January, her affect and movement disorder were unchanged. However, there was improvement in the EEG.

The patient was only hesitantly able to engage in psychotherapy. On occasion, she would wring her hands and cry, without explanation. These episodes were immediately preceded by increases in the orofacial movements. Psychotherapeutic efforts led her to speak about her disappointments in life, which centered around unmet dependency needs and, in particular, her frequently absent mother. As the patient recalled and worked through the rage she felt toward her mother, the orofacial movements and hand wringing diminished. When this was observed, she seemed relieved to report a maternal prohibition against crying and other expressions of affect. She remembered that when she was a child her chin would quiver as she fought back tears and expression of anger toward her mother.

Her abnormal orofacial movements had ceased by mid-February, despite the discontinuation of deanol and the use of doxepin (at therapeutic plasma levels) to control a persistent depression. However, the abnormal movement returned when she prepared to go home. No medications or dosages had been changed and her neurologic status was also unchanged. Remission occured rapidly with review of the previous psychotherapeutic work. By the time of discharge her I.Q. scores and EEG were well within the normal range and she was asymptomatic for movement disorder and depression.

Discussion

The differential diagnosis of orofacial dyskinesias includes basal ganglion disease, metabolic disease, post encephalitic syndromes, schizophrenic stereotypies, and drug-induced conditions related to agents such as L-dopa, neuroleptics, and anticholinergics (1). Of significance in this case are recent reports of oral dyskinesia associated with imipramine treatment (4, 5). Because of the initial encephalopathy, it could be argued that the patient's movement disorder was not psychogenic. However, the resolution of the movement disorder did not parallel the resolution of the encephalopathy, and there was no residual neurologic deficit. In addition the brief exacerbation and rapid remission did not correlate with any changes in the patient's neurologic status, but the remission closely paralleled the patient's progress in psychotherapy.

There is compelling evidence that this patient had a psychogenic tic rather than a neurologic disorder. Psychogenic tics can present as a variety of movement disorders. They are usually seen as intermittent, sudden, repetitive movements that occur without external stimuli. Tics can become automatic behaviors that are modifiable by treatment. However, even in the most successfully treated case, the tic may recur when the patient is under significant stress (6). Ferenczi (7) suggested that many tics are developed as means to avoid suffering and that they are often difficult to differentiate from compulsive acts. Abraham (8) agreed that tics may not be distinguishable from compulsive acts, but he pointed out that the patient with a tic may not attribute psychological significance to the tic and does not fear disaster if the compulsive act is omitted. Like the compulsive act, the tic may involve a hostile as well as an erotic component. The tic then represents an attempt to adapt to an "instinctual conflict" (9). In the case we have described, the patient experienced a conflict between the drives for love and dependency and the expression of hostile aggressive impulses toward her mother and others who frustrated these drives. The additional frustration of the maternal prohibition of expression of affect led the patient to adopt a motor response to ward off this conflict. This patient came to substitute the small muscle activity for other expressions of affect and aggression secondary to the fear of punishment or loss of love. This motor response had been present since childhood, and the loss of her boyfriend had intensified these fears, precipitating her depression and exacerbating the tic. In psychotherapy the patient began to work through these conflicts and was thus able to give up her tic and use more effective adaptive mechanisms.

Treatment of movement disorders varies greatly with diagnosis. The literature on therapy for tardive dyskinesia is, thus far, controversial. Tics are sometimes successfully treated with haloperidol; this patient responded to insight-oriented psychotherapy. Because treatment for one movement disorder may worsen another, it is essential that each patient be evaluated carefully before therapy is started. With symptomatically similar disorders requiring significantly different treatments, treatment must be prescribed on the basis of diagnosis rather than clinical presentation.

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Case Report: Symptomatic Mania and Phencyclidine Abuse

BY ARNOLD ROSEN, M.D.

The increasing prevalence of phencyclidine abuse, with its psychiatric consequences, has become a challenge to the clinical psychiatrist. The pharmacology of phencyclidine and its associated toxic mental conditions were recently reviewed by Showalter and Thornton (1). In 1977, Slavney and associates (2) reported two cases of symptomatic manic states following use of phencyclidine (commonly called PCP or angel dust). They emphasized the following points:

1. The manic-like psychosis took place in clear consciousness.
2. Symptomatology met research criteria proposed by Feighner and associates (3) and by Abrams and Taylor (4).
3. Symptoms persisted longer then would be expected on the basis of the drug's half-life.
4. Patients had no prior psychiatric history.
5. Patients had no family history of affective illness.

In addition, their patients' psychoses responded minimally to neuroleptics but resolved when lithium carbonate treatment was instituted. The following case report describes a post-PCP symptomatic manic episode that began to respond only after ECT.

Case Report

The patient, an 18-year-old man with no psychiatric history, came from an intact middle-class family that did not have a history of affective illness. The patient was outgoing, played on the school basketball team, and was a below average to average student. He had experimented with many street drugs 3 months before his admission: cocaine, opium, PCP, and marijuana. His psychosis began after he smoked PCP 1 week before his admission. His family had noted an immediate change in his behavior. He initially seemed withdrawn and had difficulty concentrating, but after a few days he became agitated, with hyperactivity, insomnia, thought disorganization, and paranoid delusions. He was hospitalized and required increasing doses of haloperidol (up to 60 mg/day) for 4 days before his agitation subsided. He then developed a symptom picture indistinguishable from catatonic schizophrenia: catalepsy, posturing, mutism, negativism, and stupor. On the eighth hospital day, this symptom picture abruptly ended with a burst of hyperactivity, mild euphoria, and rapid pressured speech. Two days later he calmed down and seemed coherent, logical, and relevant for the first time since his admission. The remaining 2 weeks of hospitalization were uneventful, and haloperidol was reduced to maintainence levels (5 mg/day).

Four days after discharge, the patient was readmitted in an acute psychotic state after he again used PCP. He was unable to be maintained out of seclusion—despite haloperidol in doses of 100 mg/day—because of grossly inappropriate and unpredictable behavior, which occasionally became hostile and assaultive. He was illogical, with idiosyncratic austistic thought, had visual and auditory hallucinations, and constantly denuded himself. He was maintained on high doses of neuroleptics, and this symptom picture gradually changed over the next 4 weeks, with affective elements coming into prominence. He became excessively hyperactive, with insomnia, grandiose delusional thought, euphoria, and a high-spirited affect that contained a genuinely humorous clowning, imitative aspect. Lithium carbonate was tried during this period, but oral administration was unreliable and serum lithium levels could not be obtained because of the patient's inability to cooperate with the treatment. All medications were stopped and he was given a course of bilateral ECT. He responded dramatically midway through a course of six treatments and lost essentially all of his psychotic symptomatology. He was maintained off medication and observed. After a week and a half he relapsed suddenly, becoming agitated, hyperactive, volatile, and irritable (although generally euphoric), with profuse illogical thought and inappropriate jokes and puns. He was given another short course of ECT and subsequently started on lithium carbonate (1800 mg/day). He again responded dramatically, becoming symptom-free, and was able to cooperate in the administration of lithium therapy.

The patient has been followed now for 8 months. He has continued to cooperate with lithium carbonate therapy. Clinical observation and reports from the family both indicate no recurrence of his psychosis. The patient was able to take a summer job and planned to return to school in the fall.

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Discussion

One great difficulty in the management of this patient's illness was diagnosis. He had developed a severe, prolonged psychosis after PCP abuse that was refractory to high doses of various neuroleptics but responded dramatically to ECT. However, he did not go into remission until lithium carbonate was started. His symptom picture was florid and went through many phases (including catatonia) but eventually appeared to be a severe manic episode that met research criteria proposed by Feighner and associates and Abrams and Taylor (4). Abrams and Taylor (5) have shown that catatonic signs and symptoms are most often a component of an affective illness and should not be equated with a schizophrenic disorder. The pattern of this patient's dramatic response to ECT also resembled that of an affective illness (6). Finally, the patient's lack of residual symptomatology suggests an affective illness.

Slavney and associates (2) retrospectively reviewed post-PCP psychosis and found an association with manic illness but not with schizophrenia. PCP abuse is increasing, and it is important to recognize symptomatic mania as a possible consequence. If these manic episodes are in fact "symptomatic" and causally related to PCP toxicity, one would expect this condition to be most prevalent in adolescents and young adults (in whom one does not usually expect to see an affective illness). Because the reported patients have shown a poor response to neuroleptics, the importance of a correct diagnosis is crucial.

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IN MEMORIAM

Edwin F. Gildea
1898–1977

WASHINGTON UNIVERSITY in St. Louis, standing astride the confluence of the Mississippi and Missouri Rivers, has commanded an influential position in directing the currents of American psychiatry. After the second world war psychiatry became a contemplative field, heavily invested in the subjective and individual response of patients and the complex interpretation of motives and behaviors. Many schools burgeoned—sometimes with overlapping, sometimes with diverging viewpoints, but all mainly devoted to the attempt to understand depth psychology. During this time the Washington University Department of Psychiatry flirted with this approach but gradually settled into a strong alliance with biology, a medical view of psychiatric illness, and heavy reliance on reproducible data.

Why did this aberrant development take place in St. Louis? Why did Washington University not follow the streams that were common to most other American medical schools? To a large extent the responsibility and honor belong to Edwin F. Gildea, a person with tolerance and uncommon intelligence, who died on July 19, 1977.

I first met Ed Gildea in July 1950, when I went to St. Louis for a third year of residency training in psychiatry. Dr. Gildea had been there as department head since 1942, having succeeded Dr. John Whitehorn, who had moved to Johns Hopkins. The department at that time was somewhat unstable, with a number of people coming for short periods while they were getting started in private practice and then leaving. Psychoanalysis was heavily represented by a number of people who had clinical appointments and did a good bit of teaching of residents. The psychosomatic service was relatively strong and stable under George Saslow. Electroencephalography and electrophysiological research were conducted by George Ulett. About 1949 a young man had come from Boston to join the department because of Ed Gildea's excellent reputation as a leading biological psychiatrist; that was Eli Robins. A mainstay of the department was Dr. Margaret Gildea, Ed's wife, who combined a very practical attitude about psychiatric treatment with a dynamic viewpoint. Those of us in the residency program considered her a particularly bright, attractive, and sophisticated person.

Ed himself was a large man, with the pinkish complexion that one often sees on the streets of Dublin. He smoked huge pipes that he stuffed with heavily aromatic tobacco. He had originally come from Colorado and still liked the western way of life. It was rumored that frequently when he had to take a train trip he would settle down in the club car with a wide-brimmed hat and pass himself off as a cattle rancher.

He had graduated from Harvard Medical School in 1924, interned at the Boston City Hopsital between 1924 and 1926, and was a resident at the Boston Psychopathic Hospital from 1926 to 1928 in psychiatry and neurology. He also had postgraduate training in neurology and neuropathology between 1928 and 1929 at Harvard. He joined the faculty at Yale University in 1930 and stayed there 12 years until he became the Wallace Renard Professor of Psychiatry and Head of Psychiatry and Neurology at Washington University. He remained in that position from 1942 to 1966, afterwards becoming an emeritus professor.

At all times Ed Gildea relied heavily on data. Prior to going to St. Louis, I had written a paper on a rationale for psychotherapy and anxiety, obsession, and depression. I was particularly proud of the paper because it was almost incomprehensible, something that I thought made me a distant intellectual relative of Harry Stack Sullivan. I presented the paper to Ed Gildea, who read it carefully. He told me to go ahead and publish it and then added something like, "It doesn't seem to have much data in it, does it?" I did publish the paper but in retrospect I believe I should have listened to him more carefully.

Dr. Ed had a quiet, sandy voice and a "homey" attitude, which, however, masked considerable sophistication and erudition. He seemed knowledgeable about everything of importance that had gone on in the field of psychiatry. He had seen hundreds of patients and was an excellent attending man. Residents whom he taught loved to recall his reminiscences about his father, a general practitioner in the west, who used to take care of Teddy Roosevelt when the latter would make trips to the west in the midst of significant mood changes. He also enjoyed telling about his mother, who, he said, was one of the original "anxiety neurotic" patients studied by Mandel Cohen and Paul Dudley White. He said that when the well-to-do ladies of Colorado heard that Dr. White had a new machine (the electrocardiogram) which told all about their heart they rushed to Boston to be worked up. His mother was one of them, and she received the diagnosis of anxiety neurosis. Years after meeting him I asked what had happened to his mother's anxiety neurosis (she was in her mid-90s at that time). He told me, with some amusement, that she still had it. He always did have a feeling for follow-up studies!

In 1952 I left St. Louis for a stint in the Air Force after having spent one year as a faculty member at Washington University. I recall saying to Dr. Ed when I returned in 1954 that I was certainly pleased that I had enough to do, since I had hardly been overworked in 1951–52, my first year on the staff. In fact, I noted that I had spent a good part of the lunch period sleeping on an analytic-type couch that had been provided for me. He seemed pleased that I was working a little harder, did not seem concerned that I had not been particularly productive a couple of years before, and said, "I thought you were thinking all that time."

Drs. Ed and Margaret Gildea did a very good job of raising

morale in the department. Every summer they invited the faculty and residents to their farm in Potosi, Mo. This was usually a hot and buggy occasion but was highly regarded by most concerned; Ed always was amused at the fact that the martini drinkers looked on it as a necessary kind of suffering. Every Christmas the Gildeas also held a cocktail party. In 1950, in addition to personal friends, residents, and faculty, five Nobel prize winners were in attendance. It was the big world.

Ed Gildea was one of the early people to emphasize biochemistry in psychiatry and had countless important publications in this area to his name. It was said that he had carried the available methodology for biochemical investigations in psychiatry as far as it was possible to go at the time. He studied serum proteins and lipid changes in schizophrenic and manic-depressive patients. He did early studies on the thyroid and on ACTH and cortisone in the psychoses; in his later years he wrote an important clinical paper about the differentiation of schizophrenia from schizophreniform psychosis.

It was under Ed Gildea's aegis that the psychiatry service at Barnes Hospital and Washington University became integrated for blacks. What is particularly important is that he supported this integration because he thought it was right, not because he was under any pressure to do so.

What is written above is an attempt to give a picture of Ed Gildea as a person and as a scientist. But his contributions transcend those points. Had it not been for his leadership of the Department of Psychiatry at Washington University, it is doubtful that the department would have developed in the way it did. He showed favoritism toward no one; toward all he expressed a quiet but insistent demand for evidence. What he did was provide a field on which intellectual conflict could occur. While he attempted to keep people from becoming personally injured, it was clear that he enjoyed the give-and-take of different ideas. As time went on, a biological viewpoint emerged more and more strongly. Heavy reliance on hard-nosed clinical diagnosis became apparent, while theorizing, adherence to universal theories, and dynamic interpretation became less evident. Nevertheless, he continued to give the more subjectively oriented psychiatrists opportunities to function and in particular set aside for them the area of child psychiatry. The department became tougher minded as a natural consequence of the vigorous interaction between various factions. As a result Washington University has influenced research and clinical activities and has achieved a unique role in American psychiatry.

In Gray's "Elegy" there is a line, "The paths of glory lead but to the grave." This is true, but to this we should add that in the case of Ed Gildea, the substance may fade but the reflections live on.

GEORGE WINOKUR, M.D.

LETTERS TO THE EDITOR

This section contains reactions to Journal *articles, statements of opinion, comments on Association activities, requests for information, etc. Letters should be submitted in duplicate to the Editor, who makes all decisions regarding publication. Letters must be typed double spaced throughout and should not contain more than 500 words and 5 pertinent references. Criticisms of published articles will automatically be sent to the author(s) for response. Letters will be edited for clarity and conformance with* Journal *style. We regret that we cannot inform writers of the disposition of letters or return those which are not printed.*

More on Klinefelter's Syndrome and Affective Illness

SIR: Stanley N. Caroff, M.D. ("Klinefelter's Syndrome and Bipolar Affective Illness: A Case Report," June 1978 issue) was correct in assuming that the second patient reported in our paper (see reference 1) should have been diagnosed as manic-depressive, not only because of the features Dr. Caroff quoted but also because of the patient's grandiose plans. However, the patient had been referred by another psychiatrist, and we did not feel free to comment on the diagnoses, especially since we were primarily interested in the patient's pedophilic propensities. Dr. Caroff mentioned that reports of patients with Klinefelter's syndrome and bipolar illness are rare; I would like to report another such case.

Mr. A, a 49-year-old engineering technician, sought help in August 1970 for irritability, overactivity, crying spells, biparietal headaches, early waking, and worry that had persisted for several months. He had no other psychiatric history. He described frequent clashes with his three adopted children and said his sexual interest had been decreasing for the previous 3–4 years.

He was an only child, and although he was quiet he got along well at school and with friends. He completed 12th grade. He had had several girlfriends but said he had never been at ease with girls. He married at 26 because he thought he should settle down. Neither he nor his wife knew, despite investigations, why they had not been able to have children.

Mr. A had some bronchospasm and very small testes. He was a big man (6′2½″) with long legs, a very broad pelvis, and a short trunk. He spoke loudly and emphatically, swearing often, demanding attention, and showing flight of ideas. He broke into tears when his family was mentioned. These crying spells annoyed him, and his mood would shift from short episodes of apparent sadness to a prevailing anger and irritability. Mr. A had no delusions or hallucinations, a clear sensorium, and intelligence estimated as average (his score on the Wechsler Adult Intelligence Scale 3 years later was 102).

Skull films, EEG, brain scan, and visual fields were all normal. A buccal smear was positive for sex chromatin, and Barr bodies were seen in 20% of cells. The diagnosis was manic-depressive disorder, hypomanic phase, and Mr. A was treated successfully with thioridazine and follow-up clinic visits.

In May 1971 he returned to the hospital complaining of weakness, fatigue, crying, early waking, poor appetite, depressed mood, and thoughts of suicide. He was diagnosed as having manic-depressive disorder, depressed phase, and successfully treated with imipramine. In September 1972 he suffered another depression, which again was successfully treated with imipramine and follow-up visits in the clinic. He was discharged from the clinic as well in June 1974.

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Correcting the Underreporting of Father-Son Incest

SIR: I am glad that Katharine N. Dixon, M.D., and associates wrote their paper on "Father-Son Incest: Underreported Psychiatric Problem?" (July 1978 issue). During the last five years, I have come across four cases of father-son incest, three of which were reported by patients who are now adults aged 20–25.

John had been used sexually by his father from the time he was 8 until his father's suicide while psychotic when John was 13. The almost daily activity involved masturbation and fellatio performed upon the boy, whose reaction was a mixture of pleasure, excitement, and shameful disgust. Another patient, Sam, could reveal to his therapist only by letter that his father had used him sexually from the time he was an early adolescent. Dave had sexually molested a 6-year-old boy. He had a history of pedophilic homosexual fantasies and behavior. During early adolescence, Dave had reported on a number of occasions that his physician father had slept with him and had used him sexually. Another patient, a 4-year-old boy, had a history of having his genitals fondled, sucked, and burned by his father, who also required Richard's 7-year-old half-sister to perform sexual acts on Richard.

Two of these patients are from families in upper socioeconomic groups. The adult victims were all quite disturbed and had perversions and borderline personality organizations. Paranoid trends and repetition compulsions to act sexually with either an older man or a male child were also found. The victims' reluctance, even in therapy, to reveal the history of incest was remarkable. Caprio (1, p. 104) found this reluctance in a case of father-son incest that he treated.

The standard texts on sexual perversions and behavior ignore father-son incest. However, in reviewing the literature I have been able to find at least 33 reported cases of father-son incest including 18 cases reported in one German court (2). In one case a physician was sexually active with his three young sons (3).

Now that child abuse is a national issue, I suspect we will hear about more of these cases. However, one aspect of this problem that may be overlooked is the adult victims of this incest, who tend to be reluctant about revealing the pertinent history.

In *Ulysses* James Joyce states that a father and his son

> are sundered by a bodily shame so steadfast that the criminal annals of the world stained with all other incests and bestealities hardly record its breach. Sons with mothers, sires with daughters, lesbic sisters, loves that dare not speak their name, nephews with grandmothers, jailbirds with keyholes, queens with prize bulls. (4, p. 207)

In recent years, the breach of this "bodily shame" is becoming more apparent and recorded.

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Contradictions in Survey Results

SIR: I would like to point out some contradictory aspects in the findings reported in "Focused Psychiatric Curriculum Selection: Student, Psychiatrist, and Nonpsychiatrist Physician Expectations" (October 1977 issue) by Warner Johnson, M.D., and associates. Out of a list of 21 commonly taught psychiatric topics, the survey showed the 5 most important topics to be 1) the ability to talk with patients about their personal problems, 2) an understanding of the emotional aspects of the chronically ill or dying patient, 3) familiarity with the dynamics of the doctor-patient relationship, 4) an awareness of when and how to refer a patient to a psychiatrist, and 5) the ability to evaluate a patient's potential for suicidal or destructive behavior or need for psychiatric hospitalization. Among the 5 least important topics to learn, the survey showed that "an understanding of the principles of psychoanalytic theory" is the least important. The contradiction is in how the top 5 topics can be taught and be understood without referring to psychoanalytic concepts, such as defense mechanisms in symptom formation, transference reaction in the doctor-patient relationship, identification in suicides, dependence and regression in chronically ill and dying patients, etc.

The second point is that the ability to do brief psychotherapy as a topic is ranked as sixth from the bottom in importance. Again, this contradicts the choice for a familiarity with the dynamics of the doctor-patient relationship. I tend to believe that the familiarity with the doctor-patient relationship and ability to do psychotherapy are very much interrelated. To carry the argument further, the ability to understand the emotional aspects of patients requires many activities from physicians, and such activities involve the basic quality of being a physician. These activities include listening, giving support, reassurance, and advice, empathic understanding, and even at times confrontation. All these are basic psychotherapeutic interventions. A good doctor-patient relationship is the basis for a good psychotherapeutic relationship. The moment a physician intervenes, be it in giving information, advice, or a prescription, he or she is doing psychotherapy.

I seriously question the validity of these opinions, and would like the authors to clarify these issues.

CHRIS YUNG, M.D.
El Paso, Tex.

Dr. Johnson and Associates Reply

SIR: Dr. Yung raises two interesting points about some apparent contradictions in the findings of the survey. In essence, he asks how dynamic topics such as interviewing skills, the doctor-patient relationship, and others could be selected for teaching, while concepts of psychoanalytic theory would be excluded. We do not have any factual information as to why the survey respondents made their selections but are willing to offer speculations. We suspect that the ranking of topics was based upon which ones were perceived as interesting, needed, or aversive, rather than whether they would constitute a logical sequence for a curriculum. In other words, topics were selected according to what the respondents wanted or did not want to learn. But whatever reasons lay behind their choices, formal course work in psychoanalytic theory consistently received a low priority rating by students, primary care physicians, and psychiatrists alike.

We do not agree with the premise that education in psychoanalytic theory is necessarily a prerequisite for teaching interviewing skills, the doctor-patient relationship, or other dynamic topics. At the University of Southern California School of Medicine, we teach psychodynamic concepts as they are applied to medicine without specific reference to complex speculative theories, and we rarely find the need to introduce technical psychiatric jargon. Psychoanalytic theory, along with mental retardation and behavioral modification, are topics which consistently received a low priority rating by the survey respondents. As we suggested in our article, if these topics are to be taught, it would be well for educators to take into account the low receptivity of students for these topics. Special efforts by the instructor may be needed to make the topics more interesting or relevant, if they are not to be quickly forgotten by the students.

Dr. Yung's second point is concerned with the seeming contradiction in that the survey respondents gave a low rating to learning psychotherapy while ranking an understanding of the dynamics of the doctor-patient relationship as a relatively high-priority item. He raises the question of how one can learn about interventions that are psychotherapeutic and yet exclude teaching about the principles of psychotherapy. Again, we have no hard data on the subject, but we suspect that the respondents saw no such contradiction. Formal instruction in brief psychotherapy would be perceived as

belonging more to the domain of a professional psychotherapist and therefore less as a concern for a primary care physician, while managing the doctor-patient relationship would be something that would be important for all physicians. Furthermore, we feel that it is better to define "psychotherapy" in terms of its being a distinct treatment discipline. Although psychotherapy includes many interventions and methods which are psychotherapeutic, not all that which is psychotherapeutic necessarily is part of psychotherapy. Therefore we feel that the terms cannot be used interchangeably.

We appreciate Dr. Yung's comments and affirm again our position that psychiatric educators must be aware of changing "consumer" preferences with regard to which aspects of psychiatry they are interested in being taught. We feel that it is important to set a priority of teaching topics and also to consider the expectations of the learners for any curriculum that is to be taught.

We welcome further comments on this matter.

C. WARNER JOHNSON, M.D.
JOHN SNIBBE, PH.D.
KAAREN HOFFMAN, PH.D.
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Los Angeles, Calif.

Tricyclic Antidepressants, Anxiety, Thromboxane A2, and Dipyridamole

SIR: In their article "Successful Treatment of Severe Anxiety Attacks with Tricyclic Antidepressants: A Potential Mechanism of Action" (July 1978 issue), Kenneth Jobson, M.D., Markku Linnoila, M.D., Ph.D., John Gillam, M.D., and associates suggested that low doses of tricyclic antidepressants were effective in treating anxiety attacks. The plasma levels of drug achieved were probably insufficient to modify amine uptake or to block alpha or beta receptors, and the authors suggested that inhibition of monoamine oxidase might be the relevant mechanism.

I have an alternative suggestion. My group has recently shown that tricyclic antidepressants can inhibit intracellular calcium release in muscle with a threshold of about 1 ng/ml (1). We have shown that benzodiazepines can also block intracellular calcium release at concentrations readily achieved in human plasma (2). The effect is consistent with the idea that the drugs are interfering with the action of a prostaglandin-related substance, thromboxane A2 (TXA2), which seems required for intracellular calcium release (3).

This raises the possibility that anxiety might be related to excess TXA2 formation. If so, then the inhibition of TXA2 synthesis should relieve anxiety. Dipyridamole, an antiplatelet aggregating agent, has actions consistent with inhibition of TXA2 synthesis. I have recently treated two persons who in the past had repeatedly had acute anxiety attacks after stopping smoking. The attacks were so severe that they had always started smoking again. The two patients were given 12.5 mg dipyridamole q.i.d. starting at the time of the last cigarette. No anxiety attacks occurred until the third day, when trial withdrawal of the drug led to panic attacks in both patients 8–10 hours after the last dipyridamole dose. Those attacks were terminated within one hour of the patients' taking dipyridamole again. After three weeks with no smoking dipyridamole was successfully withdrawn from both persons without any resultant panic attacks.

My associates and I have recently provided evidence that oxyheme compounds may enhance TXA2 synthesis (4). In smokers in whom carbon monoxide inactivates 10%–25% of the oxygen-carrying capacity of the blood, this oxyheme stimulation is reduced, but TXA2 levels may be maintained at normal values by other mechanisms. When smoking stops, the removal of carboxyhemoglobin leads to a rebound overproduction of TXA2 and anxiety. This phenomenon could account for the anxiety states that often occur in patients with polycythemia (even before diagnosis) and that in some may be so severe as to deserve an eponymous name, Gaisbock's syndrome.

This proposal that antianxiety and antidepressant agents may act by modifying thromboxane- and prostaglandin-regulated calcium movements opens up a completely new approach to the mechanism of action of these drugs.

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Effects of Psychoactive Drugs on Ejaculation

SIR: Psychoactive medications can cause nonerection, nonejaculation, and retrograde ejaculation. Retrograde ejaculation, described by James E. Nininger, M.D., in "Inhibition of Ejaculation by Amitriptyline" (June 1978 issue), is probably the least common. The incidence of these effects is highly idiosyncratic, as Kotin and associates (1) showed in the case of thioridazine. In a considerable but unquantified proportion of patients receiving tricyclics or the MAO inhibitor phenelzine, ejaculation is delayed or inhibited. The threshold for the ejaculatory reflex appears to be raised, since many individuals ejaculate on masturbation but not on intercourse.

The erectile mechanism involves central, parasympathetic, sympathetic, and vascular elements, all of which are potentially altered by tricyclics. It is not usually affected by beta-blocking agents, although one case of apparently pharmacogenic impotence was reported for propranolol (2). Idiosyncrasy in response is therefore difficult to analyze. Much could be learned about erectile physiology by careful history taking.

Delayed ejaculation can be distinguished from retrograde ejaculation by the criteria Nininger used—the sensation of orgasm is absent or much postponed in the former. Whether postponement or nonejaculation occurs will depend on 1) age—nonejaculation in perhaps two of three acts of intercourse is common in men over 50 who have frequent intercourse, and 2) sexual pattern—some men respond to minor stresses (fatigue, infection) with impotence and others with nonejaculation. These apparently consistent intraindividual

patterns should be inquired about in assessments of possible drug effects.

The ejaculatory effects of tricyclics tend to appear early in treatment, before there is increased libido associated with improvement, and to decline with time. Similar effects with phenelzine appear after 4–5 days and disappear rapidly on withdrawal of the drug. The decreased libido reported by Friedman and associates (3) in subjects treated for neurodermatitis with phenelzine is unusual; it may result from delayed or altered ejaculatory sensation. In prostatectomy it is not uncommon for the unprepared patient who experiences alteration of ejaculatory sensation postoperatively to become secondarily impotent because of anxiety over the change. The effects of phenelzine on ejaculation are probably local actions of the drug; they are not synchronous with MAO inhibition.

The most likely site for these effects is preorgasmic glans tumescence. In men who complain of absent or delayed orgasm the glans is flaccid even when erection is complete. In premature ejaculation the glans may enlarge before the corpora begin to fill. A raised threshold for glans enlargement may well be the mechanism involved when tricyclics are beneficial in treating premature orgasm.

Now that erectile physiology can be more freely discussed with patients, it is important for prescribers to collect and report the incidence and precise character of drug effects. Although erectile and ejaculatory deficits may be psychogenic, the mechanics of interference can be demonstrated in an increasing number of cases (4), and the incidental evidence of therapeutic experiments can greatly increase our knowledge of erectile physiology and idiosyncrasy.

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"Borderline"—Subtype of Primary Affective Disorder?

SIR: In "Discriminating Features of Borderline Patients" John G. Gunderson, M.D., and Jonathan E. Kolb, M.D. (July 1978 issue) conclude that "borderline patients can be discriminated with high accuracy from matched comparison groups with whom diagnostic confusion is common." The fallacy in their study is that "borderline patients were compared with schizophrenic patients, neurotic depressed patients, and a group of patients with differing diagnoses," whereas contemporary use of the term "borderline" is for patients with bipolar primary affective disorder (1).

A bipolar or mixed primary affective disorder comparison group was not included, perhaps because borderline is primarily an analytic concept (both authors are psychoanalytically oriented) and psychoanalytic theory is not comfortable with the idea of primary affective disorder (2).

This is not to say that the concept of borderline is useless. It is a sophisticated clinical formulation of what is ultimately a subtype of primary affective disorder.

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New York, N.Y.

Drs. Gunderson and Kolb Reply

SIR: Dr. Nakdimen states that borderline patients are a subtype of primary affective disorder. This is an interesting hypothesis for which there is some preliminary evidence in the work of Stone and in the claims for responsivity to MAO inhibitors made by Klein. However, because of contradictory trends suggesting a genetic linkage to chronic schizophrenia and the prevailing clinical wisdom of drug unresponsiveness in this group, Dr. Nakdimen seems unduly bold in his assertion. In any event, Dr. Nakdimen's belief that borderline patients would be difficult to distinguish phenomenologically from patients with primary affective disorder is naïve. The patients in our neurotic depressive group (1) appear to meet Feighner's criteria (2) for primary affective disorder and yet are relatively easy to distinguish descriptively. It would be far easier to distinguish our borderline sample from a comparison group composed of patients with bipolar primary affective disorder. Having said this, we can still agree with the idea that some undetermined fraction of patients with primary affective disorder have a borderline personality organization as outlined by Kernberg (3). The patients with the borderline personality disorder we described are in a narrower category which may find its place in the nosologic system with other forms of personality disorder but is not expected to encompass them. We expect that the most challenging comparison group we could have selected would have been patients with other forms of personality disorder—not primary affective disorder. We did not do this because of the concerns in the psychiatric literature about whether patients diagnosed as borderline could be discriminated from the schizophrenic and neurotic depressive groups which we selected.

It is our belief that diagnosis should depend on more than phenomenologic discriminability—it should depend on external validating criteria. Here our psychoanalytic orientation suggests that a particularly important validating criterion is whether a syndrome has an internal psychological coherence and that the areas to which Dr. Nakdimen refers (i.e., genetics and drug responsivity) have no necessary priority in this respect.

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3. Kernberg O: Borderline personality organization. J. Am Psychoanal Assoc 15:641–685, 1967

JOHN G. GUNDERSON, M.D.
JONATHAN E. KOLB, M.D.
Belmont, Mass.

A First-Degree Query

SIR: The paper "Psychiatric Illness in Relatives of Patients with Parkinson's Disease: An Expanded Survey" by Andrew Winokur, M.D., Ph.D., and associates (July 1978 issue) was appreciated. In the paper, the authors stated that "the 25 probands had a total of 113 male and 109 female first-degree relatives," or a total of 222. Neither I nor anyone I have asked can define first-degree relatives in a way to fit the data of almost 9 relatives for each patient unless we assume these families were unusually fecund. I had thought, apparently mistakenly, that first-degree relatives were parents and siblings. It would be helpful to learn which kin the authors include in that term.

HENRY B. BURTON, M.D.
Boone, N.C.

Dr. Winokur Replies

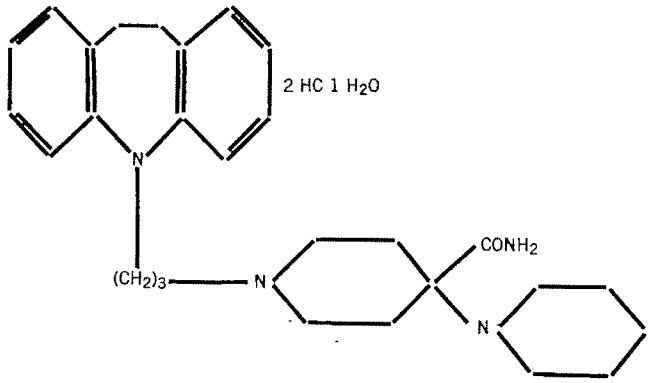
SIR: In response to Dr. Burton's questions concerning the number of relatives of the 25 probands in our study, we obtained data on all of the following: grandparents, parents, siblings, and children aged 18 and over. Only those relatives for whom a reliable history of possible psychopathology was available were included in the final data analysis.

ANDREW WINOKUR, M.D., PH.D.
Philadelphia, Pa.

Corrections

In the August 1978 issue there was an error in figure 2 of "Impact of Neuroleptic Chemotherapies on Schizophrenic Psychoses" by Pierre Deniker, M.D. The corrected figure appears below.

FIGURE 2
Structure of Carpipramine



There was also an error in table 3 of the "1977 Annual Report of the American Board of Psychiatry and Neurology, Inc." in the Official Actions section of the October 1978 issue. In the last column, headed "Foreign Graduates," the second number in the % column should be 50, indicating that 50% of the foreign graduates who took this examination passed.

In the November 1978 issue there was an error in "A Look at Psychiatric Decision Making" by Bruce Roberts, M.D. The first sentence of the second paragraph of the second column on page 1385 should read, "Assuming that we are dealing with the manifestation depressed mood, we see that condition 1 is "Y"; . . . ."

Also in the November 1978 issue there was error in "Prediction of Self-Mutilation in Hospitalized Schizophrenics" by David Shore, M.D., Douglas J. Anderson, M.D., and Neal R. Cutler, M.D. The second paragraph of the Discussion section should read, "Simultaneous with our study three investigators (J.E. Crowder, C.A. Gross, and J.E. Heiser), ..."

The staff regrets these errors.

BOOK REVIEWS

Oneness and Separateness: From Infant to Individual, *by Louise J. Kaplan, Ph.D. New York, N.Y., Simon & Schuster, 1978, 280 pp., $9.95.*

This book is an oasis—both literary and clinical. Dr. Kaplan has produced a combination of poetry and science. Both are apparent in her description of "the beginnings":

> Birth is a rupture of the state of biological oneness of mother and fetus. Mending the rupture is the major task of the mother-newborn couple. During the first month of human life, mother and newborn must come to know each other in a manner that will replace the physical oneness of the womb with psychological oneness, a oneness that is essential to life outside the womb as biological oneness was to life in the womb. (p. 59)

We have become accustomed to and perhaps stultified by the language of texts that profess to be scientific. We have forgotten that a good literary style can be as helpful to the scientist as it can to the art critic.

Clinically, in current books about childhood, we have lost the prospect of an intense and microscopic look at the early months of a child's life. We are so besieged with books describing the child's journey in a longitudinal sense and in a broad family context that we forget the intimacy as well as the intricacies of the beginnings. We are also so buffeted with the pathological (and who can deny it?) that it may be hard to imagine or even visualize true normalcy.

A pupil of Margaret Mahler, Dr. Kaplan has turned her microscope on the intimate and slowly-but-constantly changing relationships between mother and child. Many of these relate to the inevitable biological growth of the child, and many relate to the psychological growth of the mother in compensating for the changes she perceives, accepts, and adapts to in the child. The father is not forgotten, but this book primarily focuses on the small triangle and does not encompass, except in allusion, the larger world outside.

The book does not lack for usable clinical material in the detail. One cannot but be awed by the observations and knowledge represented in this small text. Dr. Kaplan has adopted a most useful device: a series of notes, arranged chapter by chapter, which can be read either before or after the narrative but which leave the actual telling of the changes uncluttered.

The descriptions of life periods are primarily Mahler's: beginnings, limbo, oneness, separation, and constancy. With true simplicity the author describes the extremely new relationships of the second year as the infant carries on the separation process that forces complex responses from parents, particularly from the mother.

Not everyone in the clinical field will accept the author's concept of the "thinking mind," but it is a useful concept to help us understand the particular responses of the child as his or her abilities grow. The thinking mind of the child certainly confronts the parent with a different phenomenon, often much less appreciated than that of the comfortable, warm oneness of the beginnings.

Following the period of growth and separation Dr. Kaplan describes the period of constancy and self-confidence, which gives the child a "sense of contributing in a real way to the real affairs of human life," thus helping the child to put his or her parents in perspective.

With a style peculiarly reminiscent of Nevil Shute, Dr. Kaplan slides from descriptions of the normal to recollections of situations in which the relationship is less than ideal to those in which it is far from ideal: "On the other hand, whenever a child is put in the position of humiliating dependence, his ordinary natural desire to please will be contaminated by fear of aloneness—under conditions of oppression the child may remain a child forever."

Dr. Kaplan closes her brief developmental travelogue with a code describing various ways of living and contrasting some of the difficulties that young mothers face today in cultures that are simpler but not necessarily more satisfying.

Although the style of this book is appealing, the complexity of the text requires careful reading and, at times, rereading. One of the notations on the cover suggests that the book should be especially helpful to "women who are questioning their role of mother as they try to redefine their role of wife." There are no doubt many such women, but those who are struggling with their own concerns about child abuse or who are suffering from economic hazards will not be entirely persuaded by this text.

Perhaps professionals in the field will get more from it. The completeness of the notes makes the entire writing valid. The depth of detail is not only immediately helpful but links together a continuum of knowledge. The theories of how this period is related to later life are made clear. Some will disparage this book for not being keyed to current technological advances. However, Dr. Kaplan provides a clear picture of the biological and psychological basis of existence.

HENRY H. WORK, M.D.
Washington, D.C.

Psychosomatic Families: Anorexia Nervosa in Context, *by Salvador Minuchin, Bernice L. Rosman, and Lester Baker. Cambridge, Mass., Harvard University Press, 1978, 346 pp., $15.00.*

At the end of this interesting, challenging, and important book the authors make the unequivocal statement that "anorexia nervosa is a disease of the child in the family." They reach this conclusion by way of a journey starting with research into the cause of episodes of diabetic acidosis in a group of juvenile diabetics. With the help of a physiological measure—the level of free fatty acids in the blood—the research team was able to track down these episodes to family-induced stress. They then studied these families along with

others, including the families of anorexic patients, and arrived at a formulation of the "psychosomatic family." The anorexic family is considered a psychosomatic family.

The principal tools used by the investigators are "the family task," in which the family is observed while they attempt to reach consensus on a series of set tasks (such as planning a menu), and the "family diagnostic interview," in which a conflict is induced by the therapist between the parents and then the introduction of the anorexic child to detour conflict is observed. The "anorexic family" is characterized by the following behavior patterns: enmeshment, overprotection, rigidity, and, preeminently, conflict avoidance—often through detouring conflict by forcing triangulation of the child into the spouse subsystem. The authors consider all previous theories of anorexia nervosa and methods of treatment of the anorexic patient outmoded. They believe that a systems paradigm using family intervention must supersede the earlier model in the interest of understanding and treating the disease.

The authors used their method of family intervention in 53 families treated over a period of 7 years. Their results were very good both medically and psychosocially in 86% of the cases. Furthermore, the improvements were maintained over a substantial follow-up period. The heart of the book consists of transcripts of the initial therapeutic encounters with four families. Three of these were conducted by Dr. Minuchin and one by Dr. Ronald Leibman. An interesting feature of these interviews is the focal role of a family lunch session during which the anorexic child's eating patterns and the reaction of the family were observed and discussed. In these chapters the reader can observe the lively, engaging, and directive therapeutic technique of the therapists, alternately challenging and ingratiating to the participants.

Have the authors proven their case that family intervention is the preferred method of treatment for anorexia nervosa over all previous and current methods? Their results, although certainly excellent, have been criticized on the grounds that Minuchin and associates were not treating true anorexics, or, if they were, the patients were very young and at an early stage of their illness. Dr. Minuchin takes note of these objections but attributes them to the defensive reaction of linear therapists. Although one cannot read this book objectively without being impressed by the authors' careful approach, the patients do appear to have been young and in a relatively early stage of their illness. The diagnosis of anorexia nervosa includes other more malignant and less tractable conditions; one can acknowledge this without downgrading the method and results reported here.

Scrutiny of the dramatic case study chapters raises questions about the applicability of the authors' theory of the psychosomatic family to all of the cases reported. In fact, the four families seem quite different. A certain amount of straining and Procrustean manipulation is required to fit them into the same framework. The authors' insistence that the theory of conflict avoidance is universally applicable to all of their cases (except one patient who, triumphantly, was found to have an organic cause for her symptoms) is the only really jarring note I found in the book. Even Dr. Minuchin at one point comments that one of the patients, Loretta, seems to have approached intrafamily conflict with a vigor that was certainly not a manifestation of avoidance. It is difficult to accept conflict avoidance as a significant factor among the members of a family who go at each other with the élan observed in Loretta's family.

Rather than ascribing all of the favorable results to the specific maneuvers described to combat the characteristics supposedly definitive of the anorexic family, the authors might have considered an additional and important nonspecific therapeutic factor—the forceful and continuing intervention of an authoritative (and sometimes charismatic) therapist into a disordered family confused and befuddled by their inability to cope with the anorexic behavior of an adolescent child. Such an intervention can be thought of as deflecting and, in a sense, detoxifying the family conflict, thus allowing resolution to take place before the symptoms harden into chronicity and take on a stubborn and resistant life of their own.

The above caveats do not detract from the value of this book and the method of family intervention it describes. This treatment approach appears to be successful for a substantial proportion of cases of anorexia nervosa. Those who use other methods of treatment ought properly to be challenged by these results. They will need to compare them with the results of their own methods.

PAUL CHODOFF, M.D.
Washington, D.C.

Communication and Social Interaction: Clinical and Therapeutic Aspects of Human Behavior, *edited by Peter F. Ostwald, M.D. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1977, 337 pp., $24.50.*

Dr. Ostwald, well-known for his own distinguished contributions to the field of human communication over many years, has accomplished a most welcome tour de force in this volume. He has assembled 25 essays from present and former staff members of the Langley Porter Neuropsychiatric Institute, together with a foreword by Lawrence Kolb, a prologue by Jurgen Ruesch, and an epilogue by Gregory Bateson. In a sense, the book is a monument to the Langley Porter group under the leadership of Jurgen Ruesch and Gregory Bateson as well as Enoch Callaway, Leon Epstein, Alexander Simon, Robert Wallerstein, and the late Karl Bowman.

In his magnificent historical survey, Reusch presents the development of the field of human communication and his own significant contributions. Bateson's exposition of the mind-body dilemma and his discussion of his own historical involvement with the theories of logical types, cybernetics, and information theory are superior. Both essays should be required reading for all students in the humanities and the physicochemical, social, and clinical sciences who are grappling for new models of mind with which to understand human behavior.

It is impossible to adequately characterize the 25 essays, which cover a vast spectrum from "Electric Potentials and the Human Brain" by Enoch Callaway to "The Impact of Leadership in the Community" by Chesley Herbert. One can say that each essay has individual excellence and is worth reading. They are grouped into four sections: Basic Elements of Human Communication (essays by Callaway, Gaarder, Ekman and Friesen, Ostwald, Starkweather, and Horowitz), Disturbed Patterns of Communication (essays by Bittner, Raskin, Berblinger, Brodsky, Simon, and Fisher), Communication As a Therapeutic Tool (essays by Langsley, Patterson, Wallerstein, Piner, Motto, Berlin, and Allen), and Communication Adapted to Space, Time, and Social Realities (essays by Flinn, Fiske, Kessler and Glick, Rosow, Lauer, and Herbert).

This alumni roll call is impressive, even startling, in it

scope and guarantees sound scholarship together with an energetic pursuit of innovations. I do not know without trying it out in seminar groups whether this book could serve as a basic text in multidisciplinary studies. I do believe it could be a valuable reference work for all who labor in these difficult fields.

HENRY W. BROSIN, M.D.
Tucson, Ariz.

The World of Emotions: Clinical Studies of Affects and Their Expression, *edited by Charles W. Socarides, M.D. New York, N.Y., International Universities Press, 1977, 612 pp., $24.95.*

Although the centrality of affects in the motivation of human behavior is a core concept in psychoanalysis, the understanding of their role has changed. Formerly considered discharge phenomena, the damming up of which produced symptoms, affects are now viewed as the feelings associated with thoughts that reflect physiological and psychological states, communicate needs, signal danger, influence behavior, affect character development, and stimulate adaptation as well as psychopathology. The articles that Dr. Socarides has collected and on which he comments illustrate the complex interaction of these factors.

The approach in these articles, each of which discusses a different affective state, is essentially clinical. All of the authors stress the importance of the recognition of affects because affect can be denied, repressed, and defended against in a variety of ways due to an individual's fear of being overwhelmed by them or the wish to avoid the pain they generate. The capacity to cope with affects depends on constitutional factors as well as experiences that promote or inhibit maturation.

Searl's article on screaming illustrates the relation of affects to physiology. The unpleasant physiological discharge accompanying the rage expressed in screaming is also an act of aggression. Because the unpleasure associated with the rage is experienced internally, it is felt as an act of aggression against the self. Thus, the hatred against the depriving object is also felt as self-hatred. The subsequent ability to master situations that could stimulate screaming is an important determinant of psychic structure. Zetzel points out that the mastery of traumatic experiences influences the capacity to bear anxiety. Mastery involves the transformation of overwhelming anxiety into signal anxiety. This transformation requires a degree of ego development that is dependent on experiences providing emotional warmth and indicating that human contact is not dangerous and mastery is possible.

The "as if" personalities described by Deutsch have been deprived of these experiences and as a consequence fear affective involvements. Their relationships are distant and superficial. In these as well as other narcissistically wounded people, fear of involvement may result in feelings of emptiness, boredom, and loneliness. Balint and Greenson point out that these states reflect the lack of feedback from others. Attempts to cope with loneliness often result in transient and unsatisfactory relationships based not on love but on narcissistic or sadomasochistic fantasies. The capacity to love, according to Bergmann, is rooted in satisfactory early symbiotic relationships from which the child is allowed to appropriately emancipate himself.

Many of these authors believe that depression early in life is a major factor in the genesis of affective states, which may either be transient or contribute to character structure. In either case, affective states express a view of the relationship and potentially influence it. Rado views melancholia as an attempt at atonement for rage and envy. Bergler relates ingratitude to the persistence of omnipotence as a defense against wounded narcissism, helplessness, and disappointment. Several of the authors feel that wounded narcissism is an important determinant of such states as bitterness, querulousness, smugness, vengeance, arrogance, and jealousy. In each case, the feeling expresses anger at the felt deprivation and is an attempt to obtain reparation. The deprived and narcissistically wounded person may also feel unworthy and may attempt to cope with this by excessive generosity or by responding with undue enthusiasm to situations that promise gratification. Although all of the authors stress the importance of pre-oedipal factors in determining affective states, they also indicate that conflicts and experiences at all levels of psychosexual development contribute to their elaboration.

The dynamics of many of the affective states are similar, but the richness of the clinical material in this book highlights their differences and enhances our understanding of them. This book also provides a stimulus for further study because the articles point out the gaps in our knowledge of the transformation of the physiological into the psychological, the influence of affective states on character formation, and the details that account for the elaboration of similar experiences into different affective states.

REBECCA Z. SOLOMON, M.D.
Hartford, Conn.

The Biochemical Basis of Neuropharmacology, 3rd ed., *by Jack R. Cooper, Ph.D., Floyd E. Bloom, M.D., and Robert H. Roth, Ph.D. New York, N.Y., Oxford University Press, 1978, 322 pp., $13.95; $6.95 (paper).*

The third edition of this textbook provides an up-to-date summary of the biochemical basis of neurophysiology and neuropharmacology. The book is highly readable, the diagrams and illustrations are helpful, and the chapters are appropriate selections from current knowledge of brain biochemistry and pharmacology. Throughout the text the authors strive to evaluate new information and new theories using rigorous scientific standards. Their chapters offer frequent and terse criticisms of studies and hypotheses that were based on imprecise methods or premature conclusions. Gaps in current knowledge are as carefully described as are new discoveries.

A brief introduction explains the book's emphasis on the synapse by noting that the best concepts of neuropharmacological drug action implicate synaptic events. The second chapter outlines the cellular foundations of neuropharmacology and includes a concise summary of the ultrastructure of neurons. This chapter also introduces the important concepts of chemical transmission at the synapse, the brain permeability barrier, and the bioelectrical properties of cells. Perhaps most importantly, the second chapter describes the theoretical and practical approaches used by neurochemists in their attempt to understand neuronal function and synaptic transmitters.

The third chapter contains a brief summary of metabolism in the CNS. The metabolic origins and interrelationships among compounds of neurochemical interest are briefly but clearly presented, and a table summarizes the hereditary

brain diseases that are associated with abnormal cerebral metabolism.

The chapter on receptors, new to the third edition, is a surprisingly succinct account of developments in this rapidly evolving field. The description of the properties of "authentic receptors," the brief but lucid account of the mathematics of receptor kinetics, and the discussion of the phenomenon of "cooperativity" and the concept of "membrane fluidity" will allow the reader to follow future developments in the exciting field of receptor neurochemistry.

The largest sections of the book are devoted to summaries of the current knowledge of putative neurotransmitters. Sections on acetylcholine, the catecholamines, serotonin, the proposed amino acid transmitters (GABA, glycine, glutamic acid, and taurine), prostaglandins, and histamine are balanced reviews of both what is known and what remains to be learned about these neurochemicals. For each proposed neurotransmitter the authors provide lucid discussions of techniques for assay, studies on anatomical localization, and descriptions of anabolic and catabolic enzymes. They also present evidence for the roles of each neurotransmitter in synaptic transmission and, where data exist, in physiology, behavior, and psychopathology. Data on the interaction of active neuropharmacological agents with the synthesis, storage, release, reuptake, or catabolism of these possible transmitters are outlined as part of the description of each compound. Part of a chapter is devoted to the role of the cyclic nucleotides in the general concept of the "second messenger" system. This section contains recent data and descriptions of potential pharmacological interactions with this system. Clearly defined criteria for establishing second messenger mediation should prepare the reader to evaluate new research in this increasingly important area of neurochemistry.

The neuroactive peptides are explored in the other new chapter of the third edition. There has been an explosion of research knowledge on these remarkable substances, and the authors recognize their potential importance. Moreover, they give an accurate and intriguing account of the natural history of research on neuroactive peptides that will guide readers in their review of the already extensive literature in this area.

This book can be strongly recommended as an introductory textbook for graduate and medical students interested in neuropharmacology. Residents in psychiatry and neurology and practicing physicians in these specialties will also find this book an excellent critical review. Owners of the first and second editions will welcome the two additional chapters on receptors and neuropeptides as well as the new information in each chapter. The rapid expansion of knowledge in neuropharmacology will no doubt make some of this book outdated in the near future. I anticipate that future editions of this book will maintain the standard of excellence established by the authors of this monograph.

PHILIP A. BERGER, M.D.
Stanford, Calif.

Psychopolitics, *by Milton Greenblatt, M.D. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1978, 268 pp., $18.50.*

Two significant events make the publication of this book particularly timely. The recently completed report of the President's Commission on Mental Health (1) as well as the nationwide concern with government spending should place this book on the required reading list of all mental health professionals. Educators in psychiatry and other mental health disciplines have long sought such a work to assist them with the difficult task of training new people in the field of "psychopolitics." In the past, program directors have learned these important lessons the hard way or have been easily discouraged. Dr. Greenblatt shares his experiences with the reader in a most candid fashion.

The style of this book is appealing and at times even dramatic. It often reads like a novel; one finds himself hurriedly looking to the outcome of the unfolding mystery. Dr. Greenblatt employs the case method to describe his successes and failures and frequently uses examples from his experience in Massachusetts (where he was Commissioner of Mental Health). He applies psychodynamic understanding in many instances.

Dr. Greenblatt is a strong advocate of services and is able to discuss ways of attempting to implement them. After a careful definition of "psychopolitics," he sets out to describe it. There are several provocative subjects covered in the book, such as bureaucracies, consumer groups, patronage, rights of various interested parties, and the use of volunteers.

The subject of the closing of state hospitals and how to phase them out when indicated occupies a great deal of this book. Having gone through a similar experience in my own state, I find the description in the book relevant and familiar. Dr. Greenblatt treats the community mental health center movement fairly and presents both sides of the controversy. He also provides an interesting chapter on the criticisms of psychiatry and discusses positions on drug treatment, ECT, and psychosurgery, dealing with these sensitive subjects fairly and educationally. He also discusses the conflict between what society can afford today and what resources are needed. No doubt this dilemma will be debated on professional as well as fiscal and political fronts in the months to come.

At the close of his book the author presents several points of view on such topics as community mental health centers, the mental health center of tomorrow, resistance to community-based treatment, the minority question, mental health and sexual politics, urbanization and poverty, and psychopolitics and psychiatry. I found the book very readable and even exciting. It should appeal to legislators, bureaucrats, mental health professionals, and critics of the mental health professions.

REFERENCE

1. President's Commission on Mental Health: Report to the President 1978, vol 1. Washington, DC, US Government Printing Office, 1978

ALAN M. ELKINS, M.D.
Portland, Me.

Violence and the Family, *edited by J.P. Martin. New York, N.Y., John Wiley & Sons, 1978, 352 pp., $29.95.*

This is one of several British volumes on family violence that resulted from government hearings on the subject. The book is competently authored and contains a blend of criminological data, clinical vignettes, and historical perspectives

However, some chapters are repetitive, and the book is not easily read because it takes on the tone of a reference text with a wide scope.

A chapter on women refugees provides insights into how another culture has attempted to institutionalize something that the United States is just beginning to grapple with. Another chapter points out why the United States has only recently come to look at violence since the political assassinations that have occurred in the last 15 years. Old themes emerge: the failure of the police to intervene in domestic violence and the use of denial by the medical profession.

There is a splendid chapter on training social workers to cope with violent families. It essentially deals with intervention strategies and countertransference issues. There is a very short "note" on men's aid and only a few pages devoted to group therapy with abusing parents. I should like to have seen more on treatment. One essay deals with the need for practical help as opposed to theory and therapy with destitute families. For such people, talking is a luxury. What they require is medical attention, food, money, and the like. I think we often forget this in our community mental health center work.

The editor of this book has an excellent chapter on bureaucratic obstacles to social and medical intervention in the case of family violence. It is refreshing and candid, clinically sound, and useful for all students of social policy in medicine.

JOHN R. LION, M.D.
Baltimore, Md.

The Adoption Triangle: The Effects of the Sealed Record on Adoptees, Birth Parents, and Adoptive Parents, *by Arthur D. Sorosky, M.D., Annette Baran, M.S.W., and Reuben Pannor, M.S.W. Garden City, N.Y., Anchor Press/Doubleday, 1978, 256 pp., $8.95.*

The adoption triangle is illustrated in the Bible by the story of Moses. As a child in Egypt Moses did not know about his Hebrew origin, although his wet nurse was his birth mother. Only as a man was he informed about his heritage; after a struggle of dual identification he finally selected the Hebrew one.

This volume deals with the quest for reunion with lost parents, documented by letters. The letters written by birth parents are fascinating. They indicate resentment, regret, and bitterness as well as interest in the fate of the "lost" child. Letters from the adoptive parents express the other side of the story. There are also letters from the adoptees. For instance, Linda, like Moses, was not told that she was an adopted child. She writes,

> When I was 10 years old I realized that I didn't look like any of the other members of the family, and also wondered why there were no pictures of me as a baby in the family album. During my teens I became preoccupied with the lack of resemblance to my parents. So when I was 18 I ran some blood type tests and determined for myself that I could not possibly be my parents' child.

The search for birth parents is described in detail and easily captures the reader's attention. The time spent on the search varies. In one case it was three years, in another case three weeks. A typical letter gives an idea of the ordeal:

> I found my biological parents within 3 weeks after deciding that I was ready to meet them. I'm a medical student, and I found a sympathetic professor who obtained my birth medical records. It was illegal, but I didn't care, nor did he. My bio-mother had listed a friend and her address as "person to be contacted" and I pursued this route. Using the records office I went through house deeds, found her original one when it was sold and then the next house she bought until I located her.

The description of reunions comprises the most dramatic part of the book. This section illustrates what happens after the search has ended. The majority of reunion experiences reveal a romantic emotional content that is unrivaled in the most imaginative fiction. Ninety percent of the adoptees were satisfied with the outcome of the reunion. Most of these reported a sense of personal fulfillment, resolution of genealogical concerns, and diminished identity conflicts. Eighty-two percent of the birth parents encountered were positive and accepting, and only 10% reacted adversely to the reunion with their relinquished child. In contrast, many of the adoptive parents had difficulty initially in adjusting to the experience. Thirty-six percent of the adoptive parents were cooperative and understanding, 20% were mildly upset, and 10% were quite hurt. In the other cases the adoptive parents had either died or were not told about the reunion in order to spare their feelings.

The authors are very open about their bias—they are in favor of opening the original birth records to adult adoptees—but there are other views as well. For example, Ann Murphy (1) has pointed out that television programs, magazine articles, and angry young people are demanding that old files be opened, that old trusts be violated so that adopted children can discover the identities of their "real" mothers and stage an emotional grand reunion. Ms. Murphy contacted many women who had given up a child for adoption in Georgia and Connecticut and was impressed with the fact that many of these women did not want the reunion. As one of the "real" mothers said, "I'm just a woman who gave birth, I'm not a mother. I contributed the months of pregnancy and the hours of delivery. The woman who raised my child contributed her whole life. She's the real mother—not me."

Clearly, there are two sides to the issue, both worthy of consideration. What makes this volume interesting is not the authors' bias, but the letters from birth parents, adoptive parents, and adoptees. The verbatim account makes the book worth reading.

REFERENCE

1. Murphy A: A familiar stranger. New York Times, July 31, 1978

LEON TEC, M.D.
Norwalk, Conn.

New Perspectives on Psychotherapy of the Borderline Adult, *edited by James F. Masterson, M.D. New York, N.Y., Brunner/Mazel, 1978, 162 pp., $10.00.*

This collection of papers by Peter Giovacchini, Otto Kernberg, James Masterson, and Harold Searles is rich in clinical offerings. The editor has woven the more formal statements about the borderline adult of each of these four experts with

the informal reactions of the other three contributors. There is, as a result, an intimate, personal characteristic flowing through the book that provides continuity, although there is much lively discontinuity of opinion among the quartet on some points.

In addition to the personal quality that brings the four differing sections of this book together, the authors' knowledge, depth of experience, and differing viewpoints coalesce in such a way that by the time I finished this short book I had a new and fuller view of a complex clinical entity whose very nature defied precise delineation at times. The Masterson group, which initiated and sponsored the conference from which these writings grew, has performed a valuable service in updating our knowledge of theory and treatment of the borderline adult.

My one major suggestion, perhaps too personal to be called a criticism, is that Dr. Kernberg's section, "Contrasting Approaches to the Psychotherapy of Borderline Conditions," should have appeared first instead of third in order in the book. Dr. Kernberg presents a beautifully precise review of the psychoanalytic definitions, concepts, and assumptions that underlie the four authors' contributions. Another minor criticism is that a more accurate title for the book would have been *New Perspectives in Psychoanalytic Psychotherapy of the Borderline Adult* because there are other psychological and psychopharmacological therapies than the psychoanalytic for helping such individuals, regardless of the unanimity among the authors about the psychoanalytic approach.

This book successfully delineates a particular clinical entity and offers a theoretical framework for understanding the psychodynamics of the entity. It also presents differing styles of psychoanalytic therapy using the common denominator of a modified psychoanalytic framework, and thoroughly discusses different and complementary views on the role of the therapist and the importance of countertransference when treating such difficult patients. In the process, the book allows one to review his or her own approaches for helping adults who suffer from the borderline syndrome.

RICHARD E. DAVIS, M.D.
Overland Park, Kan.

Evaluation of Behavior Therapy: Issues, Evidence, and Research Strategies, *by Alan E. Kazdin and G. Terence Wilson. Cambridge, Mass., Ballinger Publishing Co. (J.B. Lippincott Co.), 1978, 213 pp., $15.50.*

In 1972 Bergin and Strupp wrote *Changing Frontiers in the Science of Psychotherapy* (1). The fundamental thesis of this book was that the question of whether or not psychotherapy is effective was the wrong question at the wrong time and that it was approached with inadequate methodology and insufficient understanding of behavioral and emotional problems. This conclusion led Bergin and Strupp to recommend abandoning large group outcome studies attempting to answer this question in favor of studies smaller in scope answering more specific questions, such as, under what conditions and with what problems do certain well-defined procedures work? However, in the intervening six years these recommendations too often fell on deaf ears, as polemicists and charismatic leaders of various schools of psychotherapy continued the fruitless debate on behavior therapy versus psychoanalysis versus client-centered therapy, etc.

This debate is not continued in *Evaluation of Behavi Therapy*. The purpose of this book is not to extoll the virtu of behavior therapy as yet another school within psychoth apy. The work of these two extremely competent young c nicians and investigators proceeds from the conceptual ba of Bergin and Strupp. Kazdin and Wilson review the existi information on the effectiveness of specific procedures w specific disorders. In a sense it is probably misleading to ti the book *Evaluation of Behavior Therapy* because the cc clusions are relevant to all behavior change procedur based on psychological principles. However, the autho note that a second purpose of their book is to present frankly realistic view of the efficacy of behavior therap These two valuable goals coexist in this book in a manr that detracts slightly from the overall thrust of the work.

Chapter one begins with an overview of behavior thera that is extremely well written and a brief review of conte porary conceptualizations of behavior therapy that are creasingly splintering those within the field. These inclu applied behavior analysis, which grows out of operant tra tions; the neobehavioristic mediational S-R model, whi contains Wolpe's and Eysenck's views of behavior therap social learning theory as espoused most prominently Bandura; and, finally, the newer cognitive behavior therap a collection of procedures often identified with Beck, M honey, and Meichenbaum.

In chapter two the uneasy coexistence of the two ma goals of the book becomes more apparent. After reporting admirably exhaustive review, including tables for easy ref ence, of outcome research comparing "behavior therap with alternative therapies in the areas of neurotic disorde sexual dysfunction and deviance, addictive behaviors, ps chotic disorders, delinquency, childhood disorders, a mental retardation, the authors note that these studies are little worth. Using the well-known study of Sloane and as ciates (2) as an illustration, Kazdin and Wilson state, "'I inadequacies of the data base are for the most part pronounced, and the conceptual model that guided th comparisons sufficiently misguided, that meaningful int pretation is often impossible." Nevertheless, the revi is nicely laid out and the evaluative comments at the end each section allow the reader to determine for himself or h self which studies are useful.

Extending these arguments, the authors correctly nc that recent reviews of psychotherapy outcome literature Luborsky and associates (3) and Smith and Glass (4) cc cluding that, *in general*, all therapies produce essentially t same effect, at least with neurotic patients, are also m leading for the same reasons. With *specific*, well-defined d orders, from phobias through psychotic behaviors and va ous educational applications, and particularly in the rece study comparing cognitive behavior therapy and pharma logical approaches to depression (5), specific, well-defin procedures are superior to alternative treatments.

Although the critical review of outcome studies will pro useful to those desiring this resource, the heart of the bo and the section that advances our science of clinical resear appears in the last two chapters. Chapter three elaborates conceptual and assessment issues making most outcome search problematical at this time, and chapter four lays c detailed guidelines that should shape the direction of search and therapy evaluation for the next 10 years.

This book provides a summary evaluation of the fectiveness of behavioral procedures, but its more import contribution is to invalidate the question of the effectiven of "behavior therapy" or "psychotherapy" and to point

the unique contribution of behavior therapy as a scientifically based approach that asks specific questions about specific techniques and problems.

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3. Luborsky L, Singer G, Luborsky L: Comparative studies of psychotherapies: is it true that everyone has won and all must have prizes? Arch Gen Psychiatry 32:995-1008, 1975
4. Smith ML, Glass GV: Meta-analysis of psychotherapy outcome studies. Am Psychol 32:752-760, 1977
5. Rush AJ, Beck AT, Kovacs M, et al: Comparative efficacy of cognitive therapy and pharmacotherapy in the treatment of depressed outpatients. Cognitive Therapy and Research 1:17-37, 1977

DAVID H. BARLOW, PH.D.
Providence, R.I.

The Myth of Psychotherapy: Mental Healing as Religion, Rhetoric, and Repression, *by Thomas Szasz. Garden City, N.Y., Anchor Press (Doubleday), 1978, 226 pp., $8.95.*

Thomas Szasz, the outspoken avenger of alleged wrongdoings by the psychiatric establishment, claims in this book that psychotherapy is a combination of religion, rhetoric, and repression. As always, he underscores certain truths about abuses of psychiatric practice and makes a number of good points. He defines psychotherapy as "what two or more people do with, for, and to each other, by means of verbal and nonverbal messages."

In Szasz's view, Freud's definition of conversation as therapy and his definition of those he worked with as patients suffering from mental diseases were false, rhetorical, and self-serving. Szasz sees conversation as religious in origin, with the curing of souls as its goal and rhetoric as its method. He suggests that Freud used the language of science to transform metaphor into accounts of actual phenomena in the brain and that this language attests to Freud's power as a rhetorician rather than to the scientific validity of his constructs. This idea is consonant with current analytic attempts at reworking and/or discarding early metapsychology. Szasz's view of training analysis as an example of coercion by institutional psychoanalysis is under review by some education committees in the United States. His position that Freud was an autocratic leader trying to assure that his marketable product flourished has also been commented on by other writers.

These positions could stimulate useful discussions in professional study groups and analytic seminars. Szasz himself is an able rhetorician with a rich command of psychiatric history. Unfortunately, he is given to extreme positions and overstatements, which in my opinion render his positions meaningless and at times ludicrous. Citing Jones's account of Hoche (who apparently suggested that all analysts should be committed to asylums), Szasz states, "Ironically, many of the criticisms that Hoche leveled against psychoanalysts were well founded, but he overplayed his hand." The same, I suggest, is true of Szasz.

For instance, in a chapter on Mesmer's "medicalization" of magnetism, which Szasz views from a religious and metaphorical perspective, Szasz describes the "so-called hysterical" symptoms of a particular woman as a "case of imitated or pretended blindness." Hysterics, then, do not suffer from real afflictions but pretend to be ill. Szasz says that he does not blame the so-called neurotic, but how else can an accusation of fakery be viewed?

Szasz claims that psychotherapy is "base rhetoric" because it moves the listener to evil. Freud belittles, demeans, and attacks others only to claim that he is attacked by them; he "uses psychoanalysis not to heal but to harm, not to understand but to undermine." In discussing Heinroth, Szasz describes repression concealed as treatment and says that nothing has changed since Heinroth's day. Such accusations overlook subtlety, distinctions among therapists, and the complexities of psychoanalytic treatment.

Szasz also likes to argue freely by analogy. He equates convulsive treatments and leukotomy with psychotherapy and Mesmer's magnetic fluidism with Freud's libido. Szasz would adhere solely to his thesis of metaphor and ban from justifiable investigation all genetic and biochemical studies of brain metabolism. He analogizes Erb's nutritive disturbance of the nervous apparatus with the catecholamine hypothesis and says that ECT is equivalent to chemotherapy and that both are fakery.

Finally, Szasz's continual use of alliteration is a stylistic tic that grates on the reader. In two facing pages, for instance, he talks about "execration and execution," "diagnostic derogation," and "Freud's lexicon of loathing."

Szasz has some important observations to make about the dangers of excess in contemporary psychiatry, including the designation of disparate social and recreational activities as therapy. However, his contributions get lost in his habitual harangue. He reduces and thus dismisses as commonplace the fact that psychotherapy is conversation between "patient and doctor." Attention paid to the content and context of that conversation is precisely what makes psychotherapy a unique and revolutionary contribution. Whether called psychotherapy or "iatrologic," as Szasz suggests, the distinction is ultimately a matter of semantics.

ANDREW P. MORRISON, M.D.
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Contemporary Models in Liaison Psychiatry, *edited by Robert A. Faguet, Fawzy I. Fawzy, David K. Wellisch, and Robert O. Pasnau. New York, N.Y., SP Medical & Scientific Books (Spectrum Publications), 1978, 196 pp., $20.00.*

The authors of this book are members of the Department of Psychiatry at the University of California, Los Angeles. They have written 14 chapters covering both common and esoteric problems seen on various UCLA liaison psychiatric services. The case reports given were originally presented in consultation-liaison grand rounds at UCLA School of Medicine in response to a request for psychiatric consultation from the medical-surgical staff of the UCLA Medical Center. Although the chapters are written by different clinicians, there is a pervasive emphasis on a team approach to the various issues discussed. The authors also repeatedly emphasize that the ultimate aim of consultation-liaison psychiatry is the optimal care of the sick by attending to personal and interpersonal issues involving the patient, family, and staff that interfere with medical treatment and management. Furthermore, they explore the varieties of responses to stress and

illness and point out that each patient's reaction is unique and must be understood in a highly personalized fashion.

A listing of the chapter headings gives an overview of the wide variety of topics covered: "The Earliest Intervention: Consultation to the High-Risk Parent and Professional in the Neonatal Intensive Care Unit," "Liaison to Medically-Ill Heroin Addicts: The Mental Health Professional as a Tightrope Walker," "Will I Die? The Child with Life-Threatening Illness," "Life in a Venus-Fly Trap: Psychiatric Liaison to Patients Undergoing Bone Marrow Transplantation," "Infertility and Habitual Abortion: The Search for a Psychological Factor," "Munchausen's Syndrome: The Consultant's Dilemma," "Treating Violence Against Women: The Emergency Room Care of the Rape Victim," "Change Me: The Request for Rhinoplasty," "After Being Turned Inside Out: The Team Approach to the Ostomy Patient," "The Child with Ulcerative Colitis: Play Therapy as a Rehearsal for Surgery," "Psychiatric Aspects of Contraception, Abortion and Sterilization," "The Traction Intolerance Syndrome: Psychiatric Complications of Femoral Fractures in Young Adults," "Endocrines in or and Depression," and "Consultation to Neurology: Brain, Behavior and Patient Care."

It can be seen that the authors cover a wide range of subjects. Their approach is eclectic, and they provide numerous case reports, some of which are vignettes and some reported in detail that reflect the case conference origins of the book. Unfortunately, the writing is inconsistent, the grammar is often poor, and the index is incomplete. Trade names of drugs are used without reference to generic terms. However, the book serves to demonstrate the multidisciplinary character of liaison psychiatry and to emphasize the liaison psychiatrist's unique role of serving the traditional physician's functions of personal, comprehensive, and continuous care through the course of the patient's illness.

This book also accomplishes the objective of blending clinical observations, giving practical guidelines, and producing questions for future research studies. *New Frontiers of Liaison Psychiatry* might have been a better title for this book, which will undoubtedly be referred to by workers in the rapidly expanding field of consultation-liaison psychiatry.

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Psychosurgery and the Medical Control of Violence: Autonomy and Deviance, *by Samuel I. Shuman. Detroit, Mich., Wayne State University Press, 1977, 351 pp., $18.50.*

In recent years psychosurgery has received a great deal of attention from philosophers, lawyers, and social scientists—probably more than it deserves in terms of its role in contemporary American psychiatry. It has come to be a symbol for a number of "hot" issues—medicine as a means of social control, competence for informed consent, proxy consent, the relation of therapeutic to experimental procedures, the regulation and control of surgical as opposed to pharmacological treatment, and the significance of involuntary confinement or total institutionalization in the consideration of medical methods of behavior control.

These issues were crystallized by the widely reported "Detroit psychosurgery case," *Kaimowitz v. Department of Mental Health* (1). The Medical Committee for Human Rights brought suit to release a man who had been confined to a state institution for the criminally insane for 18 years and who was to be the subject of a study at the Lafayette Clinic in Detroit on the effect of amygdalotomy on recurrent aggressive outbursts. The committee questioned the law under which the man had been detained, the quality of his informed consent, the scientific validity of the research, and the problem created by research on involuntary detained subjects. The prisoner-subject-patient was released before the trial concluded, but the court held a 15-day trial and commented on many of the basic issues raised. The events leading up to the trial and the opinion of the court have already been described by Ronald Gass (2). They provide the framework for this extended discussion by Samuel Shuman, Professor of Law and Psychiatry at Wayne State University, who represented the Lafayette Clinic and its individual staff physicians in the trial.

Professor Shuman's book is wide-ranging—perhaps too wide-ranging. His topics for consideration include a philosophical discourse on the concept of causation and the medical malpractice dilemma and his preferred solution to it. He touches on many of the critical problems in formulating a policy about psychosurgery, but it is not easy for the reader to extricate these discussions from the text. In addition, there is one important issue that is not discussed at all, psychosurgery itself. There is no discussion of the history, technology, theory, practice, or result of psychosurgery. The book is written by a lawyer and adopts a "black box" approach to psychosurgery, talking about "it" by discussing what various experts have said, what evidence is acceptable and what is not, and what the definition should be but not about the brain or about surgery. Professor Shuman's argumentation is legalistic, and the medically oriented reader may become impatient with his interest in dissecting and destroying the arguments of his opponents and his defending and justifying his judicially rejected position in the Detroit trial rather than examining the available facts directly.

In spite of these difficulties, many critical issues are raised—the relation of abnormal brain tissue to psychosurgery, the concept of causation in brain-mind discussions, the relation of violence control to the restoration of competence, and the question of consent. Many will not agree with Professor Shuman's positions (e.g., a "medical malpractice free market mechanism is likely to be better than . . . any other currently plausible alternative for the regulation of psychosurgery," p. 89), but no one will complain that he hides his personal prejudices. The book will reward the reader with significant and relevant discussions of social, legal, and political aspects of psychosurgery, but only if the reader survives lengthy passages on Wittgenstein, evolution, and the medical model.

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Books Received

This listing acknowledges the receipt of recent books. Books of particular interest to the readers of this journal will be reviewed as space permits, and copies of the reviews will be sent to the publishers. Books cannot be returned to the publishers.

The Ann Landers Encyclopedia A to Z: Improve Your Life Emotionally, Medically, Sexually, Socially, Spiritually. *Garden City, N.Y., Doubleday & Co., 1978, 1,182 pp., $17.50.*

Satan's Power: A Deviant Psychotherapy Cult, *by William Sims Bainbridge. Berkeley, Calif., University of California Press, 1978, 305 pp., $14.95.*

Brain's Clinical Neurology, 5th ed., *revised by Sir Roger Bannister, M.A., B.Sc., D.M. New York, N.Y., Oxford University Press, 1978, 496 pp., $18.95; $14.95 (paper).*

Werner Erhard: The Transformation of a Man; the Founding of est, *by William Warren Bartley III. New York, N.Y., Clarkson N. Potter (Crown Publishers, distributor), 1978, 279 pp., $10.00.*

The American Biomedical Network: Health Care Systems in America Present and Future, *edited by Stacey B. Day, M.D., Ph.D., D.Sc., Robert V. Cuddihy, B.S., M.B.A., and H. Hugh Fudenberg, M.D. New York, N.Y., Scripta Medica & Technica, 1977, 334 pp., no price listed (paper).*

Family Violence: An International and Interdisciplinary Study, *edited by John M. Eekelaar, LL.B., B.C.L., M.A., and Sanford N. Katz. Toronto, Ont., Canada, Butterworths, 1978, 572 pp., $15.95 (paper).*

How to Choose and Use the Right Therapist for You, *by Jean and Jim Erwin. Kansas City, Kan., Sheed Andrews and McMeel (Universal Press Syndicate), 1978, 112 pp., $3.95 (paper).*

The Psychological Basis of Ideology, *edited by Hans J. Eysenck and Glenn D. Wilson. Baltimore, Md., University Park Press, 1978, 312 pp., $29.50.*

Hello Sigmund, This Is Eric: Psychoanalysis and TA in Dialogue, *by Louis H. Forman, M.D., and Janelle Smith Ramsburg, R.N., M.S.W. Kansas City, Kan., Sheed Andrews and McMeel (Universal Press Syndicate), 1978, 241 pp., $12.95; $5.95 (paper).*

Women and Sex Roles: A Social Psychological Perspective, *by Irene H. Frieze, Jacquelynne E. Parsons, Paula B. Johnson, Diane N. Ruble, and Gail L. Zellman. New York, N.Y., W.W. Norton, & Co., 1978, 417 pp., $13.95.*

Wholistic Dimensions of Healing: A Resource Guide, *compiled and edited by Leslie J. Kaslof. Garden City, N.Y., Doubleday & Co., 1978, 294 pp., $7.95 (paper).*

Action with the Elderly: A Handbook for Relatives and Friends, *by Kenneth M.G. Keddie, M.B., Ch.B., D.P.M. Oxford, England, Pergamon Press, 1978, 159 pp., no price listed (paper).*

Mind Over Math, *by Stanley Kogelman, M.S.W., Ph.D., and Joseph Warren, Ph.D. New York, N.Y., Dial Press, 1978, 239 pp., $8.95.*

Poetry in the Therapeutic Experience, *edited by Arthur Lerner, Ph.D. New York, N.Y., Pergamon Press, 1978, 142 pp., $10.00.*

Marriage and Marital Therapy: Psychoanalytic, Behavioral and Systems Theory Perspectives, *edited by Thomas J. Paolino, Jr., M.D., and Barbara S. McCrady, Ph.D. New York, N.Y., Brunner/Mazel, 1978, 566 pp., $25.00.*

The Family Roots of Adolescent Delinquency, *by Joseph F. Perez, Ph.D. New York, N.Y., Van Nostrand Reinhold Co., 1978, 231 pp., $14.95.*

American Psychoanalysis: Origins and Development. The Adolf Meyer Seminars, *edited by Jacques M. Quen, M.D., and Eric T. Carlson, M.D. New York, N.Y., Brunner/Mazel, 1978, 209 pp., $15.00.*

The Psychopath: A Comprehensive Study of Antisocial Disorders and Behaviors, *edited by William H. Reid, M.D., M.P.H. New York, N.Y., Brunner/Mazel, 1978, 329 pp., $17.50.*

Cognitive Defects in the Development of Mental Illness, *edited by George Serban, M.D. New York, N.Y., Brunner/Mazel, 1978, 390 pp., $25.00.*

Drug Treatment in Psychiatry, 2nd ed., *by Trevor Silverstone, M.A., D.M., and Paul Turner, M.D., B.Sc. Boston, Mass., Routledge & Kegan Paul, 1978, 250 pp., $16.75; $9.75 (paper).*

The Analysis of Practical Skills: The Study of Real Skills, Vol. 1, *edited by W.T. Singleton, M.A., D.Sc. Baltimore, Md., University Park Press, 1978, 322 pp., $24.50.*

Psychoanalysis and Language. Psychiatry and the Humanities, Vol. 3, *edited by Joseph H. Smith, M.D. New Haven, Conn., Yale University Press, 1978, 394 pp., $20.00.*

Psychosocial Therapy, *by Francis J. Turner. New York, N.Y., Free Press (Macmillan Publishing Co.). 1978, 240 pp., no price listed.*

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Model Law on Confidentiality of Health and Social Service Records

OFFICIAL ACTIONS

Model Law on Confidentiality of Health and Social Service Records

This document was approved by the Board of Trustees at its September 1977 meeting and by the Assembly Executive Committee at its February 1978 meeting. It was prepared by the Task Force on Confidentiality of Children's and Adolescents' Clinical Records[1] and the Committee on Confidentiality.[2]

1. *Scope:*

All confidential information is subject to the provisions of this Act. Except as hereinafter provided, or otherwise specifically required by federal, state or local law, no person shall, without the authorization of the patient/client or his/her authorized representative:

(a) Disclose or transmit any confidential information together with a patient/client identifier to any person, or
(b) Disclose or transmit a patient/client identifier to any person, or
(c) Disclose or transmit confidential information if the person disclosing or transmitting it has reason to believe that the recipient may have a patient/client identifier for such information.

2. *Definitions As Used in This Act:*

(a) "Confidential information" means:
  (i) The fact that a person is or has been a patient/client;
  (ii) Information transmitted in confidence between the patient/client and service provider in the course of service provision;
  (iii) Information relating to diagnosis, facts necessary to the provision of services, or treatment, transmitted in confidence between members of the patient/client's family and the service provider;
  (iv) Information relating to diagnosis, facts necessary to the provision of service, or treatment, transmitted between any of the persons specified in (a) (ii) and (iii) above, and persons who participate in the accomplishment of the objectives of diagnosis, fact-finding, or service under the supervision of, or in cooperation with, the service provider;
  (v) Any diagnosis or opinions formed by the service provider regarding the patient/client's physical, mental or emotional condition;
  (vi) Any advice, instructions or prescriptions issued by the service provider in the course of diagnosis, treatment, or provision of other service;
  (vii) Any summary, resumé or characterization of the substance, or any part of the information described in sub-sections (f), (i) through (v) of this section 2; and
  (viii) Any record, recording, or notation of information described in subsection (f), (i) through (vi) of this section 2, in whatever form and by whatever means recorded or noted.
  (ix) Personal information governed by the School Student Records Act is hereby excluded from the application of this statute.
(b) "Patient/client" means a person who consults, is examined, interviewed, treated, or is otherwise served to some extent by a service provider, or a clinical researcher, as hereinafter defined, with regard to a medical, mental, or emotional condition or social deprivation or dysfunction.
(c) "Patient/client identifier" means:
  (i) The patient/client's name or other descriptive data from which a person well acquainted with the client might, with reasonable certainty, recognize such patient/client as the described person, or
  (ii) A code, number, or other means to be used to match the patient/client with certain confidential information regarding him/her.
(d) "Authorized representative" means:
  (i) A person empowered by the patient/client to assert or to waive the confidentiality, or to disclose or consent to the disclosure of confidential information, as established by this Act. Such person shall not, except by explicit authorization, be empowered to waive confidentiality or to disclose or consent to the disclosure of, confidential information;
  (ii) If the patient/client is incompetent to assert or waive his rights hereunder, or is in an apparently life threatening or emergency situation, a guardian or conservator, except that pending appointment of such guardian or conservator, the nearest avail-

[1]The Task Force on Confidentiality of Children's and Adolescents' Clinical Records (1976–1977) included Frank Rafferty, M.D., chairperson, John Looney, M.D., Herbert Sacks, M.D., and Lenore Petty, M.D., Falk Fellow. Of Counsel: Sandra Nye, J.D., M.S.W.

[2]The Committee on Confidentiality (1976–1977) included Jerome Beigler, M.D., chairperson, Ben Bursten, M.D., Maurice Grossman, M.D., Alan McLean, M.D., Don Mosher, M.D., Herbert Sacks, M.D., Hugo Van Dooren, M.D., and Robert Friedman, M.D., Falk Fellow. Of Counsel: Sandra Nye, J.D., M.S.W.

able relative of such patient/client may maintain or waive the confidentiality;
(iii) If the patient/client is deceased, his personal representative or next of kin or
(iv) If the patient/client is less than twelve (12) years of age, his parent or other custodian or guardian.

(e) "Diagnosis, fact-finding, or provision of service" includes observations made for purposes of same and all efforts to prevent, ameliorate, or otherwise overcome the effects of medical, mental or emotional disorders or social deprivation or dysfunction.

(f) "In confidence" means, private disclosures made or intended to be made, so far as the discloser is aware, to no other persons except
(i) The intended recipient;
(ii) Those who are present to further the interest of the patient/client in consultation, examination or interview, diagnosis, treatment, or other service provided;
(iii) Those to whom disclosure is reasonably necessary for the transmission of the information or the accomplishment of diagnosis or treatment, including members of a therapy group of which the patient/client is a participant, and members of the client's family; supervisors or other persons participating in consultation, examination or interview, diagnosis, or treatment, or other service provided under the direction of the provider; third-party payers; and
(iv) Persons reasonably believed to be engaged in good faith in training programs relevant to the activities of the service provided.

(g) "Person" means any natural person, corporation, association, partnership, and any state, local or federal government, or any agency or other part thereof, including a court.

(h) "Service provider" means any person authorized by statute to provide medical, psychological, psychotherapeutic, psychoanalytic, child welfare and/or other social services; any person reasonably necessary for evaluation, diagnosis, consultation, treatment, or care under the supervision of the provider; and, any person reasonably believed by the client to be so authorized or engaged.

3. *Authorized Disclosures:*

(a) Consent may be given by a patient/client who is twelve (12) years of age or over or by his authorized representative, for the transmission or disclosure of confidential information. Such consent shall be effective only if it is in writing and signed, and also specifies the nature and content of the information to be disclosed, to what person such information may be transmitted or disclosed, and to what use the transmitted or disclosed information may be put. Such specifications shall constitute the limits of the authorization. Every person requesting such authorization shall inform the patient/client or authorized representative that refusal to give such consent will in no way jeopardize his right to obtain present or future service, except where and to the extent disclosure is necessary for service to said patient/client, or for the substantiation of a claim for payment from a person other than the patient/client. The patient/client, or his authorized representative, may withdraw any such consent at any time in writing transmitted to and received by the person authorized to receive such confidential information. Upon receipt of such withdrawal, the person previously authorized to receive said information shall exercise reasonable care in promptly notifying all persons who had previously transmitted information on the basis of said consent, or who might reasonably be expected to do so in the future, that the prior consent has been withdrawn. If consent had been obtained by a person other than the person thereby authorized to receive said information, the person who obtained said consent shall, upon request, promptly, and in the exercise of reasonable care, assist the patient/client in ascertaining the correct name and address to which the withdrawal should be sent. Withdrawal of such consent shall have no effect upon disclosures made prior thereto.

(b) If the patient/client is under twelve (12) years of age or incompetent, consent may be given for the transmission or disclosure of confidential information by the patient/client's authorized representative.

4. *Disclosures Without Authorization:*

Consent from the patient/client shall not be required for the disclosure or transmission of confidential information in the following situations, as specifically limited:

(a) *Within the service-providing facility:* Confidential information may be disclosed to other individuals employed by the service provider, and to officially designated auditors and surveyors for accreditation, when and to the extent to which the performance of their duties in employment, audit or accreditation requires that they have access to such information. For purposes of this subsection (a), (i) persons engaged in good faith in training programs at a service providing facility and their clinical supervisors are to be considered as being employed by the service provider and may have access to such records and information to the extent reasonably required in their training and duties, but, (ii) individuals employed by the service provider or audit or who are involved in financial audit, preparation of bills or who are otherwise engaged in the collection of charges for services to a patient/client shall not, by virtue thereof alone, have access to confidential records and information, except with respect to names, addresses, and other information essential to the preparation and submission of bills and claims for payment of charges for services to a patient/client.

(b) *Clinical supervisors or trainers not employed by the service-providing facility:* Confidential information may be disclosed to supervising or training clinicians by service providers who are in training or supervision under a clinician or bona fide training program, whether or not such supervising clinicians are employed by or affiliated with the service-providing facility. For purposes of this subsection (b), the clinical supervisor or trainer receiving such confidential information shall bear the same position and responsibility with regard to the protection thereof as the service provider.

(c) *Protection from serious injury or disease; The Abused and Neglected Child Reporting Act:* Confidential information may be disclosed, (i) in accordance with the provisions of the Abused and Neglected Child Reporting Act; and (ii), when the statute creating a legislative commission delegates authority to study the needs of minors or incompetents, and to promote services for

the protection of the rights and interest of minors or incompetent persons who are in need of, or provided with medical, social and mental health services; subject, however, to guidelines established by the director of the agency providing the service with respect to the validity of the request for material and to the proper precaution as to its confidentiality and use; (iii) when and to the extent a treating or diagnosing service provider, in his sole discretion, determines that such disclosure is necessary to initiate or continue civil commitment proceedings under the laws of this state or to otherwise protect the patient/client or other person against a clear, imminent risk of serious physical or mental injury or disease or death being inflicted upon the patient/client, or by the patient/client on himself or another; and (iv) when and to the extent such is in the sole discretion of the treating or diagnosing clinician, necessary to the provision of emergency medical care to a patient/client who is unable to assert or waive his rights hereunder and there is no relative or other third party available to give consent. Any person, institution, or agency, under this Act, participating in good faith in the making of a report under the Abused and Neglected Child Reporting Act, or in the disclosure of confidential information otherwise in accordance with this provision, shall have immunity from any liability, civil, criminal or otherwise, that might result by reason of such action.

(d) *Billing and claims:* Information supplied by a service provider to persons involved in the billing for, or collection of, charges for services, shall be limited to names, addresses, dates on which services were performed, and the amount of charges for such services, and shall not otherwise indicate the nature of the conditions for which services were provided. In the event of a claim in any civil action for payment for services, no other confidential information except names, addresses, the dates on which services were rendered, and the amount of charges for such services shall be disclosed in pleadings and motions, except to the extent necessary (i) to respond to a motion of the client for greater specificity, or (ii) to dispute a defense or counterclaim.

(e) *Patient/client-litigant exception:* Except as provided in paragraph (ii) of this subsection (e)

(i) Confidential information may be disclosed in a civil or administrative proceeding in which the client introduces his physical, mental or emotional condition or any aspect of his diagnosis or treatment for such a condition as an element of his claim or defense if and only to the extent the court in which the proceedings have been brought, or, in the case of an administrative proceeding, the court to which an appeal or other action for review of an administrative determination may be taken, finds, after *in camera* examination of testimony or other evidence, that it is relevant, probative, not unduly prejudicial or inflammatory, and otherwise clearly admissible; that other satisfactory evidence, such as the results of a present examination of the patient/client by an examining clinician other than the service provider, or stipulations of fact between the parties, are demonstrably unsatisfactory as evidence of the facts sought to be established by such evidence; and that disclosure is more important to the interests of substantial justice than protection from injury to the provider-patient/client relationship or to the patient/client or others whom disclosure is likely to harm. No confidential communication between a service provider and a patient/client shall be deemed relevant for purposes of this sub-section, except the fact of treatment, the cost of treatment and the ultimate diagnosis unless the party seeking disclosure of the communication clearly establishes in the trial court a compelling need for its production.

(ii) This subsection (e) shall not apply to preclude the assertion of the confidentiality privilege as to confidential information disclosed in the course of any treatment of an abnormal mental or emotional condition

(a) In any action brought or defended under the Divorce Act, or

(b) In any action for damages for pain and suffering that does not include a claim for the treatment of such abnormal mental or emotional condition.

(iii) Confidential information or records may be disclosed in a civil proceeding after the patient/client's death when the patient/client's physical or mental condition has been introduced as an element of a claim or defense by any party claiming or defending through or as a beneficiary of the patient/client, provided the court finds, after *in camera* examination of the evidence, that it is relevant, probative, and otherwise clearly admissible; that other satisfactory evidence, including stipulations of fact between the parties, is not available regarding the fact sought to be established by such evidence; and that disclosure is more important to the interests of substantial justice than protection from any injury which disclosure is likely to cause.

(iv) In the event of a claim made or an action filed by a patient/client, or, following the patient/client's death, by any party claiming as a beneficiary of the patient/client, for injury caused in the course of diagnosis or treatment of said patient/client, the service provider and other persons whose actions are alleged to have been the cause of injury may disclose pertinent confidential information to an attorney or attorneys engaged to render advice about and to provide representation in connection with such matter and to persons working under the supervision of such attorney or attorneys, and may testify as to the said information in any judicial or discovery proceeding for the purpose of preparing and presenting a defense against such claim or action.

(f) *Court-ordered examination:* Communications made to or diagnoses and opinions made by a service provider in the course of examination ordered by a court for good cause shown may, if otherwise relevant and admissible, be disclosed in a judicial or administrative proceeding in which the patient/client is a party or in appropriate pretrial proceedings, provided such court has found that the patient/client has been as adequately and as effectively as possible informed before submitting to such examination that such communications, diagnoses and opinions would not be considered confidential or privileged. Such communications,

diagnoses and opinions shall be admissible only on issues germane to the said proceedings and involving the patient/client's physical or mental condition.

5. *Waiver:*

(a) Particular items of confidential information may be disclosed in judicial proceedings if the court in which the proceedings have been brought finds that the information is relevant and otherwise admissible and that the patient/client or his authorized representative has, without coercion, knowingly waived confidentiality by disclosing, or consenting to disclosure of, the substance of such particular information. In the case of an administrative proceeding, prior to disclosure of confidential information, any dispute as to the issue of waiver of confidentiality shall be referred for determination to the court to which an appeal from the administrative ruling may be taken.

(b) Disclosures that are privileged, disclosures made in the course of obtaining payment for treatment and related services, and disclosures made in the interest of accomplishing a purpose for which the psychotherapist was consulted are not waivers of confidentiality.

(c) For purposes of this section 5, failure by the patient/client or his authorized representative to assert the confidentiality of information in any proceeding in which he has the legal standing and opportunity to do so shall be deemed a consent.

6. *Rulings on Claims of Confidentiality:*

(a) In a ruling on an assertion of confidentiality to prevent disclosure in judicial or administrative proceedings, the court may not require disclosure of information asserted to be confidential under the Act in order to rule on such assertion.

(b) When neither the patient/client nor his authorized representative are parties to an administrative or judicial proceeding or they otherwise lack the opportunity to assert confidentiality, (i) any person asked in administrative or judicial proceedings to disclose confidential information may assert its confidentiality; and, (ii) the presiding officer on his own motion or the motion of any party shall exclude such information. Such presiding officer may not exclude information under this section 6 if, (i) he is otherwise instructed by the client or his authorized representative to permit disclosure; or, (ii) the proponent of the evidence establishes that there is no person authorized to assert confidentiality in existence.

(c) Whenever confidentiality is asserted under this Act in a judicial or administrative proceeding, the party opposing such assertion shall have both the burden of going forward with evidence and the burden of proof with regard to issues of whether confidentiality has been waived and whether any relevant transmissions of information were not made in confidence.

(d) No person shall be held in contempt for failure to disclose confidential information unless he has failed to comply with a court order, a legislative subpoena or an order of an administrative hearing that he disclose such information.

7. *Prescriptions:*

Nothing in this Act shall be construed as limiting or interfering with state and federal regulation and monitoring of the handling and dispensing of prescription drugs; otherwise, however, prescriptions for drugs shall be considered confidential information and subject to the provision of this Act.

8. *Research:*

Persons engaged in research may have access to confidential information that identifies the patient/client where needed for such research, provided no records thereof shall be removed from the service-providing facility that prepared them. Data that do not identify patient/clients or coded data may be removed from a service-providing facility provided the key to such code shall remain on the premises of the facility and no copies thereof are removed. Where the person engaged in research is to have access to confidential information, the research plan first shall be submitted to, and approved by, an appropriate Research Review Committee and by the director of the service-providing facility or his designee. The service-providing facility, together with the person doing the research, shall be responsible for the preservation of the anonymity of the patient/clients and shall not disseminate data that identify a patient/client except as provided by this Act.

9. *Mandatory Cautions:*

(a) All nonoral disclosures of confidential information shall bear the following statement: "The protection of the confidentiality of information contained herein is required under (chapter) of laws of the State of ( ) which provides for damages and penalties for violations. This material shall not be transmitted to anyone without consent or other authorization as provided in the aforementioned statute." A copy of the pertinent consent form specifying to whom and for what specific use such communication or record is disclosed or transmitted, or a statement setting forth any other statutory authorization for disclosure or transmittal and limitations imposed thereon, shall accompany all such nonoral disclosures. In cases of oral disclosure, the person disclosing confidential information shall inform the recipient that such information is confidential under the laws of this state.

(b) Service providers shall ensure that all persons in their employ or under their supervision are aware of their responsibilities to maintain the confidentiality of information protected by this Act and of the existence of penalties and civil liabilities for violation of this Act.

10. *Civil Remedies and Criminal Penalties:*

(a) Any person aggrieved by a violation of this Act may petition the court of common pleas for the county in which he or the alleged violator resides or in which such violation occurred, for appropriate relief, including temporary and permanent injunctions, and such petition shall be first priority with respect to assignment for trial. Such aggrieved person may also prove a cause of action for general or special damages, or both, and, in cases of willful or grossly negligent violations, punitive damages.

(b) A willful or grossly negligent violation of this Act shall be punishable as a Class C misdemeanor. For purposes of this section, in cases of willful disclosure of confidential information, each such disclosure of information pertaining to any one person shall constitute a separate violation.

11. *Employee Discipline:*

(a) Any state, county or local government employee and any employee of a service-providing facility operated under contract to a state, county or local government or department or agency thereof, who repeatedly, willfully or through gross negligence violates this Act, shall be dismissed from employment, or, in the case of mitigating circumstances deemed adequate by the employer, appropriately disciplined and transferred to a position, if available and otherwise suitable, outside a service-providing facility and involving no access to confidential information.

(b) Negligent, nonrepetitive violations of this Act shall render such employees subject to appropriate disciplinary action.

(c) In the course of any disciplinary or dismissal actions against such employees, confidential information shall not be used except to the extent necessary to comply with principles of fair notice and hearing, and patient/client identifiers shall be removed from any such information prior to its use in such proceedings.

(d) All contracts between private persons and any state, county or local government or department or agency thereof involving access by such private persons or their employees, representatives, agents or subcontractors shall include a provision setting forth requirements of this section. Failure to include this clause in any such contract shall not limit the operation of this section.

12. *Patient/Client Access to Information:*

(a) Except as provided in (c) and (d) of this section 12, upon request of a patient/client, a service provider shall, within thirty days following the request, allow the patient/client access to his service record.

(b) The service provider shall establish procedures that: 1) allow a person to purchase copies of his record at a reasonable cost, not exceeding the actual cost of duplication to the service provider; 2) allow a person to contest the accuracy, completeness or relevancy of the record content; 3) allow information contained therein to be corrected on request of the person when the service provider concurs in the proposed correction; 4) allow a person who believes that the service provider maintains inaccurate or incomplete information concerning him to add a statement to the record setting forth what he believes to be an accurate or complete version of those personal data. Such a statement shall become a permanent part of the service provider's personal data system, and shall be disclosed to any individual, agency or organization to which the disputed personal data are disclosed.

(c) If a service provider determines that disclosure to a person of medical, psychiatric or psychological data concerning him would be detrimental to that person, or that nondisclosure to a person of personal data concerning him is otherwise required by law, the service provider may refuse to disclose those personal data, and shall refuse disclosure where required by law. In either case, the service provider shall advise that person of his right to appoint another clinician of his own choice as "clinical mediator" to have access to the record. The "clinical mediator" may, upon review of the record, disclose the record to the person, offer to interpret the contents of the record to the person, or may refuse to disclose. If the "clinical mediator" determines against disclosure and the person is unwilling to accept an interpretation of his record, the service provider shall advise the person of his right to seek judicial relief.

(d) If disclosure of personal data is refused by a service provider under this section 12, the person aggrieved thereby may, within 30 days of such refusal, petition the court of common pleas for the county or judicial district in which he resides or in which the service provider resides or practices, for an order requiring the service provider to disclose the personal data. The court, after hearing and an *in camera* review of the personal data in question, shall issue the order requested unless it determines that such disclosure would be detrimental to the person or is otherwise prohibited by law, or may alternatively authorize disclosure to a designated clinician or attorney.

(e) If the person is under 12 years of age, his parent or other custodian shall have the rights set forth in this section 12 on behalf of that person. Further, if the person, in consequence of physical or mental incapacity, shall have been placed under guardianship, his guardian shall have the same rights set forth in this section 12 on behalf of that person.

13. *Records and Information Pertaining to Minors:*

(a) All confidential information pertaining to the provision of health and social services to a minor shall be deemed confidential, and no disclosure of such information shall be made to the child's parent or any other person, except:

(i) If a minor who is twelve (12) years of age or older consents in writing;

(ii) As provided by sections 4 and 12 (c) hereof;

(iii) If the service provider obtains information that he or she believes requires action to prevent serious harm to the minor or another person, he or she may disclose that information to the child's parent, guardian or legal custodian, or as appropriate under the provisions of the Abused and Neglected Child Reporting Act;

(iv) All records shall be available to the child's counsel of record and professional and paraprofessional persons associated with the child's counsel and to staff members of the Juvenile Court.

14. *Personal Notes: Special Limitations on Disclosure:*

(a) A service provider is not required to but may, to the extent he or she determines it necessary and appropriate, keep personal notes regarding a patient/client wherein he or she may record:

(i) Sensitive information disclosed to him or her in confidence by other persons on condition that such information would never be disclosed to the patient/client or other persons;

(ii) Sensitive information disclosed to him or her by the patient/client that would be injurious to the patient/client's relationships to other persons; and

(iii) The service provider's speculations, impressions, hunches and reminders.

(b) Such personal notes are the work product and personal property of the service provider and shall not be subject to discovery in any judicial, administrative or legislative proceeding or any proceeding preliminary thereto.

15. *Group Health and Life Insurance:*

No person shall demand or request that information as to medical and mental health history, condition and treatment on group health and group life insurance applications, questionnaires and claim forms or any copy thereof or information therefrom, be submitted to an insurance company regarding the insurance coverage of a resident of this state, (a) to or through any member or representative of the group, or (b) to or through the employer or any representative or agent of the employer of the persons covered by such policy, but only directly from the insured or covered persons or their designees and providers of covered health care services or their designees. Insurance companies with group policies covering persons residing in this state and the representatives and agents of such companies, in accordance with rules and regulations to be promulgated by the Director of Insurance, shall, within 120 days from the effective date of this Act (a) take measures to advise group members, employers of group members and representatives and agents of such employers involved in the administration of such policies of the requirements of this section; (b) cause notices regarding this section to be printed prominently on all newly issued or renewal policies, on printed materials intended to be provided to group members and their employers regarding such policies and on all applications, questionnaires, claims and similar forms to be submitted by or on behalf of covered persons and by persons providing covered health care services. For purposes of this section, covered health care services shall include diagnostic and evaluative services. This section shall not apply to applications for life insurance benefit payments.

16. *Health and Life Insurance: Prohibition on Requirement of Consent to Disclosure by Insurance Company to Others:*

No insurance company or any employee, representative or agent thereof shall require of any person residing in this state as a condition of the issuance, continuation, renewal or reinstatement of life, health, accident, medical, hospitalization or similar insurance policy or as a condition of paying any benefits thereunder that an applicant, insured or covered person, or any person acting in his behalf, authorize or agree to authorize such insurance company to disclose or re-disclose confidential information with patient/client identifiers to persons other than itself. Advice that no such requirement may lawfully be imposed must be given in conjunction with any request for such authorization.

17. *State, County and Local Information Systems:*

(a) *Official inspections:* Nothing in this Act shall be construed as prohibiting any state, county or local government official from performing any audits, investigations or inspections of health or social service facilities in the state as required or authorized by law, *provided* that the performance of such duties shall not entail removal from any such facility of any confidential information with client identifiers or any codes or keys to electronically processed information.

(b) *Statistical reports:* Nothing in this Act shall be construed as prohibiting the issuance of statistical reports and similar anonymous data regarding the operations of health or social service facilities.

(c) *Electronic data processing:*

(i) No electronically processed data of confidential information with patient/client identifiers shall be recorded on equipment outside a mental health facility except in accordance with this section.

(ii) Confidential information regarding current patient/clients may be recorded on electronic data-processing equipment outside a mental health facility only if:

1. Such information is encoded by means that make it impossible for persons other than data-processing personnel within such facility to discern the identity of individual patient/clients;

2. The encoding means or devices by which a patient/client can be identified are delivered, within 60 days after a client is discharged or otherwise ceases to participate in diagnosis or treatment, by data-processing personnel within such facility to a person or persons under the direct supervision of the facility director, which person or persons are strictly denied access to the electronic data-processing equipment and are responsible for the safekeeping of such encoding means or devices and the denial of access thereto to all persons except as provided in subsection (iii) of this section 17;

3. The encoding means or devices by which a former patient/client can be identified may be returned to data-processing personnel for purposes of reactivating access to confidential information stored on electronic data-processing equipment when and only when, (1) the patient/client to which such information pertains has reentered diagnosis or treatment at such facility, or (2) a request for confidential information that may be honored under the provisions of this Act has been received; and

4. No later than 5 years after a patient/client has been discharged or has otherwise ceased to receive services at such facility, or in the case of a minor receiving service that was terminated during his minority, no later than 5 years after attaining his majority, either said encoding means or devices pertaining to such patient/client shall be destroyed, or, (2) all electronically processed data pertaining to such patient/client shall be returned to data personnel at such facility. Those facilities planning long-term epidemiological research may request under the research provisions of this statute special informed consent from the patient/client or authorized representative to maintain the patient/client's records for an extended period of time. Treatment may not be denied for failure to consent. The patient/client may at any time cancel consent without prejudice.

(iii) Such encoding means or devices may be disclosed (aa) to the extent necessary for auditors regularly employed by the state to inspect electronic data equipment to ensure strict and complete compliance with this Act, *provided* that such inspections shall not involve the removal of such encoded means and devices, or copies or other reproductions thereof from a mental health facility, (bb) or to the extent required for a fair hearing in connection with the dismissal of an employee charged with violating this Act, and (cc) to the extent necessary for use in a civil or criminal action arising out of violations of this Act.

(d) *Indigency investigations:* Any agency of state, county or local government charged with responsibility to investigate or audit claims of indigency, hardship or sim-

ilar status whereby individuals may receive health or social services without charge or on the basis of reduced charges, shall maintain the confidentiality of the patient/clients in the conduct of such investigations or audits and, upon the completion thereof, shall forward a report to the facility or agency for which prepared and shall keep no record of such investigation by which any patient/client can be identified.

18. *Disclosures Required in Federally Funded Programs:*

(a) Confidential information may be disclosed to federal departments and agencies to the extent required under federal law to obtain reimbursement for diagnosis, treatment and other social services under federally funded programs for review and audit that are a requisite for participation in federally funded programs.

(b) Any organization or agency designated under federal law to perform such reviews or audits of the cases of patient/clients who are residents of this state shall maintain the confidentiality of confidential information, shall not disclose confidential information except to the extent required by federal law, and shall destroy the means by which patient/clients can be identified in such information and records containing such information at the earliest opportunity consistent with the requirements of federal law.

(c) To ensure that confidential information regarding citizens and residents of this state is afforded maximum protection consistent with the provisions of this Act, the directors of each state code department delivering health or social services shall promulgate regulations that specify the minimum information required pursuant to subsections (a) and (b) of this section 18 and disclosures in excess thereof shall constitute violations of this Act. In the event a demand for confidential information in excess of that provided for in such regulations is made on any health or social service provider in this state upon pain of disallowance of reimbursement or other benefits, such provider shall immediately refer the matter to the director of the appropriate state code department or his designee, who shall, on behalf of such provider, attempt a resolution of the matter either by negotiation or appropriate court action, or by authorizing said provider to disclose if he determines that disclosure is required by federal law. Disclosures pursuant to the preceding sentence shall not constitute violations of this Act if all reasonable measures to assure confidentiality are taken.

(d) The director of each state code department delivering health or social services shall prepare written notices describing the requirements under any federally funded programs for the disclosure of confidential information and the purposes for such access; and he shall promulgate regulations establishing procedures whereby each person being provided care or other services for which reimbursement will be sought through a federally funded program involving such disclosure will be given such notice at the outset of the delivery of services. Such notices shall provide advice regarding the individual's option not to receive treatment on a basis whereunder such disclosures are required.

Commentary on Model Law on Confidentiality of Health and Social Service Records

BY SANDRA NYE, J.D., M.S.W.

THIS MODEL ACT addresses and incorporates three allied legal concepts: confidentiality, privacy, and testimonial privilege. These are highly technical and frequently misunderstood. Privilege is an evidentiary concept which provides an exception to the general principle of law that courts have the right to every man's evidence. The concept has relevance only in context of the testimonial arena. A testimonial privilege (or shield law) permits those protected by it to withhold testimony or records, notwithstanding a subpoena (1). The right to privacy protects the individual from unsolicited, unwarranted intrusion in the conduct and affairs of his life—including the right to keep to himself information about himself (2). The right of a patient/client to confidentiality—and the concomitant duty of the care provider to maintain the patient/client's confidentiality—inheres in the contractual nature of the provider-patient/client relationship. Implied in the contract is a covenant not to disclose (3, 4).

Part of the complexity of this Model Act arises by reason of the legal principles on which it is based and which it intends to alter. In overturning existing law, every detail to be changed must be explicated. Any concept not expressly altered will remain the law. Thus, the Model Act must not only create and articulate novel concepts and procedures but must expressly eliminate or alter existing ones. For example, it is clearly established by case interpretation of statutory or common law privileges that the identity of a patient/client, the fact of the professional relationship, and purely clerical data—such as dates of service delivery—are not privileged communications and are, therefore, not protected from compelled disclosure in a legal proceeding (5). Further, the presence of a third person who is not a party to the provider-patient/client relationship (e.g., a family member or group member) "pollutes" any privilege that might have existed between the provider and the patient/client (6). Such technicalities as these have severely curtailed the efficacy of the communications privilege as a protection to psychiatric patients.

The Model Act is intended to serve as a basis for examining and proposing changes in local legislation. Although it represents the product of a thorough study of this subject and the combined thinking and expertise of many learned professionals, there are doubtless aspects that may be improved. The caveat to be kept in mind in working with the Act is that much of its phraseology consists of "terms of art." A knowledgeable lawyer should be consulted in any redrafting effort. In the limited space available for annotation, it is not possible to provide thorough explication and legal authority. We ask that the reader take on faith, for the time being, that what appear to be redundancy, prolixity, or tortured sentence construction translates in "legalese" as meaningful.

1. *Scope.* This Act defines both a communications privilege and a general law of confidential information. Thus, in addition to protecting confidential information from compelled disclosure in a judicial, legislative, or administrative proceeding, it also establishes a positive statutory duty on health and social service providers to maintain patient/client confidentiality. A salient principle of the Act is that all patient/client information given for the purpose of health care and social service delivery must be protected—irrespective of the nature of service delivered or the discipline or professional status of the care provider. This is a significant departure from most existing law, which makes irrational distinctions in protecting information as to care setting and care provider credentials. The needs of the patient/client for privacy and confidentiality do not differ according to whether the care provider is a social worker, a paraprofessional, or a psychiatrist. It should be public policy to mitigate fear of stigmatization (said to be the greatest barrier to seeking mental health services) so as to encourage individuals to seek necessary health and mental health care and social services.

2. *Definitions.* Explicit and detailed definitions are required to extend protection to categories of persons and data heretofore excluded by common law principles and certain statutes.

3. *Authorized Disclosures.* The nature of consent for disclosure is defined and delimited. This section clears up many existing ambiguities and procedural questions and outlines in detail the rights and duties of persons seeking disclosure, of care providers, and of patient/clients.

The minimum age at which consent may be given is established as 12 years. This is consistent with current child development theory recognizing the privacy and confidentiality needs of adolescents receiving mental health treatment and with existing federal and state legislation authorizing persons of this age to consent to certain types of treatment and other services. It is to be noted that parental notification will be automatic in most cases simply by reason of the parent's initiation of or involvement in the service delivered to the minor, or the minor's consent that disclosure be made to his/her parents. In the few cases in which the minor obtains services by reason of legal capacity to do so and does not authorize disclosure to his/her parents, the service provider who deems it necessary to notify the parents in order to protect the minor from serious injury or health hazard has the option under section 4 (c) to do so without the minor's consent or over his/her objection.

4. *Disclosures Without Authorization.* Although the underlying philosophy of the Act is that an individual has the right to control his/her private and confidential information, there is no question but that certain disclosures of such information are not only necessary but appropriate. In recognizing the "need to know," the following principles are essential:

a. Unauthorized disclosures should be kept to a mini-

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mum, consistent with the needs of the patient/client and the exigencies of service delivery.

b. The primary duty of the service provider is to the patient/client. There is no *duty* upon a provider to protect third parties, but there may be instances in which the provider deems it in the patient/client's interest to disclose confidential information to protect the patient/client or another from serious harm. This is left to the *sole discretion* of the service provider, who is immunized against liability for any such disclosure in good faith.

The Patient/Client-Litigant Exception (subsection 4 (e)) is an almost universal exception to existing privilege laws. The adage that "confidentiality is to be a shield, not a sword" decrees that a patient/client waives any privilege he may have as to material relevant to the trial of a lawsuit in which he is a party. This section is based in part on guidelines set forth by Judge Shirley Hufstedtler in her brilliant dissenting opinion in *Caesar v. Mountanos* (7). It further excludes from the rubric of "mental condition" an action for pain and suffering per se and incorporates a refinement of Illinois law eliminating the exception in divorce cases (8). Strictures are placed on disclosure after the death of the patient/client because the threat of disclosure after death may serve to inhibit communications, particularly in mental health care delivery. This subsection clears up a problem presently existing in some jurisdictions that holds that the privilege expires with its holder.

Court-ordered examination (subsection 4 (f)). Service providers are frequently called upon to conduct examinations for trial purposes. Although clinical skills are utilized in such examinations, the information conveyed is intended to be disclosed, and communications made in the course of such examinations are not protected by a privilege. The relationship of examiner to the subject of the examination is not that of service provider and patient/client. Disclosure should, however, be limited in accordance with the purpose for which it is made.

5. *Waiver.* At common law, a communication not expressly asserted by its holder is deemed waived. This section clarifies the question of waiver and obviates certain "accidental" or "resulting" waivers.

6. *Ruling on Claims of Confidentiality.* This section protects, to the extent possible, confidential information during a controversy as to its discoverability or admissibility in a judicial or administrative proceeding. The person seeking disclosure has the burden of establishing discoverability or admissibility. The court is empowered to protect confidential information in appropriate cases in which there is no person in existence who is otherwise empowered to do so.

7. *Prescriptions.* The patient/client's need for confidentiality is balanced by the interest of the community in regulating drugs.

8. *Research.* Although confidential information disclosed by a patient/client in the course of receiving health and social services is intended by the patient/client to be utilized for his direct benefit in service provision, the value to the community in accessibility of data for research and development cannot be overlooked. This section provides access to data under strictures that will protect the patient/client.

9. *Mandatory Cautions.* The disseminator of confidential information is charged with the instruction of employees and disclosees as to the protection of the information he/she is disclosing.

10. *Remedies.* Civil (equitable and legal) relief is authorized for any person aggrieved by violation of this Act. In some jurisdictions violations of confidentiality statutes have been discouraged by criminal sanctions as well. Alternatives are provided herein.

11. *Employee Discipline.* This section is intended to enable employers to take appropriate action against employees who willfully or by gross negligence violate the Act. Employers are said to fear that, without such provisions, civil service and union procedures will effectively preclude disciplinary action against erring employees who are employed under civil service regulations or union contracts and whose wrongful acts not only injure patient/clients but also expose the employers to liability.

12. *Patient/Client Access to Information.* As a general principle, it is held that every person should have access to any record of information about him. If information is to be disclosed pursuant to consent, such access is probably mandatory. (Consent is not valid unless informed; one cannot give informed consent to disclose unless he/she has knowledge as to the content of the disclosure.) Further, a patient/client should have an opportunity and right to seek correction or at least enter his/her opinion into a record that contains an error or with which he/she disagrees. Experience with allowing patient/client access to records has been positive. At the same time, some clinicians are concerned that there may be occasional instances in which the patient/client will be harmed by such access or the treatment process compromised. A procedure is established that, although possibly cumbersome, will allow access as a general rule and will offer protection in cases in which the service provider deems access to be against the interest of the patient/client.

13. See annotation to Section 3, supra.

14. *Personal Notes.* This concept has been discussed for a number of years as a device by which clinicians can protect records of certain types of data (9). As the public insistence on patient/client right of access to records has grown, some care providers have been concerned about the effect on the individual who discovers unknown facts about him/herself or others or is exposed to speculations and interpretations of the clinician. Some information—although clinically relevant—may be so "sensitive" as to warrant exlcuding it from the case record entirely. Notes kept by the care provider for use in research, teaching, or supervision may contain material that is inappropriate for the clinical record. Further, by reason of the nature of mental health treatment, certain record content may be highly prejudicial to the patient/client if disclosure is compelled in a judicial or other proceeding.

The "personal notes" concept borrows from a protection afforded "the work product of the attorney." Certain specific types of material can be recorded in the clinician's "personal notes," which are to be utilized by the clinician for his/her own purposes and may not be disclosed or discovered. Concern has been expressed by some administrators and attorneys that this device will afford lazy, careless, or unscrupulous care providers a means of "hiding their wrongdoings" or, at best, neglecting their recordkeeping. Although it is not possible to preclude wrongdoing on the part of any person who is so inclined, the language of the section is eminently plain and clear as to the limited usage of "personal notes." The value of the device in protecting patients and enhancing service provision is deemed to outweigh any possible misuse potential.

15. *Group Health and Life Insurance.* Although the insurance industry denies the charge, there is a widespread belief that it is the major perpetrator of privacy and confidentiality offenses. In any case, care providers and patient/clients have expressed outrage over insurance company demands for in-

formation. A particular source of concern has been the practice of claims processing through employers. This section establishes parameters for data collection by insurors and requires that information about these parameters be supplied to the insured.

16. *Health and Life Insurance Disclosure of Information.* Of the several insurance company practices objected to by providers and patient/clients, one of the most decried is the exchange and dissemination of data among insurors. The practice is widespread; the insurors insist they have a need and right to protect themselves. The many documented abuses of this practice lead to the conclusion that it must be curbed. The interests of the individual and the community in encouraging health care—and particularly mental health care—are held to outweigh the financial interest of the insurors.

17. *State, County and Local Information Systems.* Electronic data collection and storage is perceived as a threat to individual liberty (10). Despite the best intentioned efforts at safeguarding data banks, they are vulnerable to invasion and misuse. Rules for protecting patient/client privacy and confidentiality are established in the light of the realities of audit and accountability requirements.

18. *Disclosures Required in Federally Funded Programs.* The practical exigencies of federal funding are recognized in this section, with safeguards established for confidential information being disclosed. Notice to the patient/client relative to disclosures and options is required.

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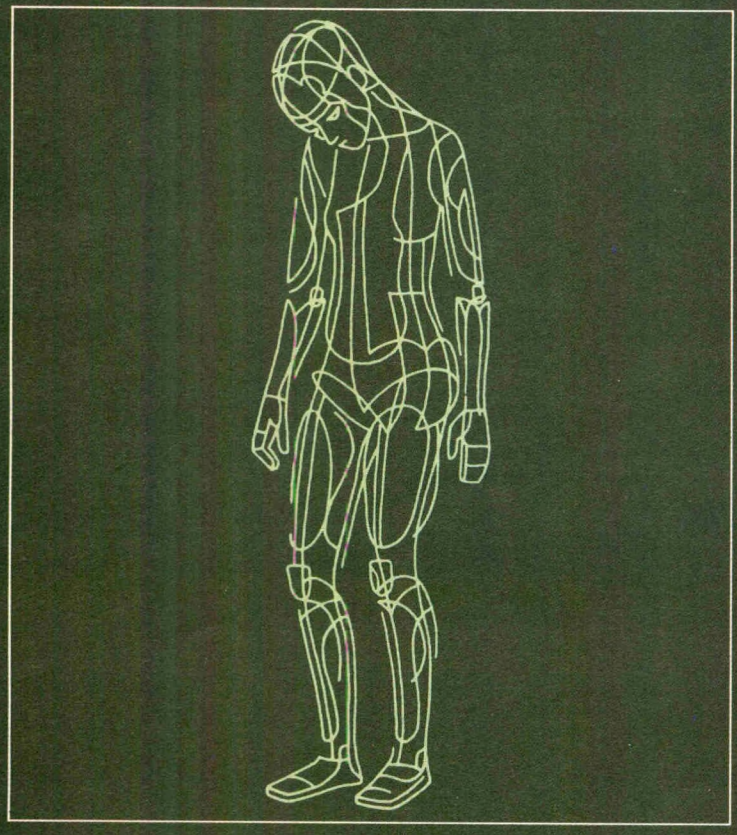
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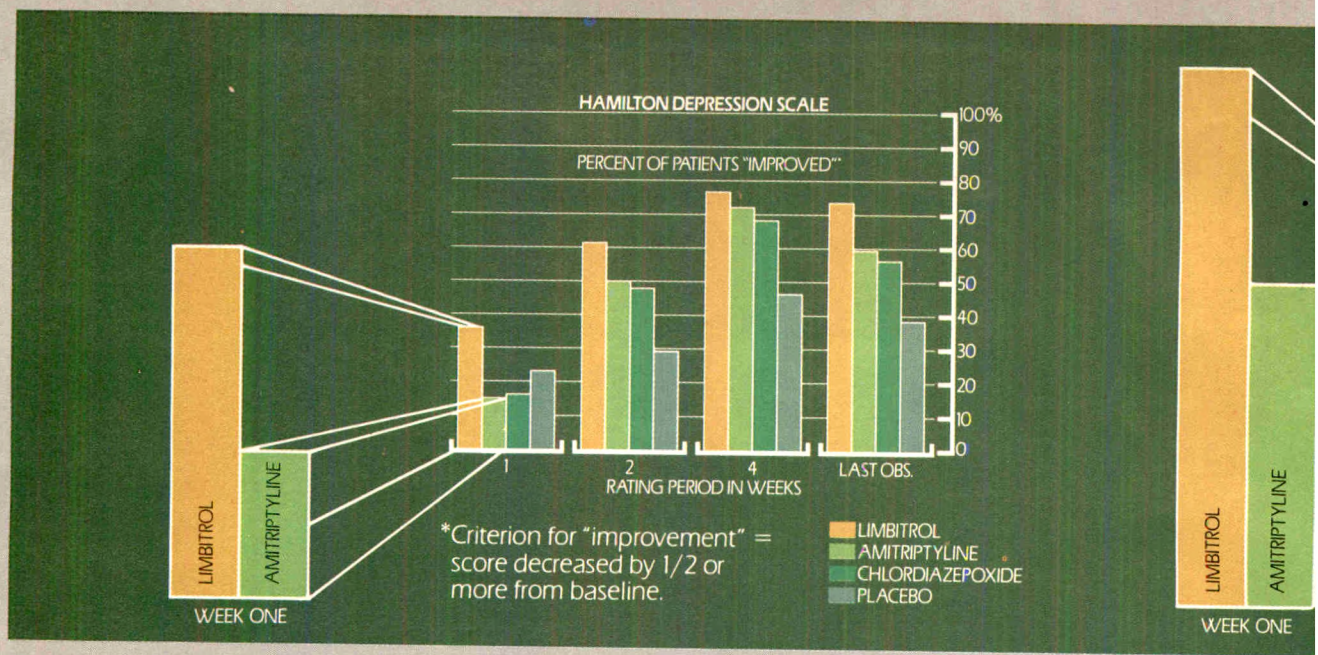


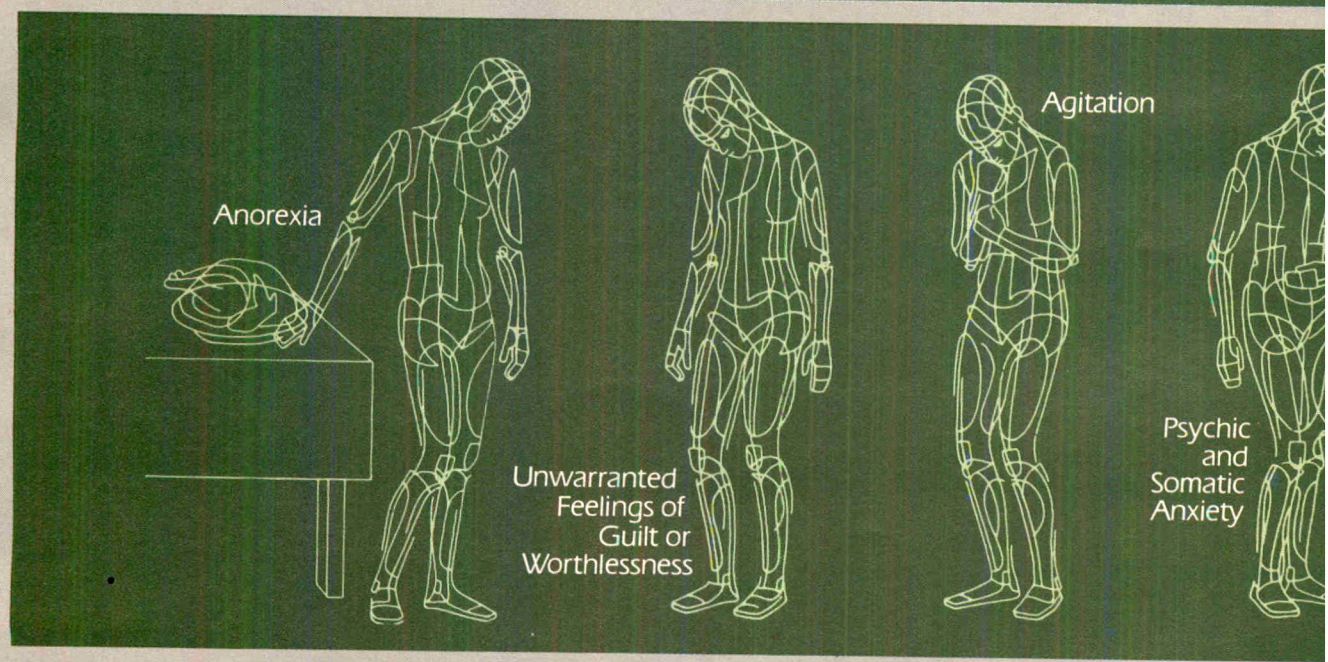
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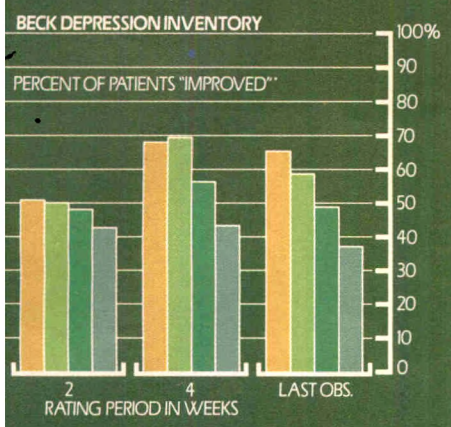
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HAMILTON DEPRESSION SCALE
PERCENT OF PATIENTS "IMPROVED"*
100%
90
80
70
60
50
40
30
20
10
0
1
2
4
LAST OBS.
RATING PERIOD IN WEEKS
LIMBITROL
AMITRIPTYLINE
WEEK ONE
*Criterion for "improvement" = score decreased by 1/2 or more from baseline.
LIMBITROL
AMITRIPTYLINE
CHLORDIAZEPOXIDE
PLACEBO
LIMBITROL
AMITRIPTYLINE
WEEK ONE


Anorexia
Unwarranted Feelings of Guilt or Worthlessness
Agitation
Psychic and Somatic Anxiety





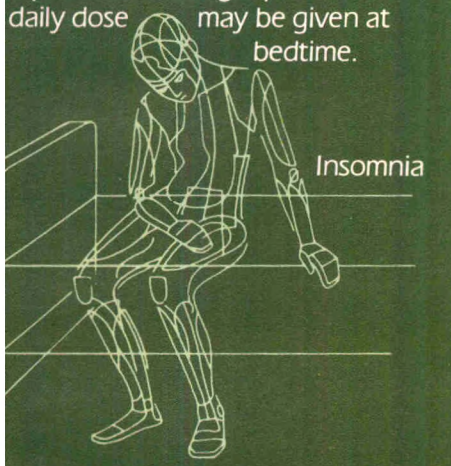
ion for "improvement" =
decreased by 1/3 or
from baseline.

Greater patient improvement

Within the first week—

twice as many patients achieved significant relief—sooner than with amitriptyline alone

omnia, a major target symptom, can
elieved sooner by taking advantage
ne sedative effect of Limbitrol. For
e patients, the larger part or all of
daily dose may be given at
bedtime.



Better relief of symptoms

Within the first week—

symptom severity was reduced to a greater extent than with amitriptyline alone

As measured by the Hamilton Depression Scale

- ☐ insomnia
- ☐ psychic anxiety
- ☐ somatic anxiety
- ☐ agitation
- ☐ suicidal ideation
- ☐ worthlessness

As measured by the Beck Depression Inventory

- ☐ pessimism
- ☐ dissatisfaction
- ☐ guilt
- ☐ social withdrawal
- ☐ anorexia

imbitrol®

ets 10-25 each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt)

ets 5-12.5 each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)

rapid relief of
derate to severe
pression and anxiety

Greater patient acceptance

☐ Despite a higher incidence of drowsiness, there was a lower dropout rate due to side effects with Limbitrol than with amitriptyline

(Limbitrol 4%, amitriptyline 13%, chlordiazepoxide 1%)

☐ The dropout rate due to therapeutic failure was lower with Limbitrol than with amitriptyline

(Limbitrol 2%, amitriptyline 6%, chlordiazepoxide 12%)

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ROCHE

How to Initiate and Maintain Therapy

Select dosage strength appropriate for each patient

- Limbitrol 10-25 is recommended for most patients
- Limbitrol 5-12.5 may be indicated for those who do not tolerate higher doses and for elderly patients

Specify daily dosage based on symptom severity

- An initial dosage of three tablets is recommended
- Dosage may be increased to six tablets or decreased to two tablets daily as necessary
- Once a satisfactory response is obtained, evaluate response, and if necessary, reduce dosage to smallest amount needed to maintain remission

Utilize dosage options to best accommodate individual patient needs

- T.I.D. or Q.I.D., familiar regimens best suited for patients who tolerate medication without undue drowsiness
- Two tablets one hour before bedtime and one tablet midday may minimize daytime drowsiness and help relieve a common target symptom—insomnia
- Entire dosage h.s. to take maximum advantage of the sedative effect

How to make each patient an informed patient

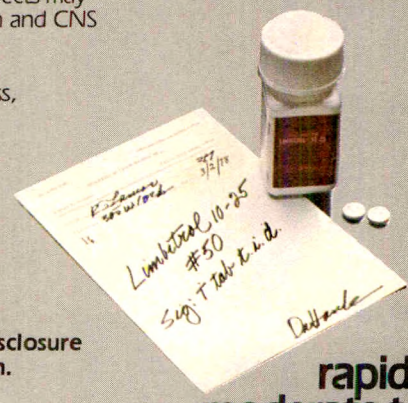
1. Discuss with patients the probability that they will experience drowsiness, especially during the first week.
2. Reassure your patients that drowsiness is one indication that the medication is working and that it may help alleviate their insomnia.
3. Encourage patients to report if drowsiness becomes troublesome so that, if necessary, dosage schedule can be adjusted or the symptom treated.
4. Caution patients about the combined effects with alcohol or other CNS depressants. Let them know that the additive effects may produce a harmful level of sedation and CNS depression.
5. Caution patients about activities requiring complete mental alertness, such as operating machinery or driving a car.
6. Warn pregnant patients and patients of childbearing age that the safety of Limbitrol in pregnancy has not yet been established.

Please see complete product disclosure for other pertinent information.

Limbitrol should not be used under t following circumstances

1. Hypersensitivity to benzodiazepines or tricyclic antidepressants.
2. Should not be given with an MAO inhibitor. To replace an MAO inhibitor with Limbitrol, discontinue MAO inhibitor for minimum of 14 days before cautiously ini ing Limbitrol therapy.
3. During the acute recovery phase follo ing myocardial infarction.

Limbitro



F
rapid relief
moderate to seve
depression and anxie

LIMBITROL® TABLETS
Tranquilizer–Antidepressant

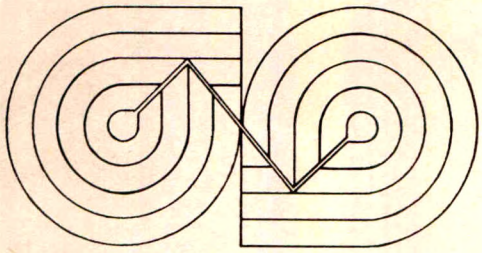
Before prescribing, please consult complete product information, a summary of which follows:
Indications: Relief of moderate to severe depression associated with moderate to severe anxiety.
Contraindications: Known hypersensitivity to benzodiazepines or tricyclic antidepressants. Do not use with monoamine oxidase (MAO) inhibitors or within 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use; then initiate cautiously, gradually increasing dosage until optimal response is achieved. Contraindicated during acute recovery phase following myocardial infarction.
Warnings: Use with great care in patients with history of urinary retention or angle-closure glaucoma. Severe constipation may occur in patients taking tricyclic antidepressants and anticholinergic-type drugs. Closely supervise cardiovascular patients. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, especially high doses. Myocardial infarction and stroke reported with use of this class of drugs.)

***Usage in Pregnancy:* Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Since physical and psychological dependence to chlordiazepoxide have been reported rarely, use caution in administering Limbitrol to addiction-prone individuals or those who might increase dosage; withdrawal symptoms following discontinuation of either component alone have been reported (nausea, headache and malaise for amitriptyline; symptoms [including convulsions] similar to those of barbiturate withdrawal for chlordiazepoxide).
Precautions: Use with caution in patients with a history of seizures, in hyperthyroid patients or those on thyroid medication, and in patients with impaired renal or hepatic function. Because of the possibility of suicide in depressed patients, do not permit easy access to large quantities in these patients. Caution patients about possible combined effects with alcohol and other CNS depressants and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).
Periodic liver function tests and blood counts are recommended during prolonged treatment.
Amitriptyline component may block action of guanethidine or similar antihypertensives.
Concomitant use with other psychotropic drugs has not been evaluated: sedative effects may be additive.
Discontinue several days before surgery. Limit concomitant administration of ECT to essential treatment. See Warnings for precautions about pregnancy. Limbitrol should not be taken during the nursing period. Not recommended in children under 12.
In the elderly and debilitated, limit to smallest effective dosage to preclude ataxia, oversedation, confusion or anticholinergic effects.
Adverse Reactions: Most frequently reported are those associated with either component alone: drowsiness, dry mouth, constipation, blurred vision, dizziness and bloating. Less frequently occurring reactions include vivid dreams, impotence, tremor, confusion and nasal congestion. Many depressive symptoms including anorexia, fatigue, weakness, restlessness and lethargy have been reported as side effects of both Limbitrol and amitriptyline.
Granulocytopenia, jaundice and hepatic dysfunction have been observed rarely.
This list includes adverse reactions not reported with Limbitrol but requiring consideration because they have been reported with one or both components or closely related drugs.
Cardiovascular: Hypotension, hypertension, tachycardia, palpitations, myocardial infarction, arrhythmias, heart block, stroke.
Psychiatric: Euphoria, apprehension, poor concentration, delusions, hallucinations, hypomania and increased or decreased libido.
Neurologic: Incoordination, ataxia, numbness, tingling and paresthesias of the extremities, extrapyramidal symptoms, syncope, changes in EEG patterns.
Anticholinergics: Disturbance of accommodation, paralytic ileus, urinary retention, dilatation of urin tract.
Allergic: Skin rash, urticaria, photosensitization, edema of face and tongue, pruritus.
Hematologic: Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia.
Gastrointestinal: Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, black tongue.
Endocrine: Testicular swelling and gynecomasti the male, breast enlargement, galactorrhea and minor menstrual irregularities in the female and elevation and lowering of blood sugar levels.
Other: Headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, jaund alopecia, parotid swelling.
Overdosage: Immediately hospitalize patient suspected of having taken an overdose. Treatme symptomatic and supportive. I.V. administration to 3 mg physostigmine salicylate has been repo to reverse the symptoms of amitriptyline poisonir See complete product information for manifestat and treatment.
Dosage: Individualize according to symptom se and patient response. Reduce to smallest effecti dosage when satisfactory response is obtained. Larger portion of daily dose may be taken at bed Single h.s. dose may suffice for some patients. L dosages are recommended for the elderly.
Limbitrol 10-25, initial dosage of three to four tab daily in divided doses, increased up to six tablet decreased to two tablets daily as required. Limb 5-12.5, initial dosage of three to four tablets daily divided doses, for patients who do not tolerate h doses.
Supplied: Limbitrol 10-25 tablets each containin mg chlordiazepoxide and 25 mg amitriptyline (a hydrochloride salt) and Limbitrol 5-12.5 tablets e containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)—bottles 100 and 500, Tel-E-Dose® packages of 100; Prescription Paks of 50.

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Children can be cruel. Their teasing about taking noontime medication can cause serious problems for the child with MBD.

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Cylert should not be used for (and will not be effective in) simple cases of overactivity in school-age children.

Neither should it be used in the child who exhibits symptoms secondary to environmental factors and/or primary psychiatric disorders, including psychosis.

The physician should rely on a complete history of the child and a thorough description of symptoms from both parents and teacher before postulating a diagnosis of MBD.

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A5

Cylert and Cylert Chewable Tablets

(pemoline)

Prescribing Information

Indications: *MINIMAL BRAIN DYSFUNCTION IN CHILDREN*—as adjunctive therapy to other remedial measures (psychological, educational, social).

Special Diagnostic Considerations: The cause of minimal brain dysfunction (MBD) is unknown. Diagnosis of MBD involves the use of medical, psychological, educational, and social tools, since no single diagnostic test is adequate.

MBD is characterized by chronic moderate to severe hyperactivity, short attention span, distractibility, emotional lability, and impulsivity. Nonlocalizing (soft) neurological signs, learning disability, and abnormal EEG may or may not be present. The diagnosis of MBD must be based upon a complete history and evaluation of the child and not solely on the presence of one or more of these characteristics.

Drug treatment is not indicated for all children with MBD. In the primary therapy of MBD, appropriate educational placement is essential and psychosocial intervention is generally necessary. When these measures alone are insufficient, the decision to prescribe stimulant medication will depend upon the physician's assessment of the chronicity and severity of the child's symptoms. Stimulants are not intended for use in the child who exhibits symptoms secondary to environmental factors and/or primary psychiatric disorders, including psychosis.

Contraindications: Cylert (pemoline) is contraindicated in patients with known hypersensitivity or idiosyncrasy to the drug. (See ADVERSE REACTIONS.)

Warnings: Cylert is not recommended for children less than 6 years of age since its safety and efficacy in this age group have not been established.

Sufficient data on the safety and the efficacy of the long-term use of Cylert in children with minimal brain dysfunction are not yet available.

A temporary suppression of the predicted growth rate (weight and/or height gain) has been reported for children receiving long-term stimulant therapy. A definite causal relationship between stimulant drugs and this finding has not been established.

Precautions: Liver function tests should be performed periodically during therapy with Cylert. The drug should be discontinued if abnormalities are revealed and confirmed by follow-up tests. (See ADVERSE REACTIONS regarding reports of abnormal liver function tests and jaundice.)

Cylert should be administered with caution to patients with significantly impaired hepatic or renal function.

The interaction of Cylert with other drugs has not been studied in humans. Patients who are receiving Cylert concurrently with other drugs, especially drugs with CNS activity, should be monitored carefully.

Cylert failed to demonstrate a potential for self-administration in primates. However, the pharmacologic similarity of pemoline to other psychostimulants with known dependence liability suggests that psychological and/or physical dependence might also occur with Cylert. There have been isolated reports of transient psychotic symptoms occurring in adults following the long-term misuse of excessive oral doses of pemoline. Cylert should be given with caution to emotionally unstable patients who may increase the dosage on their own initiative.

Usage during Pregnancy and Lactation: The safety of Cylert (pemoline) for use during pregnancy and lactation has not been established.

Fertility, reproduction, and teratology studies were conducted in laboratory animals. Pemoline, in doses of 18.75 or 37.5 mg./kg./day, had no effect on the fertility of male or female rats. The drug, when given to pregnant rats (from gestation day 15 through weaning) and to rabbits (from gestation days 6-18) at these same dosage levels, produced no teratogenic or embryotoxic effects, and had no effect on the viability of the young at birth. However, increased incidences of stillbirths and cannibalization were observed when pemoline was given to rats at these dosage levels, beginning 14 days prior to conception.

Adverse Reactions: Insomnia is the most frequently reported side effect of Cylert; it usually occurs early in therapy, prior to an optimum therapeutic response. In the majority of cases it is transient in nature or responds to a reduction in dosage.

Anorexia with weight loss may occur during the first weeks of therapy. In the majority of cases it is transient in nature; weight gain usually resumes within three to six months.

Stomach ache, skin rashes, increased irritability, mild depression, nausea, dizziness, headache, drowsiness, and hallucinations have been reported.

Elevations of SGOT, SGPT, and serum LDH have occurred in patients taking Cylert, usually after several months of therapy. These effects appear to be reversible upon withdrawal of the drug, and are thought to be manifestations of a delayed hypersensitivity reaction. There have also been a few reports of jaundice occurring in patients taking Cylert; a causal relationship between the drug and this clinical finding has not been established.

There have been reports of dyskinetic movements of the lips, face, and extremities occurring with the use of Cylert. Convulsive seizures have also been reported. A definite causal relationship between Cylert and these reactions has not been established.

Mild adverse reactions appearing early during the course of treatment with Cylert often remit with continuing therapy. If adverse reactions are of a significant or protracted nature, dosage should be reduced or the drug discontinued.

How Supplied: Cylert (pemoline) is supplied as monogrammed, grooved tablets in three dosage strengths:

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37.5 mg. tablets (orange-colored) in bottles of 100 (**NDC** 0074-6057-13)
75 mg. tablets (tan-colored) in bottles of 100 (**NDC** 0074-6073-13)

Cylert Chewable is supplied as monogrammed, grooved tablets in one dosage strength:

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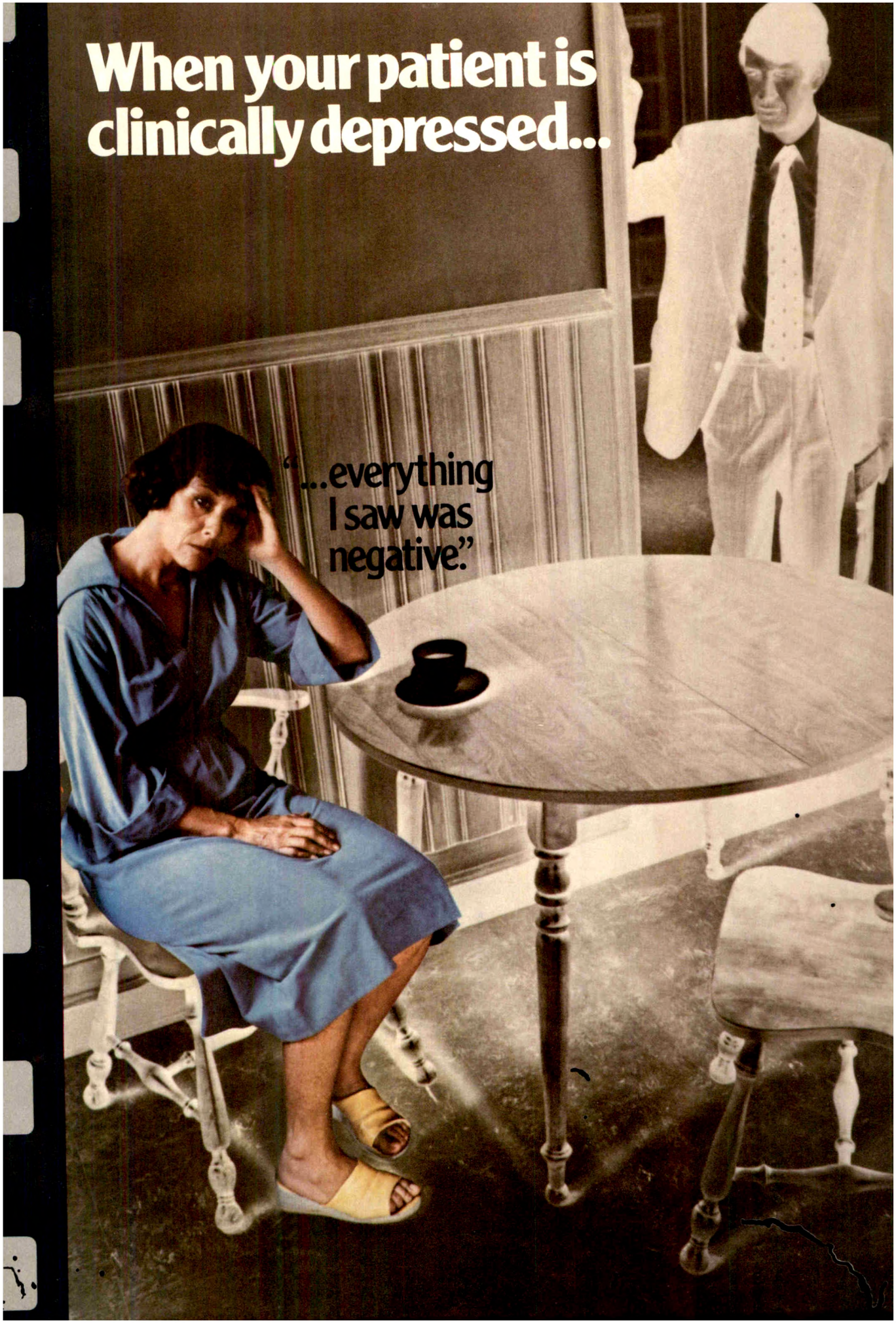
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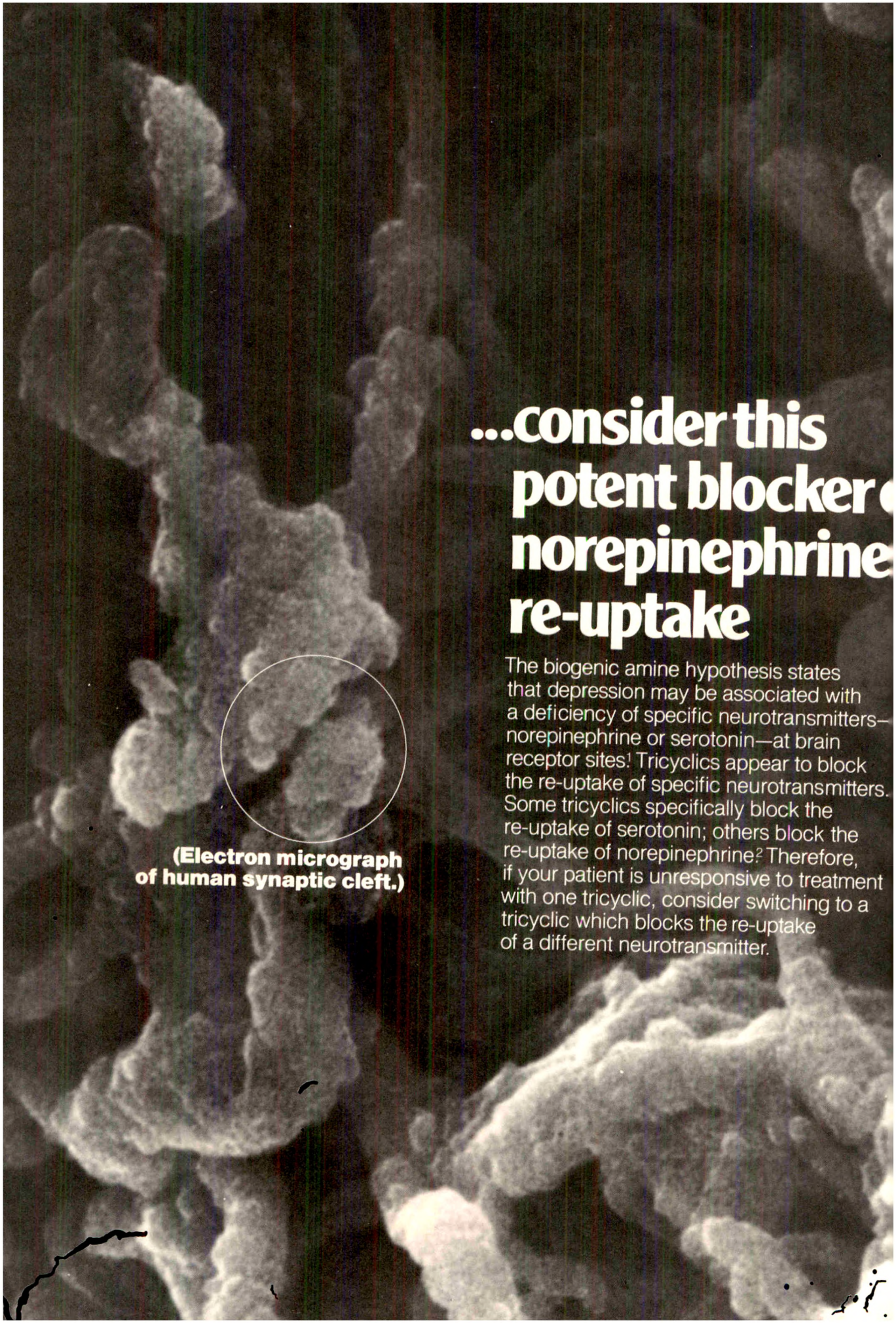
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When your patient is clinically depressed...
"...everything I saw was negative."



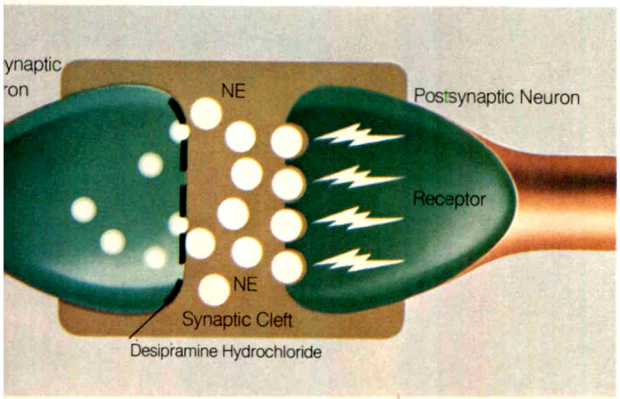
...consider this potent blocker of norepinephrine re-uptake

The biogenic amine hypothesis states that depression may be associated with a deficiency of specific neurotransmitters–norepinephrine or serotonin—at brain receptor sites.[1] Tricyclics appear to block the re-uptake of specific neurotransmitters. Some tricyclics specifically block the re-uptake of serotonin; others block the re-uptake of norepinephrine.[2] Therefore, if your patient is unresponsive to treatment with one tricyclic, consider switching to a tricyclic which blocks the re-uptake of a different neurotransmitter.

(Electron micrograph of human synaptic cleft.)

Norpramin®
lesipramine
ydrochloride
ablets NF)
,50,75,100,150 mg.

tent blocker
norepinephrine re-uptake
cording to current theory[1,3] some
yclic antidepressants may fail in certain
es of depression because they are not
iciently potent blockers of norepi-
hrine re-uptake. Laboratory studies
e shown that desipramine is the most
ent inhibitor of norepinephrine
uptake.[4] Norpramin may work in some
ents when other tricyclic antidepres-
ts do not.



onsider it now,
onsider it next.

Less anticholinergic activity
(See Warnings section.)
Studies in animals[5] and in normal human subjects[6,7] have shown that desipramine has significantly less anticholinergic activity than amitriptyline or doxepin. The single bond side chain is associated with lower anticholinergic activity than these other leading antidepressants. This may mean:

- less dry mouth
- less blurred vision
- less urinary retention

Helps patients remain calm but active
Norpramin does not usually inhibit normal activity although patients should be cautioned as to driving or operating machinery if drowsiness occurs.

In addition…

Begins to improve sleep patterns within one week in some patients[8]

- Norpramin helps relieve the sleep disturbances that often accompany depression.
- As the depression is relieved, the difficulty in falling asleep, restlessness, and early morning awakening diminish.

References:
1. Maas, J.W.: Biogenic amines and depression. Biochemical and pharmacological separation of two types of depression. Arch. Gen. Psychiat. 32:1357-1361, 1975. **2.** Mendels, J., Stern, S., and Frazer, A.: Dis. Nerv. Syst. 37:3, 1976. **3.** Davis, J.M.: Central biogenic amines and theories of depression and mania. In, Phenomenology and Treatment of Depression, W.E. Fann, I. Karacan, A.D. Pokorny, and R.L. Williams, Ed., New York, Spectrum Publications, Inc., 1977, pp. 17-32. **4.** Ross, S.B. and Renyi, A.L.: Tricyclic antidepressant agents. 1. Comparison of the inhibition of the uptake of ^{3}H-noradrenaline and ^{14}C-5-hydroxytryptamine in slices and crude synaptosome preparations of the midbrain-hypothalamus region of the rat brain. Acta Pharmacol. (Kobenhavn) 36:382-394, 1975. **5.** Snyder, S.H. and Yamamura, H.I.: Antidepressants and the muscarinic acetylcholine receptor. Arch. Gen. Psychiat. 34:236-239, 1977. **6.** Blackwell, B., Stefopoulos, A., Enders, P., Kuzma, R., and Adolphe, A.: The anticholinergic activity of two tricyclic antidepressants. Amer. J. Psychiat., 135:722-724, June 1978. **7.** Peterson, G.R., Hostetler, R.M., Blackwell, B., Kuzma, R., and Adolphe, A.B.: Effects of the tricyclic antidepressants desipramine and doxepin on anticholinergic and CNS activity in non-depressed volunteers. Neuroscience Abstr. 3:447, 1977. **8.** Zung, W.W.K.: The pharmacology of disordered sleep. A laboratory approach. J.A.M.A. 211:1532-1534, 1970.

(For prescribing information see next page.)

Merrell

Norpramin®
(desipramine hydrochloride tablets NF)

AVAILABLE ONLY ON PRESCRIPTION

Brief Summary

MECHANISM OF ACTION: Available evidence suggests that many depressions have a biochemical basis in the form of a relative deficiency of neurotransmitters such as norepinephrine and serotonin. Norepinephrine deficiency may be associated with relatively low urinary 3-methoxy-4-hydroxyphenyl glycol (MHPG) levels, while serotonin deficiencies may be associated with low spinal fluid levels of 5-hydroxyindolacetic acid.

While the precise mechanism of action of the tricyclic antidepressants is unknown, a leading theory suggests that they restore normal levels of neurotransmitters by blocking the re-uptake of these substances from the synapse in the central nervous system.

Evidence indicates that the secondary amine tricyclic antidepressants, including Norpramin, may have greater activity in blocking the re-uptake of norepinephrine. Tertiary amine tricyclic antidepressants, such as amitriptyline, may have greater effect on serotonin re-uptake.

Norpramin (desipramine hydrochloride) is not a monoamine oxidase (MAO) inhibitor and does not act primarily as a central nervous system stimulant. It has been found in some studies to have a more rapid onset of action than imipramine. Earliest therapeutic effects may occasionally be seen in 2 to 5 days, but full treatment benefit usually requires 2 to 3 weeks to obtain.

INDICATIONS: Norpramin (desipramine hydrochloride) is indicated for relief of symptoms in various depressive syndromes, especially endogenous depression.

CONTRAINDICATIONS: Desipramine hydrochloride should not be given in conjunction with, or within 2 weeks of, treatment with an MAO inhibitor drug; hyperpyretic crises, severe convulsions, and death have occurred in patients taking MAO inhibitors and tricyclic antidepressants. When Norpramin (desipramine hydrochloride) is substituted for an MAO inhibitor, at least 2 weeks should elapse between treatments. Norpramin should then be started cautiously and should be increased gradually.

The drug is contraindicated in the acute recovery period following myocardial infarction. It should not be used in those who have shown prior hypersensitivity to the drug. Cross sensitivity between this and other dibenzazepines is a possibility.

WARNINGS: 1. Extreme caution should be used when this drug is given in the following situations: a. In patients with cardiovascular disease, because of the possibility of conduction defects, arrhythmias, tachycardias, strokes, and acute myocardial infarction. b. In patients with a history of urinary retention or glaucoma, because of the anticholinergic properties of the drug. c. In patients with thyroid disease or those taking thyroid medication, because of the possibility of cardiovascular toxicity, including arrhythmias. d. In patients with a history of seizure disorder, because this drug has been shown to lower the seizure threshold. 2. This drug is capable of blocking the antihypertensive effect of guanethidine and similarly acting compounds. 3. USE IN PREGNANCY: Safe use of desipramine hydrochloride during pregnancy and lactation has not been established; therefore, if it is to be given to pregnant patients, nursing mothers, or women of childbearing potential, the possible benefits must be weighed against the possible hazards to mother and child. Animal reproductive studies have been inconclusive. 4. USE IN CHILDREN: Norpramin (desipramine hydrochloride) is not recommended for use in children since safety and effectiveness in the pediatric age group have not been established. 5. The patient should be cautioned that this drug may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery. 6. In patients who may use alcohol excessively, it should be borne in mind that the potentiation may increase the danger inherent in any suicide attempt or overdosage.

PRECAUTIONS: 1. It is important that this drug be dispensed in the least possible quantities to depressed outpatients, since suicide has been accomplished with this class of drug. Ordinary prudence requires that children not have access to this drug or to potent drugs of any kind; if possible this drug should be dispensed in containers with child-resistant safety closures. Storage of this drug in the home must be supervised responsibly. 2. If serious adverse effects occur, dosage should be reduced or treatment should be altered. 3. Norpramin (desipramine hydrochloride) therapy in patients with manic-depressive illness may induce a hypomanic state after the depressive phase terminates. 4. The drug may cause exacerbation of psychosis in schizophrenic patients. 5. Close supervision and careful adjustment of dosage are required when this drug is given concomitantly with anticholinergic or sympathomimetic drugs. 6. Patients should be warned that while taking this drug their response to alcoholic beverages may be exaggerated. 7. Clinical experience in the concurrent administration of ECT and antidepressant drugs is limited. Thus, if such treatment is essential, the possibility of increased risk relative to benefits should be considered. 8. The sedative effects of Norpramin and benzodiazepines (*e.g.*, chlordiazepoxide or diazepam) are additive. Both the sedative and anticholinergic effects of the major tranquilizers are additive to those of Norpramin. 9. This drug should be discontinued as soon as possible prior to elective surgery because of the possible cardiovascular effects. Hypertensive episodes have been observed during surgery in patients taking desipramine hydrochloride. 10. Both elevation and lowering of blood sugar levels have been reported. 11. Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during therapy; the drug should be discontinued if there is evidence of pathologic neutrophil depression.

ADVERSE REACTIONS: Note: Included in the following listing are a few adverse reactions that have not been reported with this specific drug. However, the pharmacologic similarities among the tricyclic antidepressant drugs require that each of the reactions be considered when Norpramin (desipramine hydrochloride) is given.

Cardiovascular: hypotension, hypertension, tachycardia, palpitation, arrhythmias, heart block, myocardial infarction, stroke.

Psychiatric: confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia and nightmares; hypomania; exacerbation of psychosis.

Neurologic: numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures; alteration in EEG patterns; tinnitus.

Anticholinergic: dry mouth, and rarely associated sublingual adenitis; blurred vision, disturbance of accommodation, mydriasis, increased intraocular pressure; constipation, paralytic ileus; urinary retention, delayed micturition, dilatation of urinary tract.

Allergic: skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight), edema (of face and tongue or general), drug fever, cross sensitivity with other tricyclic drugs.

Hematologic: bone marrow depressions including agranulocytosis, eosinophilia, purpura, thrombocytopenia.

Gastrointestinal: anorexia, nausea and vomiting, epigastric distress, peculiar taste, abdominal cramps, diarrhea, stomatitis, black tongue.

Endocrine: gynecomastia in the male, breast enla ment and galactorrhea in the female; increase decreased libido, impotence, testicular swelling; el tion or depression of blood sugar levels.

Other: jaundice (simulating obstructive), altered function; weight gain or loss; perspiration, flus urinary frequency, nocturia; parotid swelling; dro ness, dizziness, weakness and fatigue, headache; pecia.

Withdrawal Symptoms: Though not indicative of ad tion, abrupt cessation of treatment after prolo therapy may produce nausea, headache, and mal

DOSAGE AND ADMINISTRATION: Not recommended fo in children. Lower dosages are recommended for el patients and adolescents. Lower dosages are recommended for outpatients compared to hospita patients, who are closely supervised. Dosage shou initiated at a low level and increased accordin clinical response and any evidence of intolera Following remission, maintenance medication ma required for a period of time and should be at the lo dose that will maintain remission.

Usual Adult Dose: The usual adult dose is 100 to 20 per day. In more severely ill patients, dosage ma further increased gradually to 300 mg./day if neces Dosages above 300 mg./day are not recommende

Dosage should be initiated at a lower level increased according to tolerance and clinical respe

Treatment of patients requiring as much as 30 should generally be initiated in hospitals, where re visits by the physician, skilled nursing care, and quent electrocardiograms (ECG's) are available.

The best available evidence of impending to from very high doses of Norpramin is prolongation QRS or QT intervals on the ECG. Prolongation of th interval is also significant, but less closely corre with plasma levels. Clinical symptoms of intolera especially drowsiness, dizziness, and postural hyp sion, should also alert the physician to the nee reduction in dosage. Plasma desipramine measure would constitute the optimal guide to dosage mo ing.

Initial therapy may be administered in divided d or a single daily dose.

Maintenance therapy may be given on a once- schedule for patient convenience and compliance.

Adolescent and Geriatric Dose: The usual adolescen geriatric dose is 25 to 100 mg. daily.

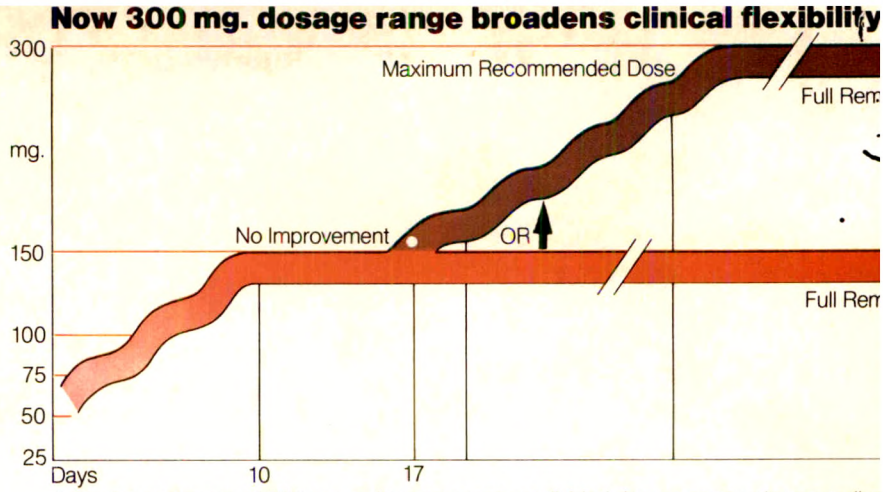
Dosage should be initiated at a lower level increased according to tolerance and clinical resp to a usual maximum of 100 mg. daily. In more severe patients, dosage may be further increased to 150 day. Doses above 150 mg./day are not recommend these age groups.

Initial therapy may be administered in divided d or a single daily dose.

Maintenance therapy may be given on a once- schedule for patient convenience and compliance.

OVERDOSAGE: See prescribing information for a di sion of symptoms and treatment of overdose.

Product Information as of April, 1978



A maximum dosage of 300 mg. a day, as a single or divided dose, may now be prescribe Norpramin. Since inadequate dosage is thought to be one of the major causes of treatme failure, the new therapeutic range should prove clinically helpful. Plasma desipramine measurement would constitute the optimal guide to dosage monitoring.

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The American Psychiatric Association announces publication of two volumes reporting the Conference on Education of Psychiatrists

held at Lake of the Ozarks, Missouri, June 9-15, 1975. Organized by the A.P.A. in cooperation with the American Academy of Child Psychiatry, the American Association of Chairmen of Departments of Psychiatry, and the American Association of Directors of Psychiatric Residency Training.

1. PSYCHIATRIC EDUCATION: PROLOGUE TO THE 1980's, Library Bound, 544 pages, $15.00.

This is a substantive, highly readable, report of the Conference, written by Anne H. Rosenfeld with an Editorial Board comprising: Ewald W. Busse, M.D., Chairperson of the Conference, Walter E. Barton, M.D., Bernard Holland, M.D., Donna Norris, M.D., Melvin Sabshin, M.D., Jeanne Spurlock, M.D., and Robert L. Robinson, M.A. (Staff Consultant).

The volume contains ten chapters covering the topical structure of the Conference: Goals and Values; Competence, Evaluation, and Quality Control; The Resident; Faculty; Educational Methods: Means in the Service of Ends; The Program Environment; Research; The Economics of Residency Training; Psychiatry in Its Social Context; and Retrospect and Prospect. It also contains nine appendices describing the organization of the Conference and its participants, together with selected model programs and related informational materials of value.

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Papers commenting on the reports of the commissions, published here for the first time, were presented at the Conference by Melvin Sabshin, M.D., Eugene B. Brody, M.D., Jeanne Spurlock, M.D., Jack R. Ewalt, M.D., Robert J. Stoller, M.D., James R. Comer, M.D., and Bernard Holland, M.D.

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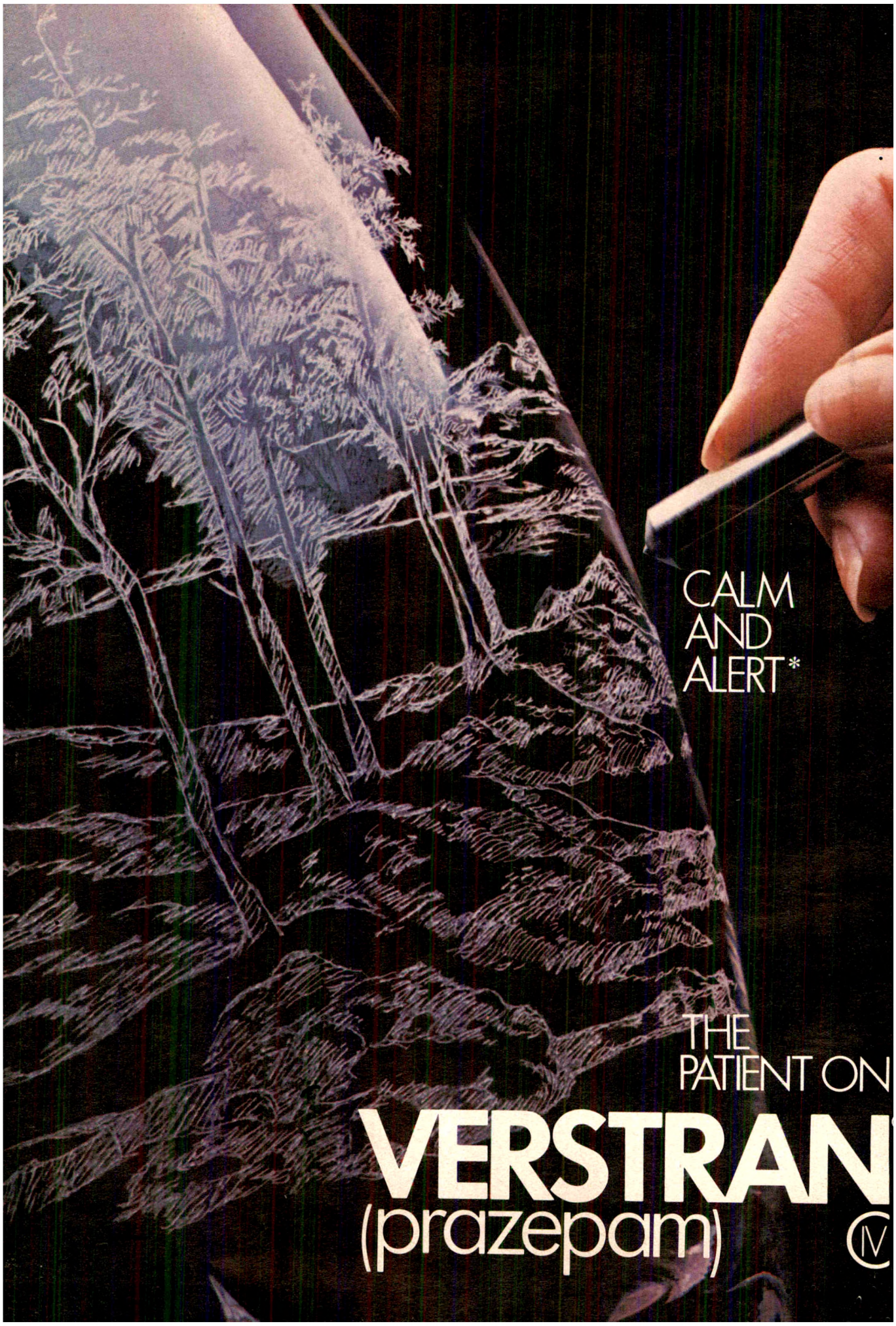
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See package insert.

At bedtime.

Before prescribing, see complete product information on the following page.

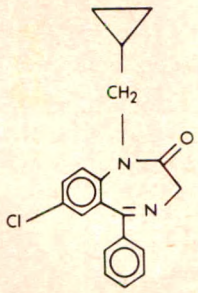
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Description: Verstran (prazepam), a benzodiazepine derivative, is identified chemically as 7-chloro-1-(cyclopropylmethyl)-1,3-dihydro-5-phenyl-2H-1,4-benzodiazepin-2-one. The molecular weight is 324.8 and the structural formula is as follows:



Clinical Pharmacology: Studies in normal subjects have shown that Verstran (prazepam) has depressant effects on the central nervous system. Oral administration of single doses as high as 60 mg and divided doses up to 100 mg t.i.d. (300 mg total daily dose) were without toxic effects.

Single oral doses of Verstran in normal subjects produced peak blood levels at 6 hours postadministration, with significant amounts still present after 48 hours. Verstran was slowly absorbed over a prolonged period, rather constant blood levels were maintained, and excretion was prolonged. The mean half-life of prazepam measured in subjects given 10 mg t.i.d. for one week was 63 (±15 s.d.) hours before and 70 (±10 s.d.) hours after multiple dosing — a nonsignificant difference. Human metabolism studies showed that prior to elimination from the body, prazepam is metabolized in large part to 3-hydroxyprazepam and oxazepam.

Indications: Verstran (prazepam) is indicated for the symptomatic relief of anxiety associated with anxiety neurosis, in other psychoneuroses in which anxiety symptoms are prominent features, and as an adjunct in disease states in which anxiety is manifested.

The effectiveness of Verstran (prazepam) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should reassess periodically the usefulness of the drug for the individual patient.

Contraindications: Verstran (prazepam) is contraindicated in patients with a known sensitivity to the drug and in those with acute narrow angle glaucoma.

Warnings: Verstran (prazepam) is not recommended in psychotic states and in those psychiatric disorders in which anxiety is not a prominent feature.

Patients taking Verstran should be cautioned against engaging in hazardous occupations requiring mental alertness, such as operating dangerous machinery, including motor vehicles.

Because Verstran has a central nervous system depressant effect, patients should be advised against the simultaneous use of other CNS-depressant drugs, including phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants. The effects of alcohol may also be increased.

Physical and Psychological Dependence: Withdrawal symptoms similar in character to those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepine drugs. These symptoms include convulsions, tremor, abdominal and muscle cramps, vomiting and sweating. Addiction-prone individuals, such as drug addicts and alcoholics, should be under careful surveillance when receiving benzodiazepines because of the predisposition of such patients to habituation and dependence.

Precautions:

Use in Pregnancy and Lactation:

An increased risk of congenital malformations associated with the use of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) during the first trimester of pregnancy has been suggested in several studies. Verstran (prazepam) a benzodiazepine derivative, has not been studied adequately to determine whether it, too, may be associated with an increased risk of fetal abnormality. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physician about the desirability of discontinuing the drug. In view of their molecular size, Verstran and its metabolites are probably excreted in human milk. Therefore, this drug should not be given to nursing mothers.

In those patients in whom a degree of depression accompanies the anxiety, suicidal tendencies may be present, and protective measures may be required. The least amount of drug that is feasible should be available to the patient at any one time.

Patients taking Verstran for prolonged periods should have blood counts and liver function tests periodically. The usual precautions in treating patients with impaired renal or hepatic function should also be observed. Hepatomegaly and cholestasis were observed in chronic toxicity studies in rats and dogs.

In elderly or debilitated patients, the initial dose should be small, and increments should be made gradually, in accordance with the response of the patient, to preclude ataxia or excessive sedation.

Pediatric Use: Safety and effectiveness in patients below the age of 18 have not been established.

Adverse Reactions: The side effects most frequently reported during double-blind placebo-controlled trials employing a typical 30 mg divided total daily dose and the percent incidence in the Verstran (prazepam) group were: fatigue (11.6%), dizziness (8.7%), weakness (7.7%), drowsiness (6.8%), lightheadedness (6.8%), and ataxia (5.0%). Less frequently reported were: headache, confusion, tremor, vivid dreams, slurred speech, palpitation, stimulation, dry mouth, diaphoresis, and various gastrointestinal complaints. Other side effects included: pruritus, transient skin rashes, swelling of feet, joint pains, various genitourinary complaints, blurred vision, and syncope. Single nightly dose, controlled trials of variable dosages showed a dose-related incidence of these same side effects. Transient and reversible aberrations of liver function tests have been reported, as have slight decreases in blood pressure and increases in body weight.

These findings are characteristic of benzodiazepine drugs.

Overdosage: As in the management of overdosage with any drug, it should be borne in mind that multiple agents may have been taken.

If vomiting has not occurred spontaneously, it should be induced. Immediate gastric lavage is also recommended. General supportive care, including frequent monitoring of the vital signs and close observation of the patient, is indicated. Hypotension though unlikely, may be controlled with Levophed® (levarterenol bitartrate) or Aramine® (metaraminol bitartrate). Caffeine and Sodium Benzoate Injection USP, may be used to counteract central nervous system depressant effects.

Dosage and Administration: Verstran (prazepam) is administered orally in divided doses. The usual daily dose is 30 mg. The dose should be adjusted gradually within the range of 20 to 60 mg daily in accordance with the response of the patient. In elderly or debilitated patients it is advisable to initiate treatment at a divided daily dose of 10 mg to 15 mg. (See Precautions.)

Prazepam may also be administered as a single dose daily at bedtime. The recommended starting nightly dose is 20 mg. The response of the patient to several days' treatment will permit the physician to adjust the dose upward or, occasionally, downward to maximize antianxiety effect with a minimum of daytime drowsiness. The optimum dosage will usually range from 20 to 40 mg.

Drug Interactions: If Verstran (prazepam) is to be combined with other drugs acting on the central nervous system, careful consideration should be given to the pharmacology of the agents to be employed. The actions of the benzodiazepines may be potentiated by barbiturates, narcotics, phenothiazines, monoamine oxidase inhibitors or other antidepressants.

If Verstran is used to treat anxiety associated with somatic disease states, careful attention must be paid to possible drug interaction with concomitant medication.

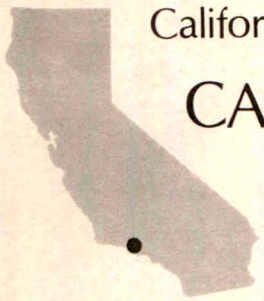
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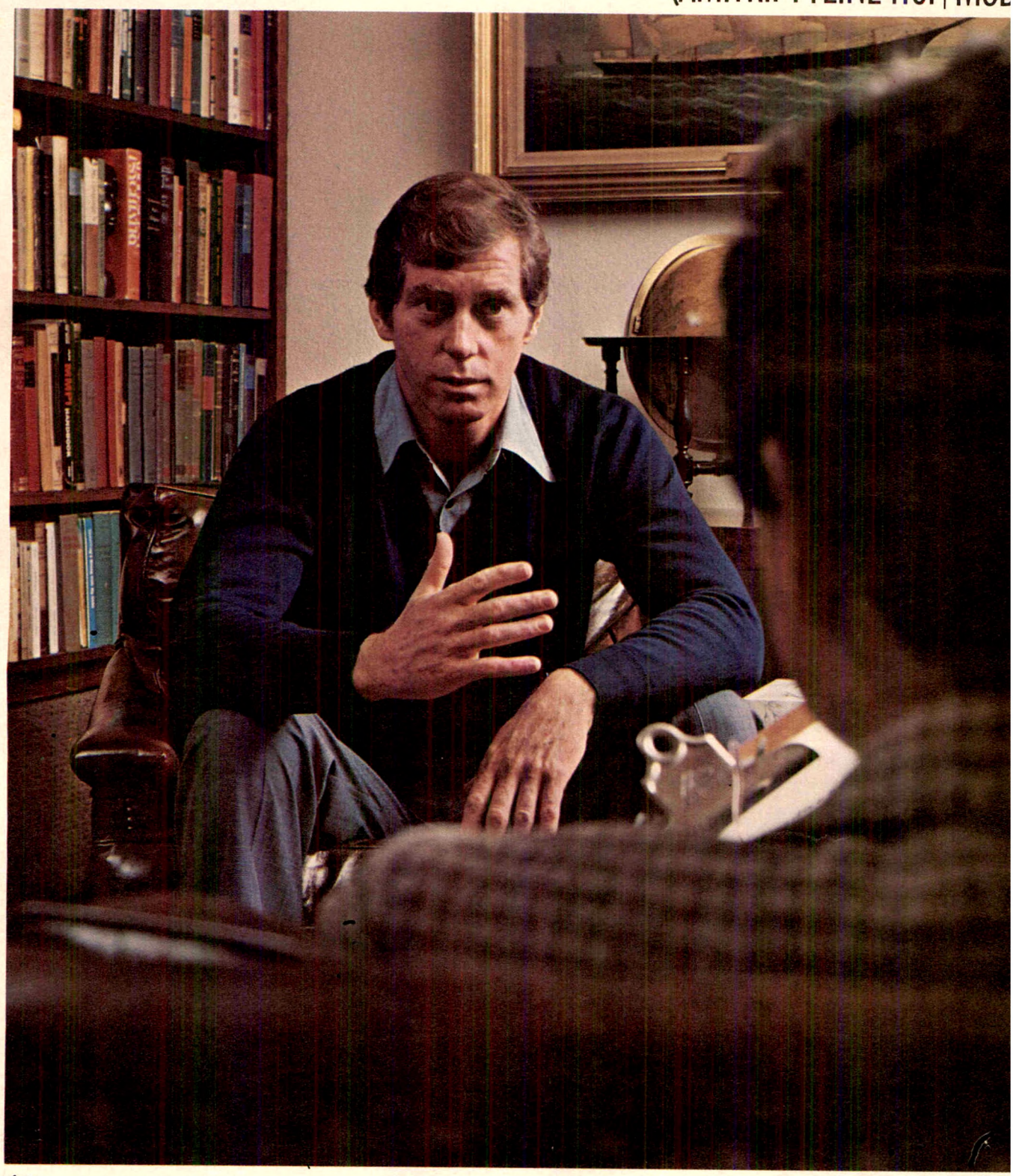
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MSD
MERCK SHARP & DOHME

Contraindications: Known hypersensitivity. Should not be given concomitantly with a monoamine oxidase inhibitor since hyperpyretic crises, severe convulsions, and deaths have occurred. When used to replace a monoamine oxidase inhibitor, allow a minimum of 14 days to elapse before initiating therapy with amitriptyline HCl. Initiate dosage of amitriptyline HCl cautiously with gradual increase in dosage until optimum response is achieved. Not recommended during the acute recovery phase following myocardial infarction.

Warnings: May block the antihypertensive action of guanethidine or similarly acting compounds. Should be used with caution in patients with a history of seizures or a history of urinary retention, or with angle-closure glaucoma or increased intraocular pressure; in patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely; arrhythmias, sinus tachycardia and prolongation of the conduction time have been reported, particularly with high doses; myocardial infarction and stroke have been reported with drugs of this class. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. May impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle. In patients who use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage. Safe use during pregnancy and lactation has not been established; in pregnant patients, nursing mothers, or women who may become pregnant, weigh possible benefits against possible hazards to mother and child. Not recommended for patients under 12 years of age.

Precautions: Schizophrenic patients may develop increased symptoms of psychosis; patients with paranoid symptomatology may have an exaggeration of such symptoms; manic depressive patients may experience a shift to the manic phase. In these circumstances, the dose of amitriptyline HCl may be reduced or a major tranquilizer, such as perphenazine, may be administered concurrently.

When given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required; paralytic ileus may occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs. Use cautiously in patients receiving large doses of ethchlorvynol, since transient delirium has been reported on concurrent administration. May enhance the response to alcohol and the effects of barbiturates and other CNS depressants. The possibility of suicide in depressed patients remains until significant remission occurs. Potentially suicidal patients should not have access to large quantities of this drug. Prescriptions should be written for the smallest amount feasible. Concurrent electroshock therapy may increase the hazards associated with such therapy; such treatment should be limited to patients for whom it is essential. When possible, discontinue the drug several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported. Use with caution in patients with impaired liver function.

Adverse Reactions: *Note:* Included in this listing are a few adverse reactions not reported with this specific drug. However, pharmacological similarities among the tricyclic antidepressant drugs require that each reaction be considered when amitriptyline is administered. *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, increased intraocular pressure, constipation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, leukopenia, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, black tongue, rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache, and malaise; these are not indicative of addiction.

Overdosage: Hospitalize as soon as possible all patients suspected of having taken an overdose. Treatment is symptomatic and supportive. In addition, the intravenous administration of 1 to 3 mg physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage should be repeated as required, particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine.

How Supplied: Tablets containing 10 mg and 25 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100, 1000, and 5000; tablets containing 50 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100 and 1000; tablets containing 75 mg and 100 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100; tablets containing 150 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 30 and 100; for intramuscular use, in 10-ml vials containing per ml: 10 mg amitriptyline HCl, 44 mg dextrose, 1.5 mg methylparaben and 0.2 mg propylparaben as preservatives, and water for injection q.s. 1 ml.

For more detailed information, consult your MSD representative-or see full prescribing information. Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa. 19486

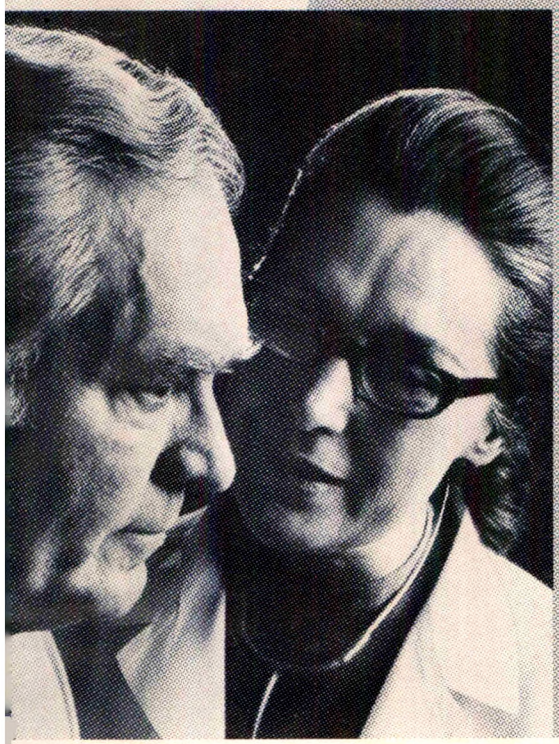
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Please see next page for summary of product information.

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Before prescribing, please consult complete product information, a summary of which follows:

Indications: Relief of anxiety and tension occurring alone or accompanying various disease states. Efficacy beyond four months not established by systematic clinical studies. Periodic reassessment of therapy recommended.

Contraindications: Patients with known hypersensitivity to the drug.

Warnings: Warn patients that mental and/or physical abilities required for tasks such as driving or operating machinery may be impaired, as may be mental alertness in children, and that concomitant use with alcohol or CNS depressants may have an additive effect. Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported.

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (*e.g.*, excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.

Usual Daily Dosage: Individualize for maximum beneficial effects. *Oral–Adults:* Mild and moderate anxiety and tension, 5 or 10 mg *t.i.d.* or *q.i.d.*; severe states, 20 or 25 mg *t.i.d.* or *q.i.d.* *Geriatric patients:* 5 mg *b.i.d.* to *q.i.d.* (See Precautions.)

Supplied: Librium® (chlordiazepoxide HCl) *Capsules*, 5 mg, 10 mg and 25 mg—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50, available singly and in trays of 10. Libritabs® (chlordiazepoxide) *Tablets*, 5 mg, 10 mg and 25 mg—bottles of 100 and 500. With respect to clinical activity, capsules and tablets are indistinguishable.

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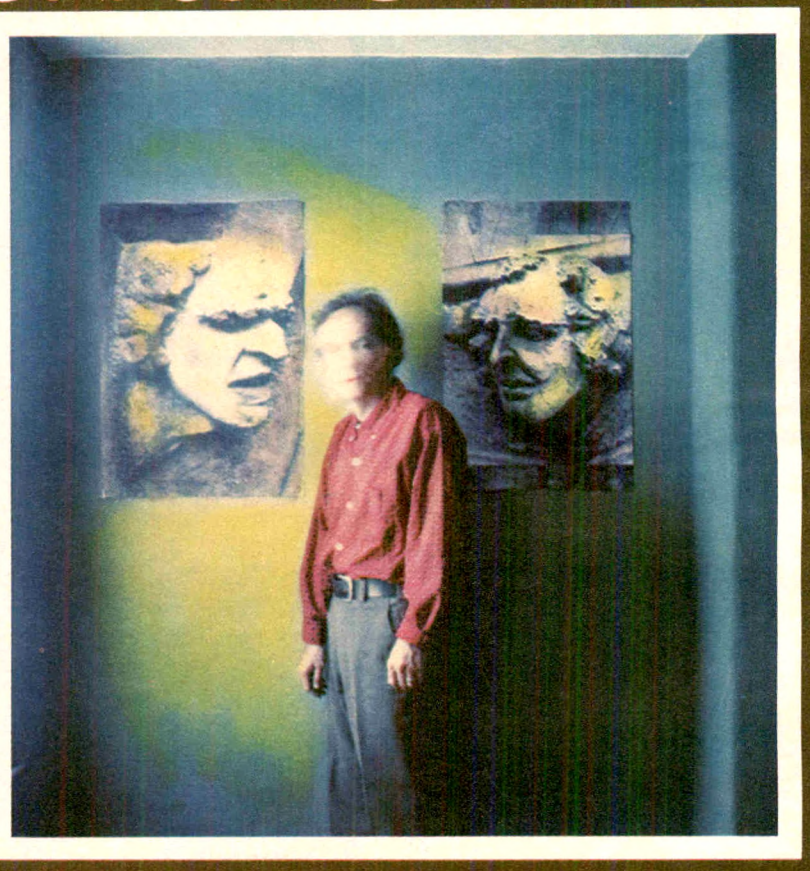
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Before prescribing, see complete prescribing information in SK&F literature or PDR. The following is a brief summary.

Indications

Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

Effective: For the management of the manifestations of psychotic disorders.

Possibly effective: To control excessive anxiety, tension and agitation as seen in neuroses or associated with somatic conditions.

'Stelazine' has not been shown effective in the management of behavioral complications in patients with mental retardation.

Final classification of the less-than-effective indications requires further investigation.

Contraindications: Comatose or greatly depressed states due to C.N.S. depressants; blood dyscrasias; bone marrow depression; liver damage.

Warnings: Generally avoid using in patients hypersensitive (e.g., blood dyscrasias, jaundice) to any phenothiazine. Caution patients about activities requiring alertness (e.g., operating vehicles or machinery), especially during the first few days' therapy.

Use in pregnancy only when necessary for patient's welfare.

Precautions: Use cautiously in angina. Avoid high doses and parenteral administration when cardiovascular system is impaired. Antiemetic effect may mask signs of toxic drug overdosage or physical disorders. Additive effect is possible with other C.N.S. depressants. Prolonged administration of high doses may result in cumulative effects with severe C.N.S. or vasomotor symptoms. If retinal changes occur, discontinue drug. Agranulocytosis, thrombocytopenia, pancytopenia, anemia, cholestatic jaundice, liver damage have been reported.

Patients on long-term therapy, especially high doses, should be evaluated periodically for possible adjustment or discontinuance of drug therapy.

Adverse Reactions: Drowsiness, dizziness, skin reactions, rash, dry mouth, insomnia, amenorrhea, fatigue, muscular weakness, anorexia, lactation, blurred vision. Neuromuscular (extrapyramidal) reactions: motor restlessness, dystonias, pseudo-parkinsonism, persistent tardive dyskinesia.

Other adverse reactions reported with Stelazine (trifluoperazine HCl, SK&F) or other phenothiazines: Some adverse effects are more frequent or intense in specific disorders (e.g., mitral insufficiency or pheochromocytoma).

Grand mal convulsions; altered cerebrospinal fluid proteins; cerebral edema; prolongation and intensification of the action of C.N.S. depressants, atropine, heat, and organophosphorus insecticides; nasal congestion, headache, nausea, constipation, obstipation, adynamic ileus, inhibition of ejaculation; reactivation of psychotic processes, catatonic-like states; hypotension (sometimes fatal); cardiac arrest; leukopenia, eosinophilia, pancytopenia, agranulocytosis, thrombocytopenic purpura; jaundice, biliary stasis; menstrual irregularities, galactorrhea, gynecomastia, false positive pregnancy tests; photosensitivity, itching, erythema; urticaria, eczema up to exfoliative dermatitis; asthma, laryngeal edema, angioneurotic edema, anaphylactoid reactions; peripheral edema; reversed epinephrine effect; hyperpyrexia; a systemic lupus erythematosus-like syndrome; pigmentary retinopathy; with prolonged administration of substantial doses, skin pigmentation, epithelial keratopathy, and lenticular and corneal deposits. EKG changes have been reported, but relationship to myocardial damage is not confirmed. Discontinue long-term, high-dose therapy gradually. NOTE: Sudden death in patients taking phenothiazines (apparently due to cardiac arrest or asphyxia due to failure of cough reflex) has been reported, but no causal relationship has been established.

Supplied: Tablets, 1 mg., 2 mg., 5 mg. and 10 mg., in bottles of 100; in Single Unit Packages of 100 (intended for institutional use only); Injection, 2 mg./ml.; and Concentrate (intended for institutional use only), 10 mg./ml.

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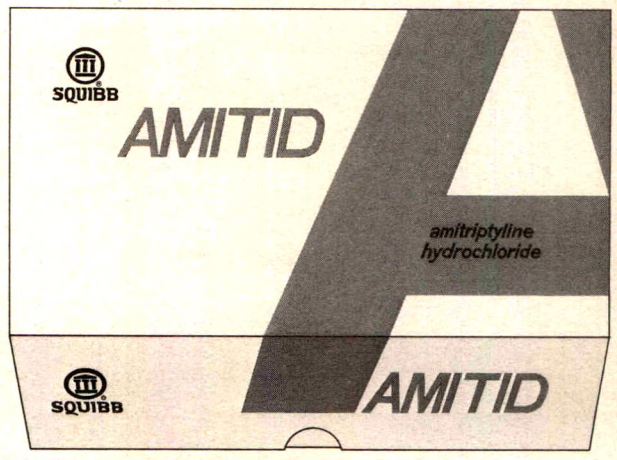
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Please see following page for brief summary.

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DESCRIPTION: Amitriptyline hydrochloride is a dibenzocycloheptadiene derivative available for oral administration as FILMLOK® tablets (FILMLOK is a Squibb trademark for veneer-coated tablets).

INDICATIONS: For relief of symptoms of depression alone or accompanied by anxiety. Endogenous depression is more likely to be alleviated than are other depressive states.

CONTRAINDICATIONS: Contraindicated in patients with a history of hypersensitivity to amitriptyline. Do not administer concomitantly with a monamine oxidase (MAO) inhibitor since hyperpyretic crises, severe convulsions, and deaths occurred when tricyclic antidepressants and MAO inhibitors were administered simultaneously. When an MAO inhibitor is to be replaced by amitriptyline, allow 14 days to elapse after discontinuation of the former and then initiate amitriptyline cautiously and gradually increase dosage until optimum response is achieved. Amitriptyline is not recommended for use during the acute recovery phase following myocardial infarction.

WARNINGS: Amitriptyline may block antihypertensive action of guanethidine or similarly acting drugs. Use with caution in patients with history of seizures and (because of atropine-like action of amitriptyline) in patients with history of narrow-angle glaucoma (even average doses may precipitate an attack), increased intraocular pressure, or urinary retention. Closely watch patients with cardiovascular disorders since, in addition to having caused myocardial infarction and stroke, tricyclic antidepressants (including amitriptyline) particularly with high dosage have been reported to produce arrhythmias, sinus tachycardia, and prolongation of conduction time. Close supervision is required when amitriptyline is given to hyperthyroid patients or those on thyroid medication. Amitriptyline may impair mental and/or physical abilities required for performance of hazardous tasks such as operating machinery or driving a motor vehicle. Bear in mind that in patients who may use alcohol excessively the potentiation may increase the danger inherent in any suicide attempt or overdosage.

Usage in Pregnancy: Safe use during pregnancy and lactation has not been established; therefore, in administering the drug to pregnant patients, nursing mothers, or women who may become pregnant, weigh the possible benefits against the possible hazards to the mother and child. Animal reproduction studies have been inconclusive, and clinical experience has been limited.

Usage in Children: At the present time, not recommended for patients under 12 years of age because of lack of experience with use in children.

PRECAUTIONS: Schizophrenic patients may develop increased symptoms of psychosis; patients with paranoid symptomatology may have an exaggeration of such symptoms; manic depressive patients may experience a shift to the manic phase. In these circumstances the dose of amitriptyline may be reduced or a major tranquilizer may be administered concurrently.

Closely supervise and carefully adjust dosage in concomitant use with anticholinergic or sympathomimetic drugs, including combination of epinephrine and local anesthetics. Paralytic ileus may occur with concomitant use of tricyclic antidepressants and anticholinergic-type drugs. Caution is advised if used concurrently with large doses of ethchlorvynol since transient delirium has been reported when one gram of that drug and 75 to 150 mg of amitriptyline HCl were administered. Amitriptyline may enhance response to alcohol and the effects of barbiturates and other CNS depressants. The possibility of suicide in depressed patients remains during treatment and until significant remission occurs. Potentially suicidal patients should not have easy access to large quantities of the drug. Prescriptions should be written for the smallest amount feasible. Limit concurrent administration of this drug and electroshock therapy to patients for whom it is essential since the hazards associated with such therapy may be increased. Discontinue this drug, when possible, several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported. Use amitriptyline with caution in presence of impaired liver function.

ADVERSE REACTIONS: NOTE: A few of the adverse reactions listed below have not been reported with this specific drug, but each of the reactions should be considered when administering amitriptyline because of pharmacological similarities among tricyclic antidepressants.

Cardiovascular: Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, and stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness; tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; and syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, increased intraocular pressure, constipation, paralytic ileus, urinary retention, and dilatation of the urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, and edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, leukopenia, eosinophilia, purpura, and thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, and black tongue. Rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, and elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, and alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache, and malaise. These are not indicative of addiction.

For full prescribing information, consult package insert.

HOW SUPPLIED: Available for oral administration in tablets providing 10, 25, 50, 75, and 100 mg amitriptyline hydrochloride. The 10, 25, and 50 mg tablets are available in bottles of 100 and 1000. The 75 and 100 mg tablets are available in bottles of 100.

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CAPSULES,
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Brief Summary

ABBOTT

ACTIONS — Pharmacologically, clorazepate dipotassium has the characteristics of the benzodiazepines. It has depressant effects on the central nervous system. The primary metabolite, nordiazepam, quickly appears in the blood stream. The serum half-life is about 2 days. The drug is metabolized in the liver and excreted primarily in the urine. (See CLINICAL PHARMACOLOGY section.)

INDICATIONS — TRANXENE is indicated for the symptomatic relief of anxiety associated with anxiety neurosis, in other psychoneuroses in which anxiety symptoms are prominent features, and as an adjunct in disease states in which anxiety is manifested.

TRANXENE is indicated for the symptomatic relief of acute alcohol withdrawal.

The effectiveness of TRANXENE in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should reassess periodically the usefulness of the drug for the individual patient.

CONTRAINDICATIONS — TRANXENE is contraindicated in patients with a known hypersensitivity to the drug, and in those with acute narrow angle glaucoma.

WARNINGS — TRANXENE is not recommended for use in depressive neuroses or in psychotic reactions.

Patients on TRANXENE should be cautioned against engaging in hazardous occupations requiring mental alertness, such as operating dangerous machinery including motor vehicles.

Since TRANXENE has a central nervous system depressant effect, patients should be advised against the simultaneous use of other CNS-depressant drugs, and cautioned that the effects of alcohol may be increased.

Because of the lack of sufficient clinical experience, TRANXENE is not recommended for use in patients less than 18 years of age.

Physical and Psychological Dependence:

Withdrawal symptoms (similar in character to those noted with barbiturates and alcohol) have occurred following abrupt discontinuance of clorazepate. Symptoms of nervousness, insomnia, irritability, diarrhea, muscle aches and memory impairment have followed abrupt withdrawal after long-term use of high dosage.

Caution should be observed in patients who are considered to have a psychological potential for drug dependence.

Evidence of drug dependence has been observed in dogs and rabbits which was characterized by convulsive seizures when the drug was abruptly withdrawn or the dose was reduced; the syndrome in dogs could be abolished by administration of clorazepate.

Usage in Pregnancy:

An increased risk of congenital malformations associated with the use of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) during the first trimester of pregnancy has been suggested in several studies. TRANXENE, a benzodiazepine derivative, has not been studied adequately to determine whether it, too, may be associated with an increased risk of fetal abnormality. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physician about the desirability of discontinuing the drug.

Usage during Lactation:

TRANXENE should not be given to nursing mothers since it has been reported that nordiazepam is excreted in human breast milk.

PRECAUTIONS — In those patients in which a degree of depression accompanies the anxiety, suicidal tendencies may be present and protective measures may be required. The least amount of drug that is feasible should be available to the patient.

Patients on TRANXENE for prolonged periods should have blood counts and liver function tests periodically. The usual precautions in treating patients with impaired renal or hepatic function should also be observed.

In elderly or debilitated patients, the initial dose should be small, and increments should be made gradually, in accordance with the response of the patient, to preclude ataxia or excessive sedation.

ADVERSE REACTIONS — The side effect most frequently reported was drowsiness. Less commonly reported (in descending order of occurrence) were: dizziness, various gastrointestinal complaints, nervousness, blurred vision, dry mouth, headache, and mental confusion. Other side effects included insomnia, transient skin rashes, fatigue, ataxia, genitourinary complaints, irritability, diplopia, depression and slurred speech.

There have been reports of abnormal liver and kidney function tests and of decrease in hematocrit.

Decrease in systolic blood pressure has been observed.

DOSAGE AND ADMINISTRATION

For the symptomatic relief of anxiety:

TRANXENE is administered orally. The capsules may be given in divided doses. The usual daily dose is 30 mg. The dose should be adjusted gradually within the range of 15 to 60 mg. daily in accordance with the response of the patient. In elderly or debilitated patients it is advisable to initiate treatment at a daily dose of 7.5 to 15 mg.

TRANXENE capsules may also be administered as a single dose daily at bedtime; the recommended initial dose is 15 mg. After the initial dose, the response of the patient may require adjustment of subsequent dosage. Lower doses may be indicated in the elderly patient. Drowsiness may occur at the initiation of treatment and with dosage increment.

TRANXENE-SD tablets (22.5 mg.) may be administered as a single dose every 24 hours. This tablet is intended as an alternate dosage form for the convenience of patients stabilized on a dose of 7.5 mg. capsules three times a day. TRANXENE-SD tablets should not be used to initiate therapy.

TRANXENE-SD HALF STRENGTH tablets (11.25 mg.) may be administered as a single dose every 24 hours.

For the symptomatic relief of acute alcohol withdrawal:

Recommended schedule: 1st 24 hours, 30 mg. TRANXENE initially, followed by 30 to 60 mg. in divided doses; 2nd 24 hours, 45 to 90 mg. in divided doses; 3rd 24 hours, 22.5 to 45 mg. in divided doses; 4th day, 15 to 30 mg. in divided doses. Thereafter gradually reduce to 7.5 to 15 mg. daily, and discontinue as soon as condition is stable. Maximum daily dose is 90 mg. Avoid excessive reductions in total drug on successive days.

DRUG INTERACTIONS — If TRANXENE is to be combined with other drugs acting on the central nervous system, careful consideration should be given to the pharmacology of the agents to be employed. Animal experience indicates that TRANXENE prolongs the sleeping time after hexobarbital or after ethyl alcohol, increases the inhibitory effects of chlorpromazine, but does not exhibit monoamine oxidase inhibition. Clinical studies have shown increased sedation with concurrent hypnotic medications. The actions of the benzodiazepines may be potentiated by barbiturates, narcotics, phenothiazines, monoamine oxidase inhibitors or other antidepressants.

If TRANXENE is used to treat anxiety associated with somatic disease states, careful attention must be paid to possible drug interaction with concomitant medication.

MANAGEMENT OF OVERDOSAGE — Overdosage is usually manifested by varying degrees of CNS depression ranging from slight sedation to coma. As in the management of overdosage with any drug, it should be borne in mind that multiple agents may have been taken.

There are no specific antidotes for the benzodiazepines. The treatment of overdosage should consist of the general measures employed in the management of overdosage of any CNS depressant. Gastric evacuation either by the induction of emesis, lavage, or both, should be performed immediately. General supportive care, including frequent monitoring of the vital signs and close observation of the patient, is indicated. Hypotension, though rarely reported, may occur with large overdoses. In such cases the use of agents such as Levophed® (levarterenol) or Aramine® (metaraminol) should be considered.

While reports indicate that individuals have survived overdoses of TRANXENE (clorazepate dipotassium) as high as 450 to 675 mg., these doses are not necessarily an accurate indication of the amount of drug absorbed since the time interval between ingestion and the institution of treatment was not always known. Sedation in varying degrees was the most common physiological manifestation of TRANXENE overdosage. Deep coma when it occurred was usually associated with the ingestion of other drugs in addition to TRANXENE.

CLINICAL PHARMACOLOGY — Studies in healthy men have shown that TRANXENE has depressant effects on the central nervous system. Prolonged administration of single daily doses as high as 120 mg. was without toxic effects. Abrupt cessation of high doses was followed in some patients by nervousness, insomnia, irritability, diarrhea, muscle aches, or memory impairment.

Absorption — Excretion:

After oral administration of TRANXENE, there is essentially no circulating parent drug. Nordiazepam, its primary metabolite, quickly appears in the blood stream. In 2 volunteers given 15 mg. (50 μC) of ^{14}C-TRANXENE, about 80% was recovered in the urine and feces within 10 days. Excretion was primarily in the urine with about 1% excreted per day on day 10.

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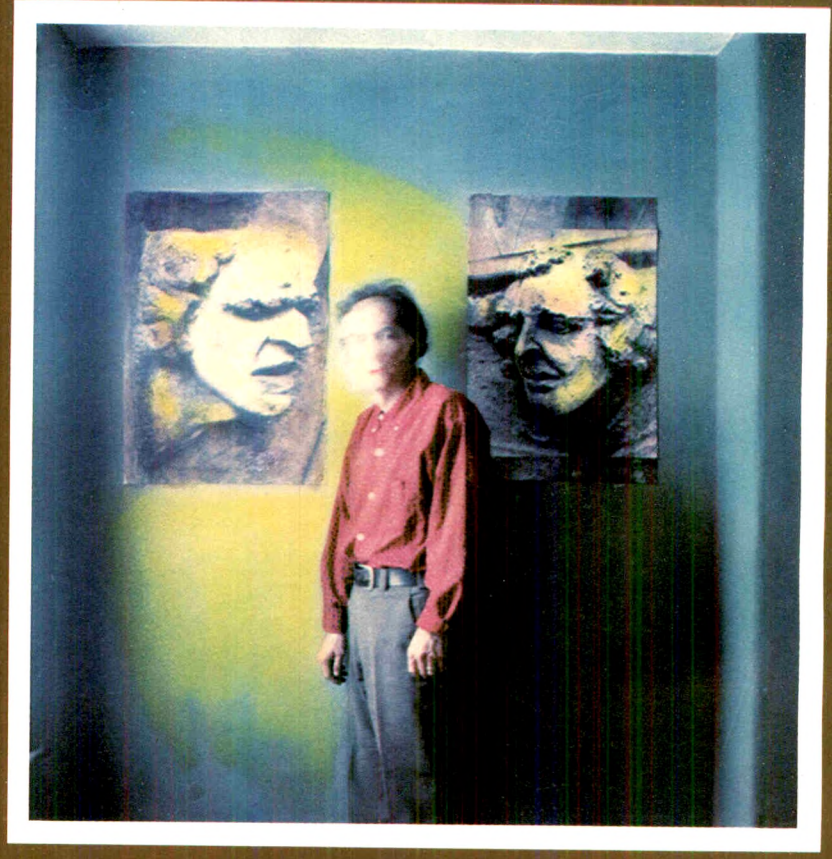
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Warnings: Generally avoid using in patients hypersensitive (e.g., blood dyscrasias, jaundice) to any phenothiazine. Caution patients about activities requiring alertness (e.g., operating vehicles or machinery), especially during the first few days' therapy.

Use in pregnancy only when necessary for patient's welfare.

Precautions: Use cautiously in angina. Avoid high doses and parenteral administration when cardiovascular system is impaired. Antiemetic effect may mask signs of toxic drug overdosage or physical disorders. Additive effect is possible with other C.N.S. depressants. Prolonged administration of high doses may result in cumulative effects with severe C.N.S. or vasomotor symptoms. If retinal changes occur, discontinue drug. Agranulocytosis, thrombocytopenia, pancytopenia, anemia, cholestatic jaundice, liver damage have been reported.

Patients on long-term therapy, especially high doses, should be evaluated periodically for possible adjustment or discontinuance of drug therapy.

Adverse Reactions: Drowsiness, dizziness, skin reactions, rash, dry mouth, insomnia, amenorrhea, fatigue, muscular weakness, anorexia, lactation, blurred vision. Neuromuscular (extrapyramidal) reactions: motor restlessness, dystonias, pseudo-parkinsonism, persistent tardive dyskinesia.

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Partial List of Topics

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Depression simulating organic brain disease — diagnosis and lithium treatment of manic-depressive disorder in the retarded — delineation of an MMPI symptom pattern unique to lithium responders

Treatment, Management

Depression treated with imipramine and ECT — possible cardiovascular effect of lithium — lithium maintenance: factors affecting outcome — efficacy of drugs and psychotherapy in the treatment of acu depressive episodes

Etiology, Phenomenology

Life events at the onset of bipolar affective illness childhood depression: interpersonal interactions a depressive phenomena — drug abuse among alc holic women — psychiatric problems in women d ing alcoholic detoxification — life events, coping, stress, and depressive symptomatology

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This section contains very brief articles (maximum of 1,000 words, 10 references, 1 table, and *no figures*) reporting new research findings, including preliminary data from pilot studies, and case reports that 1) describe new syndromes, 2) cast a new light on established ones, 3) indicate a new therapeutic procedure of potential value, or 4) describe adverse effects or previously unreported complications of drugs or therapeutic interventions. Because of the stringent criteria for this section, authors can expect more rapid publication than is possible in other sections. Submitted papers that substantially exceed the stated maximum length or contain figures will be returned to the author unreviewed.

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The précis should be up to 100 words for regular articles and Brief Communications (no précis is used in the Clinical and Research Reports section). The précis should be a single paragraph using complete, connected sentences, active verbs, and the third person. In most cases, the précis replaces a summary.

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Headings and subheadings should be inserted at reasonable intervals in all types of papers. Footnotes to text material should be typed on a separate page at the end of the manuscript. Summaries are rarely desirable, although a final "Comment" or "Conclusions" section may be used. The body of the paper should be written in the active voice and first person where appropriate.

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Drugs. Generic rather than trade names of drugs should be used; trade names may be given parenthetically if necessary.

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References should be restricted to closely pertinent material; a complete review of the literature is rarely desirable. *Accuracy of citation is the author's responsibility.* References should conform exactly to the original spelling, accents, punctuation, etc. Authors should be sure that all references listed have been cited in text; no bibliographies can be used.

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1. Berne E: Principles of Group Treatment. New York, Oxford University Press, 1966, p 26

2. Blackwell B, Marley E, Price J, et al: Hypertensive interactions between monoamine oxidase inhibitors and foodstuffs. Br J Psychiatry 113:349–365, 1967
3. Gold PW, Goodwin FK: Urinary free cortisol in depression and mania. Presented at the 130th annual meeting of the American Psychiatric Association, Toronto, Ont, Canada, May 2–6, 1977
4. Brosin H: Communication systems of the consultation process, in The Psychiatric Consultation. Edited by Mendel W, Solomon P. New York, Grune & Stratton, 1968

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Contraindications. Navane is contraindicated in patients with circulatory collapse, comatose states, central nervous system depression due to any cause, and blood dyscrasias. Navane is contraindicated in individuals who have shown hypersensitivity to the drug. It is not known whether there is a cross-sensitivity between the thioxanthenes and the phenothiazine derivatives, but this possibility should be considered.

Warnings. *Usage in Pregnancy*—Safe use of Navane during pregnancy has not been established. Therefore, this drug should be given to pregnant patients only when, in the judgment of the physician, the expected benefits from the treatment exceed the possible risks to mother and fetus. Animal reproduction studies and clinical experience to date have not demonstrated any teratogenic effects.

In the animal reproduction studies with Navane, there was some decrease in conception rate and litter size, and an increase in resorption rate in rats and rabbits, changes which have been similarly reported with other psychotropic agents. After repeated oral administration to rats (5 to 15 mg/kg/day), rabbits (3 to 50 mg/kg/day), and monkeys (1 to 3 mg/kg/day) before and during gestation, no teratogenic effects were seen. (See Precautions.)

Usage in Children—The use of Navane in children under 12 years of age is not recommended because safety and efficacy in the pediatric age group have not been established.

As is true with many CNS drugs, Navane may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery, especially during the first few days of therapy. Therefore, the patient should be cautioned accordingly.

As in the case of other CNS-acting drugs, patients receiving Navane should be cautioned about the possible additive effects (which may include hypotension) with CNS depressants and with alcohol.

Precautions. An antiemetic effect was observed in animal studies with Navane; since this effect may also occur in man, it is possible that Navane may mask signs of overdosage of toxic drugs and may obscure conditions such as intestinal obstruction and brain tumor.

In consideration of the known capability of Navane and certain other psychotropic drugs to precipitate convulsions, extreme caution should be used in patients with a history of convulsive disorders or those in a state of alcohol withdrawal since it may lower the convulsive threshold. Although Navane potentiates the actions of the barbiturates, the dosage of the anticonvulsant therapy should not be reduced when Navane is administered concurrently.

Caution as well as careful adjustment of the dosage is indicated when Navane is used in conjunction with other CNS depressants other than anticonvulsant drugs.

Though exhibiting rather weak anticholinergic properties, Navane should be used with caution in patients who are known or suspected to have glaucoma, or who might be exposed to extreme heat, or who are receiving atropine or related drugs.

Use with caution in patients with cardiovascular disease.

Also, careful observation should be made for pigmentary retinopathy, and lenticular pigmentation (fine lenticular pigmentation has been noted in a small number of patients treated with Navane for prolonged periods). Blood dyscrasias (agranulocytosis, pancytopenia, thrombocytopenic purpura), and liver damage (jaundice, biliary stasis) have been reported with related drugs.

Undue exposure to sunlight should be avoided. Photosensitive reactions have been reported in patients on Navane.

Intramuscular Administration—As with all intramuscular preparations, Navane Intramuscular should be injected well within the body of a relatively large muscle. The preferred sites are the upper outer quadrant of the buttock (i.e., gluteus maximus) and the mid-lateral thigh.

The deltoid area should be used only if well developed, such as in certain adults and older children, and then only with caution to avoid radial nerve injury. Intramuscular injections should not be made into the lower and mid-thirds of the upper arm. As with all intramuscular injections, aspiration is necessary to help avoid inadvertent injection into a blood vessel.

Adverse Reactions. *Note:* Not all of the following adverse reactions have been reported with Navane (thiothixene). However, since Navane has certain chemical and pharmacologic similarities to the phenothiazines, all of the known side effects and toxicity associated with phenothiazine therapy should be borne in mind when Navane is used.

Cardiovascular effects: Tachycardia, hypotension, lightheadedness, and syncope. In the event hypotension occurs, epinephrine should not be used as a pressor agent since a paradoxical further lowering of blood pressure may result. Nonspecific EKG changes have been observed in some patients receiving Navane. These changes are usually reversible and frequently disappear on continued Navane therapy. The incidence of these changes is lower than that observed with some phenothiazines. The clinical significance of these changes is not known.

CNS effects: Drowsiness, usually mild, may occur although it usually subsides with continuation of Navane therapy. The incidence of sedation appears similar to that of the piperazine group of phenothiazines, but less than that of certain aliphatic phenothiazines. Restlessness, agitation and insomnia have been noted with Navane (thiothixene). Seizures and paradoxical exacerbation of psychotic symptoms have occurred with Navane infrequently.

Hyperreflexia has been reported in infants delivered from mothers having received structurally related drugs.

In addition, phenothiazine derivatives have been associated with cerebral edema and cerebrospinal fluid abnormalities.

Extrapyramidal symptoms, such as pseudo-parkinsonism, akathisia, and dystonia have been reported. Management of these extrapyramidal symptoms depends upon the type and severity. Rapid relief of acute symptoms may require the use of an injectable antiparkinson agent. More slowly emerging symptoms may be managed by reducing the dosage of Navane and/or administering an oral antiparkinson agent.

Persistent Tardive Dyskinesia: As with all antipsychotic agents tardive dyskinesia may appear in some patients on long term therapy or may occur after drug therapy has been discontinued. The risk seems to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities.

There is no known effective treatment for tardive dyskinesia: antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear.

Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked.

It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time, the syndrome may not develop.

Hepatic effects: Elevations of serum transaminase and alkaline phosphatase, usually transient, have been infrequently observed in some patients. No clinically confirmed cases of jaundice attributable to Navane have been reported.

Hematologic effects: As is true with certain other psychotropic drugs, leukopenia and leukocytosis, which are usually transient, can occur occasionally with Navane. Other antipsychotic drugs have been associated with agranulocytosis, eosinophilia, hemolytic anemia, thrombocytopenia and pancytopenia.

Allergic reactions: Rash, pruritus, urticaria, photosensitivity and rare cases of anaphylaxis have been reported with Navane. Undue exposure to sunlight should be avoided. Although not experienced with Navane, exfoliative dermatitis and contact dermatitis (in nursing personnel) have been reported with certain phenothiazines.

Endocrine disorders: Lactation, moderate breast enlargement and amenorrhea have occurred in a small percentage of females receiving Navane. If persistent, this may necessitate a reduction in dosage or the discontinuation of therapy. Phenothiazines have been associated with false positive pregnancy tests, gynecomastia, hypoglycemia, hyperglycemia, and glycosuria.

Autonomic effects: Dry mouth, blurred vision, nasal congestion, constipation, increased sweating, increased salivation, and impotence have occurred infrequently with Navane therapy. Phenothiazines have been associated with miosis, mydriasis, and adynamic ileus.

Other adverse reactions: Hyperpyrexia, anorexia, nausea, vomiting, diarrhea, increase in appetite and weight, weakness or fatigue, polydipsia and peripheral edema.

Although not reported with Navane, evidence indicates there is a relationship between phenothiazine therapy and the occurrence of a systemic lupus erythematosus-like syndrome.

NOTE: Sudden deaths have occasionally been reported in patients who have received certain phenothiazine derivatives. In some cases the cause of death was apparently cardiac arrest or asphyxia due to failure of the cough reflex. In others, the cause could not be determined nor could it be established that death was due to phenothiazine administration.

Dosage and Administration. Dosage of Navane should be individually adjusted depending on the chronicity and severity of the condition. In general, small doses should be used initially and gradually increased to the optimal effective level, based on patient response.

Some patients have been successfully maintained on once-a-day Navane therapy.

Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

Navane Intramuscular Solution—For Intramuscular Use Only. Where more rapid control and treatment of acute behavior is desirable, the intramuscular form of Navane may be indicated. It is also of benefit where the very nature of the patient's symptomatology, whether acute or chronic, renders oral administration impractical or even impossible.

For treatment of acute symptomatology or in patients unable or unwilling to take oral medication, the usual dose is 4 mg of Navane Intramuscular administered 2 to 4 times daily. Dosage may be increased or decreased depending on response. Most patients are controlled on a total daily dosage of 16 to 20 mg. The maximum recommended dosage is 30 mg/day. An oral form should supplant the injectable form as soon as possible. It may be necessary to adjust the dosage when changing from the intramuscular to oral dosage for Dosage recommendations for Navane Capsules and C centrate appear in the following paragraphs.

Navane Capsules; Navane Concentrate—In milder cor tions, an initial dose of 2 mg three times daily. If indicate subsequent increase to 15 mg/day total daily dose is of effective.

In more severe conditions, an initial dose of 5 mg tv daily.

The usual optimal dose is 20 to 30 mg daily. If indicated increase to 60 mg/day total daily dose is often effect Exceeding a total daily dose of 60 mg rarely increases beneficial response.

Overdosage. Manifestations include muscular twitch drowsiness, and dizziness. Symptoms of gross overdosage include CNS depression, rigidity, weakness, torticollis, tren salivation, dysphagia, hypotension, disturbances of gait coma.

Treatment: Essentially symptomatic and supportive. Navane oral, early gastric lavage is helpful. For Navane oral Intramuscular, keep patient under careful observation maintain an open airway, since involvement of the ex pyramidal system may produce dysphagia and respirat difficulty in severe overdosage. If hypotension occurs, standard measures for managing circulatory shock should used (I.V. fluids and/or vasoconstrictors).

If a vasoconstrictor is needed, levarterenol and phen ephrine are the most suitable drugs. Other pressor age including epinephrine, are not recommended, sin phenothiazine derivatives may reverse the usual pressor act of these agents and cause further lowering of blood pressure

If CNS depression is present, recommended stimula include amphetamine, dextroamphetamine, or caffeine sodium benzoate. Picrotoxin or pentylenetetrazol should avoided. Extrapyramidal symptoms may be treated w antiparkinson drugs.

There are no data on the use of peritoneal or hemodialy but they are known to be of little value in phenothia intoxication.

How Supplied. Navane (thiothixene) is available as caps containing 1 mg, 2 mg, 5 mg, and 10 mg of thiothixene bottles of 100 and 1,000. Navane is also available as caps containing 20 mg of thiothixene in bottles of 100 and 500.

Navane (thiothixene hydrochloride) Concentrate available in 120 ml (4 oz.) bottles with an accompany dropper calibrated at 2 mg, 4 mg, 5 mg, 6 mg, 8 mg, 10 mg, and in 30 ml (1 oz.) bottles with an accompany dropper calibrated at 2 mg, 4 mg, and 5 mg. Each ml c tains thiothixene hydrochloride equivalent to 5 mg thiothixene. Contains alcohol, U.S.P. 7.0% v/v (small unavoidable).

Navane (thiothixene hydrochloride) Intramuscular solu is available in a 2 ml amber glass vial in packages of 10. Each contains thiothixene hydrochloride equivalent to 2 mg thiothixene, dextrose 5% w/v, benzyl alcohol 0.9% w/v, propyl gallate 0.02% w/v.

References. 1. Stotsky BA: Relative efficacy of parent haloperidol and thiothixene for the emergency treatmen acutely excited and agitated patients. *Dis Nerv Syst* 38:967-9 1977. 2. Bressler B, Friedel RO: A comparison betw chlorpromazine and thiothixene in a Veterans Administra hospital population. *Psychosomatics* 12:275-277, 1971. 3. Der HCB, Turns D: Double blind comparison of thiothixene trifluoperazine in acute schizophrenia. *Psychosomatics* 13: 104, 1972. 4. Sterlin C, Ban TA, Lehman HE, Saxena BM: place of thiothixene in the treatment of schizophrenic patie *Can Psychiatr Assoc J* 15:3-4, 1970. 5. Brauzer B, Goldstein Comparative effects of intramuscular thiothixene and fluoperazine in psychotic patients. *J Clin Pharmacol* 8:400-4 1968. 6. Engelhardt DM, Rudorfer L: The chronic sch phrenic outpatient in an urban community: Social and vo tional adjustment. Presented as a Scientific Exhibit at the 13 Annual Meeting, American Psychiatric Association, Toro Canada, May 2-6, 1977. 7. Itil TM, Unverdi C, Wohlrab Larsen V, Levitt J: Drug therapy of psychosis associated w organic brain syndrome, presented as a scientific exhibit at American Public Health Association Centennial, Atlantic New Jersey, November 12-16, 1972. 8. Dillenkoffer RL, e Electrocardiographic evaluations of schizophrenic patie Presented as a Scientific Exhibit at the 125th Annual Meetin the American Psychiatric Association, Dallas, Texas, May 1972. 9. Goldstein B, Weiner D, Banas F: Clinical evaluation thiothixene in chronic ambulatory schizophrenic patients Lehmann HE, Ban TA (eds): *Modern Problems in Pharmaco chiatry*. Basel, S Karger, 1969, vol 2, pp 45-52.

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HUMAN AUTOEROTIC PRACTICES

Foreword by Dr. Albert Ellis

This comprehensive volume which reflects contemporary thinking, current research, and clinical findings of eminent sexologists, such as Alex Comfort, Havelock Ellis, Helen Kaplan, Masters and Johnson, and James L. McCary, as well as sex therapists, psychotherapists, and clinicians on the subject of autoeroticism, is a landmark work. Not only do the authors make it clear that the practice of autoeroticism is normal and healthy in females and males of any age and in many different circumstances, but some psychotherapists also state that the absence of masturbatory behavior during one's life may lead to sexual problems.

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Warnings: May block the antihypertensive action of guanethidine or similarly acting compounds. Should be used with caution in patients with a history of seizures or a history of urinary retention, or with angle-closure glaucoma or increased intraocular pressure; in patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely; arrhythmias, sinus tachycardia and prolongation of the conduction time have been reported, particularly with high doses; myocardial infarction and stroke have been reported with drugs of this class. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. May impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle. In patients who use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage. Safe use during pregnancy and lactation has not been established; in pregnant patients, nursing mothers, or women who may become pregnant, weigh possible benefits against possible hazards to mother and child. Not recommended for patients under 12 years of age.

Precautions: Schizophrenic patients may develop increased symptoms of psychosis; patients with paranoid symptomatology may have an exaggeration of such symptoms; manic depressive patients may experience a shift to the manic phase. In these circumstances, the dose of amitriptyline HCl may be reduced or a major tranquilizer, such as perphenazine, may be administered concurrently.

When given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required; paralytic ileus may occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs. Use cautiously in patients receiving large doses of ethchlorvynol, since transient delirium has been reported on concurrent administration. May enhance the response to alcohol and the effects of barbiturates and other CNS depressants. The possibility of suicide in depressed patients remains until significant remission occurs. Potentially suicidal patients should not have access to large quantities of this drug. Prescriptions should be written for the smallest amount feasible. Concurrent electroshock therapy may increase the hazards associated with such therapy; such treatment should be limited to patients for whom it is essential. When possible, discontinue the drug several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported. Use with caution in patients with impaired liver function.

Adverse Reactions: *Note:* Included in this listing are a few adverse reactions not reported with this specific drug. However, pharmacological similarities among the tricyclic antidepressant drugs require that each reaction be considered when amitriptyline is administered. *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, increased intraocular pressure, constipation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, leukopenia, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, black tongue, rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache, and malaise; these are not indicative of addiction.

Overdosage: Hospitalize as soon as possible all patients suspected of having taken an overdose. Treatment is symptomatic and supportive. In addition, the intravenous administration of 1 to 3 mg physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage should be repeated as required, particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine.

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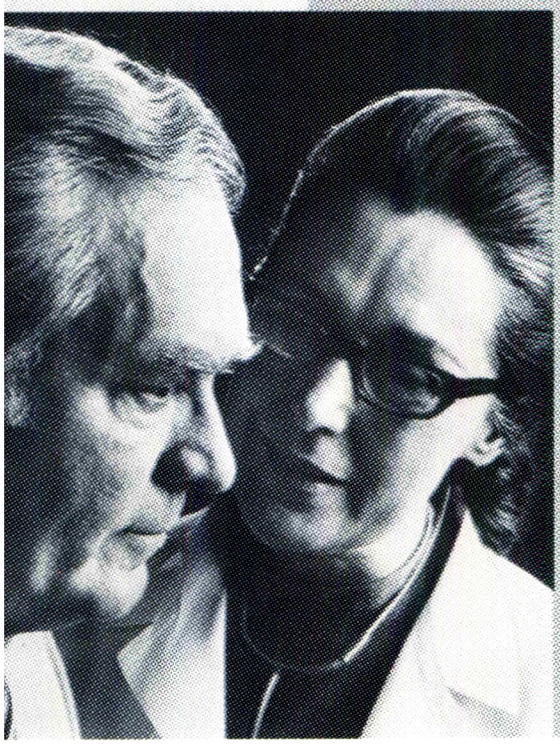
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Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (*e.g.*, excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.

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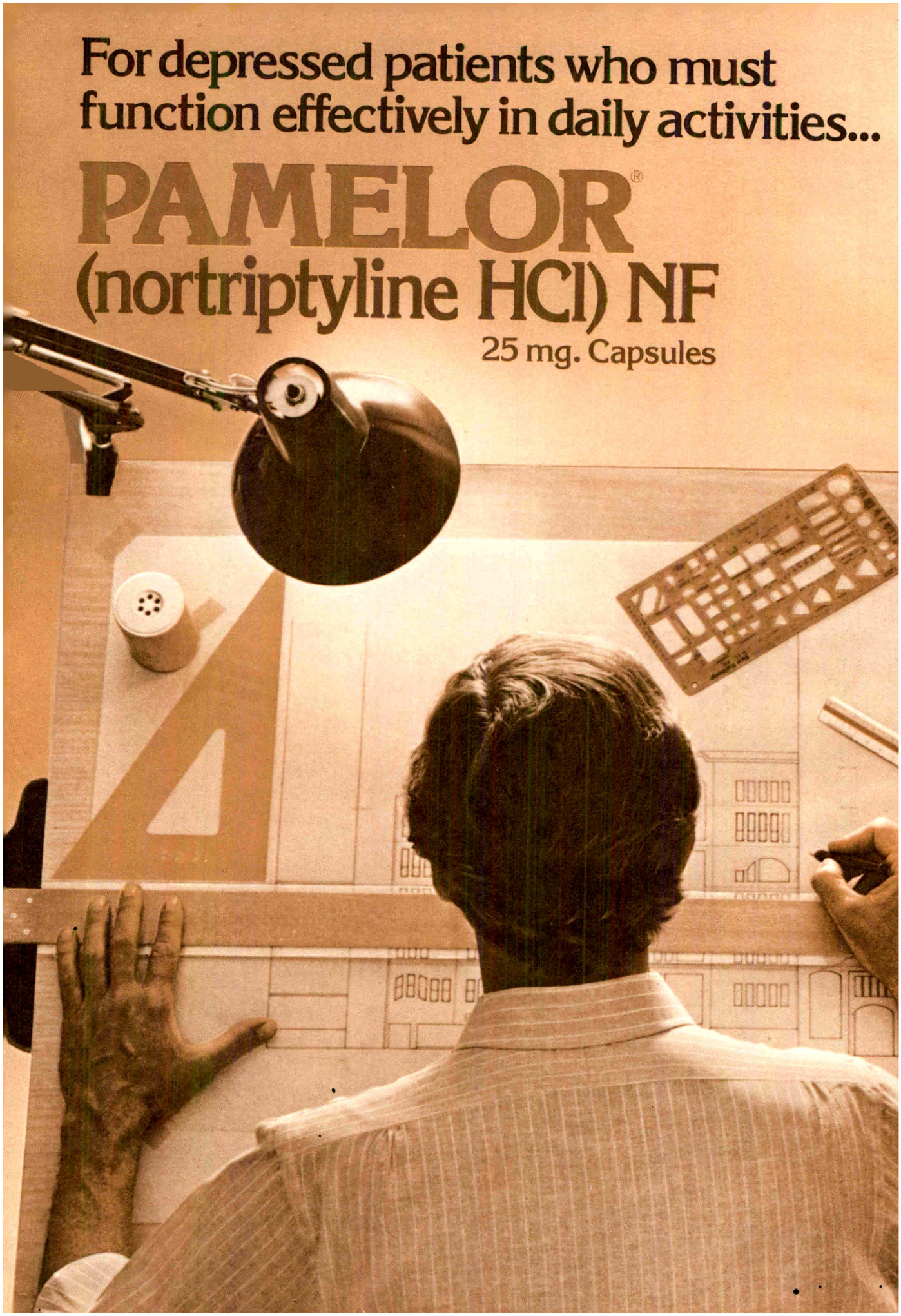
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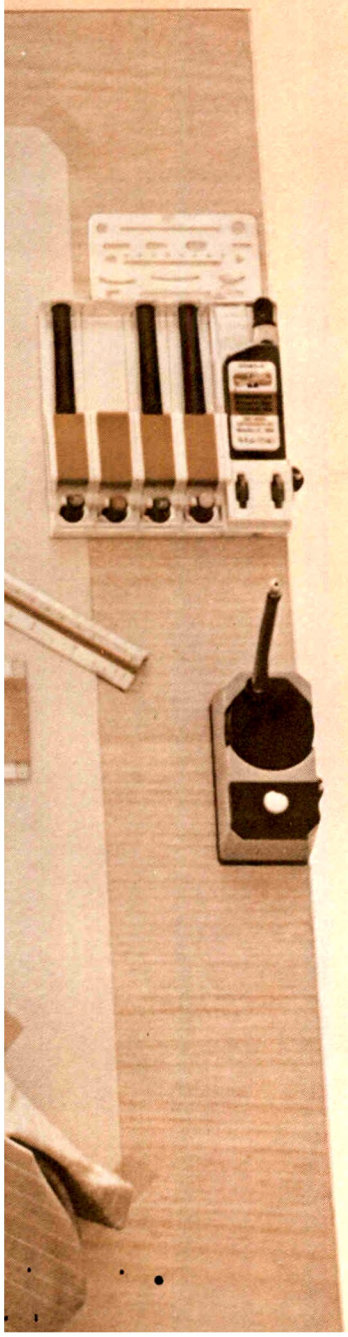
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Warnings: Use with caution in patients with cardiovascular disease because of tendency to produce sinus tachycardia and prolong conduction time. Myocardial infarction, arrhythmia, and strokes have occurred. May block antihypertensive action of guanethidine and similar agents. Because of anticholinergic activity, use cautiously in patients with glaucoma or a history of urinary retention. Patients with a history of seizures should be followed closely because the drug is known to lower the convulsive threshold. Great care is required for hyperthyroid patients and those taking thyroid medication because of possible development of cardiac arrhythmia. Caution patients about possibility of impaired mental and/or physical ability to operate a motor vehicle or dangerous machinery. Response to alcoholic beverages may be exaggerated and may lead to suicidal attempts. Safe use during pregnancy, lactation, and women of childbearing potential has not been established and the drug should not be given unless clinical situation warrants potential risk. Not recommended for use in children.

Precautions: Psychotic symptoms may be exacerbated in schizophrenic patients. Increased anxiety and agitation may occur in overactive or agitated patients. Manic-depressive patients may experience shift to manic phase. Hostility may be aroused. Concomitant administration of reserpine may produce a "stimulating" effect. Watch for possible epileptiform seizures during treatment. Use cautiously with anticholinergic or sympathomimetic drugs. Concurrent electroconvulsive therapy may increase hazards associated with nortriptyline HCl. When possible, discontinue drug several days prior to surgery. Potentially suicidal patients require supervision and protective measures during therapy. Prescriptions should be limited to the least possible quantity. Both elevation and lowering of blood sugar levels have been reported.

Adverse Reactions: Note: The pharmacologic similarities among the tricyclic antidepressant drugs require that each of the following reactions be considered when nortriptyline is administered.

Cardiovascular: Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke.

Psychiatric: Confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia, panic, nightmares; hypomania; exacerbation of psychosis.

Neurologic: Numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures, alteration in EEG patterns; tinnitus.

Anticholinergic: Dry mouth and rarely, associated sublingual adenitis; blurred vision, disturbance of accommodation, mydriasis; constipation, paralytic ileus; urinary retention, delayed micturition, dilation of the urinary tract.

Allergic: Skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight); edema (general or of face and tongue), drug fever, cross-sensitivity with other tricyclic drugs.

Hematologic: Bone-marrow depression, including agranulocytosis; eosinophilia; purpura; thrombocytopenia.

Gastrointestinal: Nausea and vomiting, anorexia, epigastric distress, diarrhea, peculiar taste, stomatitis, abdominal cramps, black-tongue.

Endocrine: Gynecomastia in the male, breast enlargement and galactorrhea in the female; increased or decreased libido, impotence, testicular swelling; elevation or depression of blood sugar levels.

Other: Jaundice (simulating obstructive); altered liver function; weight gain or loss; perspiration; flushing; urinary frequency, nocturia; drowsiness, dizziness, weakness, fatigue; headache; parotid swelling; alopecia.

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Precautions: Liver function tests should be performed periodically during therapy with Cylert. The drug should be discontinued if abnormalities are revealed and confirmed by follow-up tests. (See ADVERSE REACTIONS regarding reports of abnormal liver function tests and jaundice.)

Cylert should be administered with caution to patients with significantly impaired hepatic or renal function.

The interaction of Cylert with other drugs has not been studied in humans. Patients who are receiving Cylert concurrently with other drugs, especially drugs with CNS activity, should be monitored carefully.

Cylert failed to demonstrate a potential for self-administration in primates. However, the pharmacologic similarity of pemoline to other psychostimulants with known dependence liability suggests that psychological and/or physical dependence might also occur with Cylert. There have been isolated reports of transient psychotic symptoms occurring in adults following the long-term misuse of excessive oral doses of pemoline. Cylert should be given with caution to emotionally unstable patients who may increase the dosage on their own initiative.

Usage during Pregnancy and Lactation: The safety of Cylert (pemoline) for use during pregnancy and lactation has not been established.

Fertility, reproduction, and teratology studies were conducted in laboratory animals. Pemoline, in doses of 18.75 or 37.5 mg./kg./day, had no effect on the fertility of male or female rats. The drug, when given to pregnant rats (from gestation day 15 through weaning) and to rabbits (from gestation days 6-18) at these same dosage levels, produced no teratogenic or embryotoxic effects, and had no effect on the viability of the young at birth. However, increased incidences of stillbirths and cannibalization were observed when pemoline was given to rats at these dosage levels, beginning 14 days prior to conception.

Adverse Reactions: Insomnia is the most frequently reported side effect of Cylert; it usually occurs early in therapy, prior to an optimum therapeutic response. In the majority of cases it is transient in nature or responds to a reduction in dosage.

Anorexia with weight loss may occur during the first weeks of therapy. In the majority of cases it is transient in nature; weight gain usually resumes within three to six months.

Stomach ache, skin rashes, increased irritability, mild depression, nausea, dizziness, headache, drowsiness, and hallucinations have been reported.

Elevations of SGOT, SGPT, and serum LDH have occurred in patients taking Cylert, usually after several months of therapy. These effects appear to be reversible upon withdrawal of the drug, and are thought to be manifestations of a delayed hypersensitivity reaction. There have also been a few reports of jaundice occurring in patients taking Cylert; a causal relationship between the drug and this clinical finding has not been established.

There have been reports of dyskinetic movements of the lips, face, and extremities occurring with the use of Cylert. Convulsive seizures have also been reported. A definite causal relationship between Cylert and these reactions has not been established.

Mild adverse reactions appearing early during the course of treatment with Cylert often remit with continuing therapy. If adverse reactions are of a significant or protracted nature, dosage should be reduced or the drug discontinued.

How Supplied: Cylert (pemoline) is supplied as monogrammed, grooved tablets in three dosage strengths:

18.75 mg. tablets (white) in bottles of 100 (**NDC** 0074-6025-13)
37.5 mg. tablets (orange-colored) in bottles of 100 (**NDC** 0074-6057-13)
75 mg. tablets (tan-colored) in bottles of 100 (**NDC** 0074-6073-13)

Cylert Chewable is supplied as monogrammed, grooved tablets in one dosage strength:

37.5 mg. tablets (orange-colored)
in bottles of 100 (**NDC** 0074-6088-13)

ABBOTT

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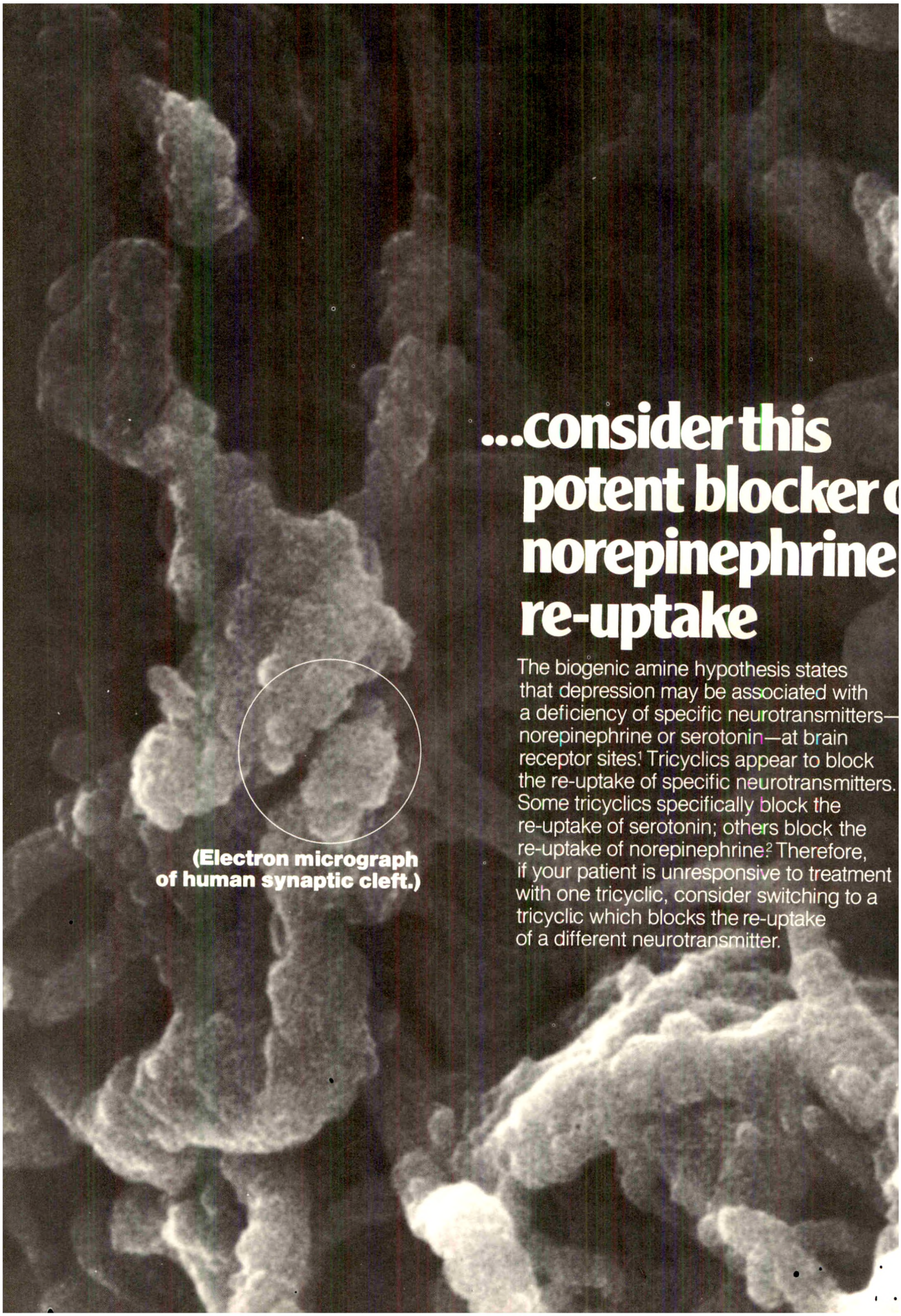
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State_____ Zip_____

AJP


When your patient is clinically depressed...
"...everything I saw was negative."



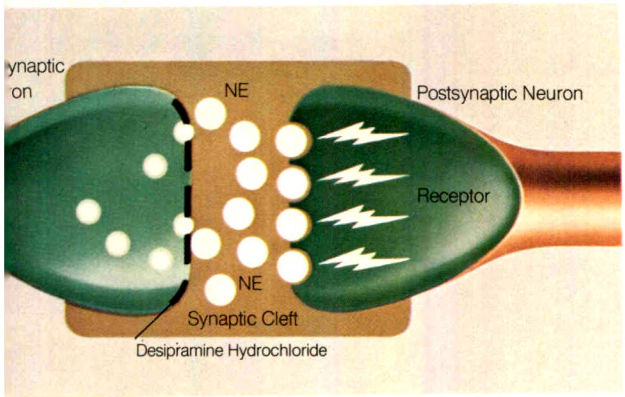
...consider this potent blocker of norepinephrine re-uptake

The biogenic amine hypothesis states that depression may be associated with a deficiency of specific neurotransmitters—norepinephrine or serotonin—at brain receptor sites.[1] Tricyclics appear to block the re-uptake of specific neurotransmitters. Some tricyclics specifically block the re-uptake of serotonin; others block the re-uptake of norepinephrine.[2] Therefore, if your patient is unresponsive to treatment with one tricyclic, consider switching to a tricyclic which blocks the re-uptake of a different neurotransmitter.

(Electron micrograph of human synaptic cleft.)

lorpramin®
lesipramine ydrochloride ablets NF)
,50,75,100,150 mg.

tent blocker norepinephrine re-uptake
cording to current theory[1,3] some clic antidepressants may fail in certain es of depression because they are not iciently potent blockers of norepi- hrine re-uptake. Laboratory studies e shown that desipramine is the most nt inhibitor of norepinephrine ptake.[4] Norpramin may work in some ents when other tricyclic antidepres- ts do not.



onsider it now, onsider it next.

Less anticholinergic activity
(See Warnings section.)
Studies in animals[5] and in normal human subjects[6,7] have shown that desipramine has significantly less anticholinergic activity than amitriptyline or doxepin. The single bond side chain is associated with lower anticholinergic activity than these other leading antidepressants. This may mean:
- less dry mouth
- less blurred vision
- less urinary retention

Helps patients remain calm but active
Norpramin does not usually inhibit normal activity although patients should be cautioned as to driving or operating machinery if drowsiness occurs.

In addition…

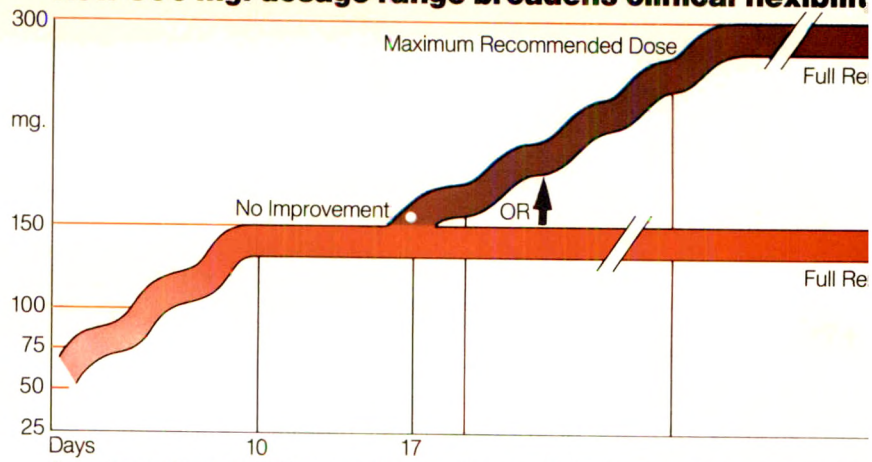
Begins to improve sleep patterns within one week in some patients[8]
- Norpramin helps relieve the sleep disturbances that often accompany depression.
- As the depression is relieved, the difficulty in falling asleep, restlessness, and early morning awakening diminish.

References:
1. Maas, J.W.: Biogenic amines and depression. Biochemical and pharmacological separation of two types of depression. Arch. Gen. Psychiat. 32:1357-1361, 1975. **2.** Mendels, J., Stern, S., and Frazer, A.: Dis. Nerv. Syst. 37:3, 1976. **3.** Davis, J.M.: Central biogenic amines and theories of depression and mania. In, Phenomenology and Treatment of Depression, W.E. Fann, I. Karacan, A.D. Pokorny, and R.L. Williams, Ed., New York, Spectrum Publications, Inc., 1977, pp. 17-32. **4.** Ross, S.B. and Renyi, A.L.: Tricyclic antidepressant agents. 1. Comparison of the inhibition of the uptake of ^{3}H-noradrenaline and ^{14}C-5-hydroxytryptamine in slices and crude synaptosome preparations of the midbrain-hypothalamus region of the rat brain. Acta Pharmacol. (Kobenhavn) 36:382-394, 1975. **5.** Snyder, S.H. and Yamamura, H.I.: Antidepressants and the muscarinic acetylcholine receptor. Arch. Gen. Psychiat. 34:236-239, 1977. **6.** Blackwell, B., Stefopoulos, A., Enders, P., Kuzma, R., and Adolphe, A.: The anticholinergic activity of two tricyclic antidepressants. Amer. J. Psychiat., 135:722-724, June 1978. **7.** Peterson, G.R., Hostetler, R.M., Blackwell, B., Kuzma, R., and Adolphe, A.B.: Effects of the tricyclic antidepressants desipramine and doxepin on anticholinergic and CNS activity in non-depressed volunteers. Neuroscience Abstr. 3:447, 1977. **8.** Zung, W.W.K.: The pharmacology of disordered sleep. A laboratory approach. J.A.M.A. 211:1532-1534, 1970.

(For prescribing information see next page.)

Merrell

Now 300 mg. dosage range broadens clinical flexibilit



A maximum dosage of 300 mg. a day, as a single or divided dose, may now be prescrib Norpramin. Since inadequate dosage is thought to be one of the major causes of treatm failure, the new therapeutic range should prove clinically helpful. Plasma desipramine measurement would constitute the optimal guide to dosage monitoring.

Norpramin®
(desipramine hydrochloride tablets NF)

AVAILABLE ONLY ON PRESCRIPTION

Brief Summary

MECHANISM OF ACTION: Available evidence suggests that many depressions have a biochemical basis in the form of a relative deficiency of neurotransmitters such as norepinephrine and serotonin. Norepinephrine deficiency may be associated with relatively low urinary 3-methoxy-4-hydroxyphenyl glycol (MHPG) levels, while serotonin deficiencies may be associated with low spinal fluid levels of 5-hydroxyindolacetic acid.

While the precise mechanism of action of the tricyclic antidepressants is unknown, a leading theory suggests that they restore normal levels of neurotransmitters by blocking the re-uptake of these substances from the synapse in the central nervous system.

Evidence indicates that the secondary amine tricyclic antidepressants, including Norpramin, may have greater activity in blocking the re-uptake of norepinephrine. Tertiary amine tricyclic antidepressants, such as amitriptyline, may have greater effect on serotonin re-uptake.

Norpramin (desipramine hydrochloride) is not a monoamine oxidase (MAO) inhibitor and does not act primarily as a central nervous system stimulant. It has been found in some studies to have a more rapid onset of action than imipramine. Earliest therapeutic effects may occasionally be seen in 2 to 5 days, but full treatment benefit usually requires 2 to 3 weeks to obtain.

INDICATIONS: Norpramin (desipramine hydrochloride) is indicated for relief of symptoms in various depressive syndromes, especially endogenous depression.

CONTRAINDICATIONS: Desipramine hydrochloride should not be given in conjunction with, or within 2 weeks of, treatment with an MAO inhibitor drug; hyperpyretic crises, severe convulsions, and death have occurred in patients taking MAO inhibitors and tricyclic antidepressants. When Norpramin (desipramine hydrochloride) is substituted for an MAO inhibitor, at least 2 weeks should elapse between treatments. Norpramin should then be started cautiously and should be increased gradually.

The drug is contraindicated in the acute recovery period following myocardial infarction. It should not be used in those who have shown prior hypersensitivity to the drug. Cross sensitivity between this and other dibenzazepines is a possibility.

WARNINGS: 1. Extreme caution should be used when this drug is given in the following situations: a. In patients with cardiovascular disease, because of the possibility of conduction defects, arrhythmias, tachycardias, strokes, and acute myocardial infarction. b. In patients with a history of urinary retention or glaucoma, because of the anticholinergic properties of the drug. c. In patients with thyroid disease or those taking thyroid medication, because of the possibility of cardiovascular toxicity, including arrhythmias. d. In patients with a history of seizure disorder, because this drug has been shown to lower the seizure threshold. **2.** This drug is capable of blocking the antihypertensive effect of guanethidine and similarly acting compounds. **3.** USE IN PREGNANCY: Safe use of desipramine hydrochloride during pregnancy and lactation has not been established; therefore, if it is to be given to pregnant patients, nursing mothers, or women of childbearing potential, the possible benefits must be weighed against the possible hazards to mother and child. Animal reproductive studies have been inconclusive. **4.** USE IN CHILDREN: Norpramin (desipramine hydrochloride) is not recommended for use in children since safety and effectiveness in the pediatric age group have not been established. **5.** The patient should be cautioned that this drug may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery. **6.** In patients who may use alcohol excessively, it should be borne in mind that the potentiation may increase the danger inherent in any suicide attempt or overdosage.

PRECAUTIONS: 1. It is important that this drug be dispensed in the least possible quantities to depressed outpatients, since suicide has been accomplished with this class of drug. Ordinary prudence requires that children not have access to this drug or to potent drugs of any kind; if possible this drug should be dispensed in containers with child-resistant safety closures. Storage of this drug in the home must be supervised responsibly. **2.** If serious adverse effects occur, dosage should be reduced or treatment should be altered. **3.** Norpramin (desipramine hydrochloride) therapy in patients with manic-depressive illness may induce a hypomanic state after the depressive phase terminates. **4.** The drug may cause exacerbation of psychosis in schizophrenic patients. **5.** Close supervision and careful adjustment of dosage are required when this drug is given concomitantly with anticholinergic or sympathomimetic drugs. **6.** Patients should be warned that while taking this drug their response to alcoholic beverages may be exaggerated. **7.** Clinical experience in the concurrent administration of ECT and antidepressant drugs is limited. Thus, if such treatment is essential, the possibility of increased risk relative to benefits should be considered. **8.** The sedative effects of Norpramin and benzodiazepines (*e.g.*, chlordiazepoxide or diazepam) are additive. Both the sedative and anticholinergic effects of the major tranquilizers are additive to those of Norpramin. **9.** This drug should be discontinued as soon as possible prior to elective surgery because of the possible cardiovascular effects. Hypertensive episodes have been observed during surgery in patients taking desipramine hydrochloride. **10.** Both elevation and lowering of blood sugar levels have been reported. **11.** Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during therapy; the drug should be discontinued if there is evidence of pathologic neutrophil depression.

ADVERSE REACTIONS: Note: Included in the following listing are a few adverse reactions that have not been reported with this specific drug. However, the pharmacologic similarities among the tricyclic antidepressant drugs require that each of the reactions be considered when Norpramin (desipramine hydrochloride) is given.

Cardiovascular: hypotension, hypertension, tachycardia, palpitation, arrhythmias, heart block, myocardial infarction, stroke.

Psychiatric: confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia and nightmares; hypomania; exacerbation of psychosis.

Neurologic: numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures; alteration in EEG patterns; tinnitus.

Anticholinergic: dry mouth, and rarely associated sublingual adenitis; blurred vision, disturbance of accommodation, mydriasis, increased intraocular pressure; constipation, paralytic ileus; urinary retention, delayed micturition, dilatation of urinary tract.

Allergic: skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight), edema (of face and tongue or general), drug fever, cross sensitivity with other tricyclic drugs.

Hematologic: bone marrow depressions including agranulocytosis, eosinophilia, purpura, thrombocytopenia.

Gastrointestinal: anorexia, nausea and vomiting, epigastric distress, peculiar taste, abdominal cramps, diarrhea, stomatitis, black tongue.

Endocrine: gynecomastia in the male, breast enl ment and galactorrhea in the female; increas decreased libido, impotence, testicular swelling; e tion or depression of blood sugar levels.

Other: jaundice (simulating obstructive), altered function; weight gain or loss; perspiration, flus urinary frequency, nocturia; parotid swelling; dr ness, dizziness, weakness and fatigue, headache pecia.

Withdrawal Symptoms: Though not indicative of a tion, abrupt cessation of treatment after prolo therapy may produce nausea, headache, and ma

DOSAGE AND ADMINISTRATION: Not recommended fo in children. Lower dosages are recommended for el patients and adolescents. Lower dosages are recommended for outpatients compared to hospita patients, who are closely supervised. Dosage shou initiated at a low level and increased accordir clinical response and any evidence of intoler Following remission, maintenance medication ma required for a period of time and should be at the lo dose that will maintain remission.

Usual Adult Dose: The usual adult dose is 100 to 20 per day. In more severely ill patients, dosage ma further increased gradually to 300 mg./day if neces Dosages above 300 mg./day are not recommende

Dosage should be initiated at a lower level increased according to tolerance and clinical resp

Treatment of patients requiring as much as 30 should generally be initiated in hospitals, where re visits by the physician, skilled nursing care, anc quent electrocardiograms (ECG's) are available.

The best available evidence of impending to from very high doses of Norpramin is prolongation QRS or QT intervals on the ECG. Prolongation of th interval is also significant, but less closely corre with plasma levels. Clinical symptoms of intoler especially drowsiness, dizziness, and postural hyp sion, should also alert the physician to the nee reduction in dosage. Plasma desipramine measure would constitute the optimal guide to dosage mor ing.

Initial therapy may be administered in divided d or a single daily dose.

Maintenance therapy may be given on a once- schedule for patient convenience and compliance.

Adolescent and Geriatric Dose: The usual adolescen geriatric dose is 25 to 100 mg. daily.

Dosage should be initiated at a lower level increased according to tolerance and clinical resp to a usual maximum of 100 mg. daily. In more severe patients, dosage may be further increased to 150 day. Doses above 150 mg./day are not recommend these age groups.

Initial therapy may be administered in divided d or a single daily dose.

Maintenance therapy may be given on a once- schedule for patient convenience and compliance.

OVERDOSAGE: See prescribing information for a dis sion of symptoms and treatment of overdose.

Product Information as of April, 1978

Merrell

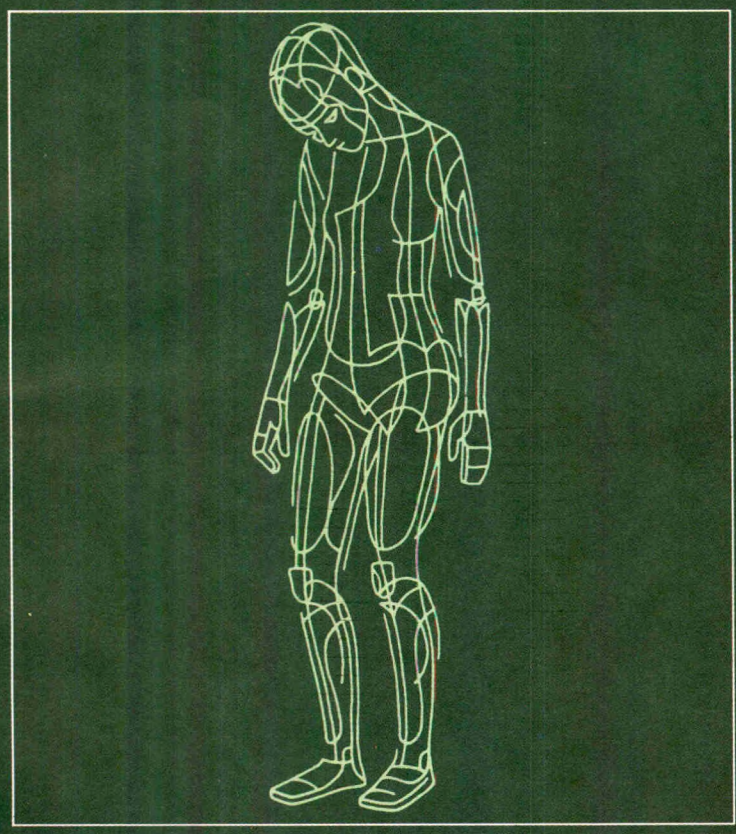
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Because it works so fast–
Because it works so well–
Because it is so well accepted–

Limbitrol®

Provides the well established antidepressant effect of amitriptyline with the proven antianxiety effect of Librium® (chlordiazepoxide HCl)

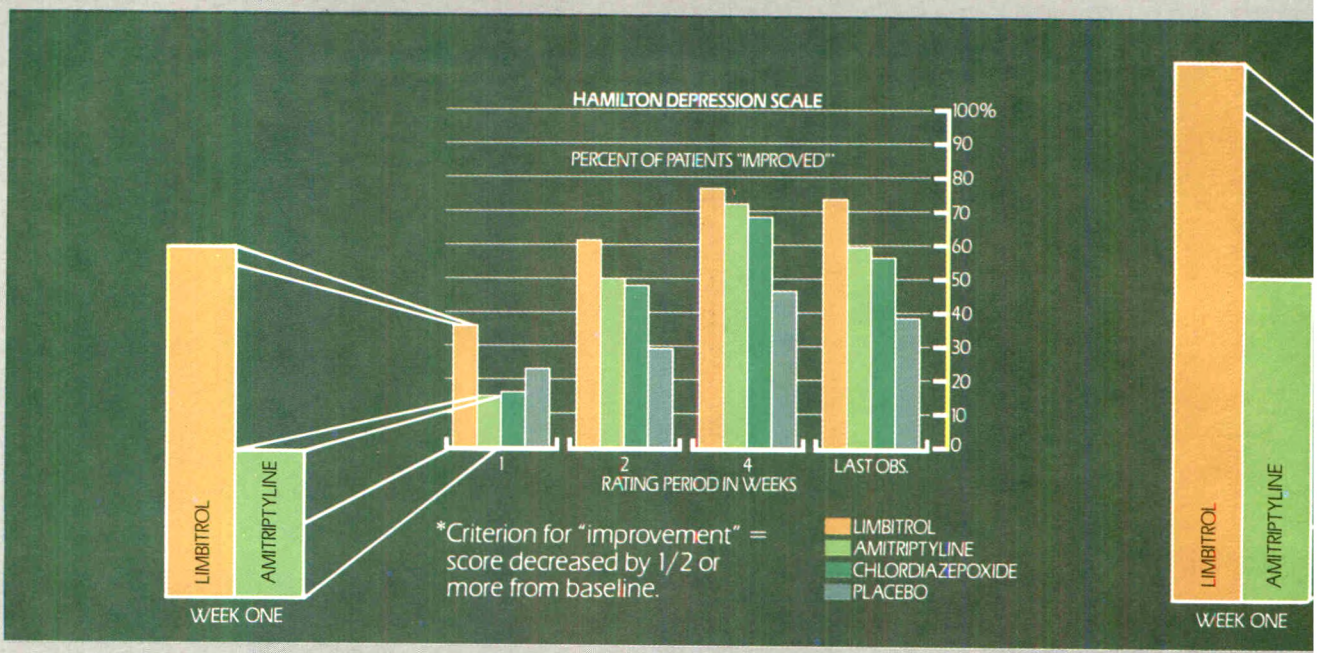


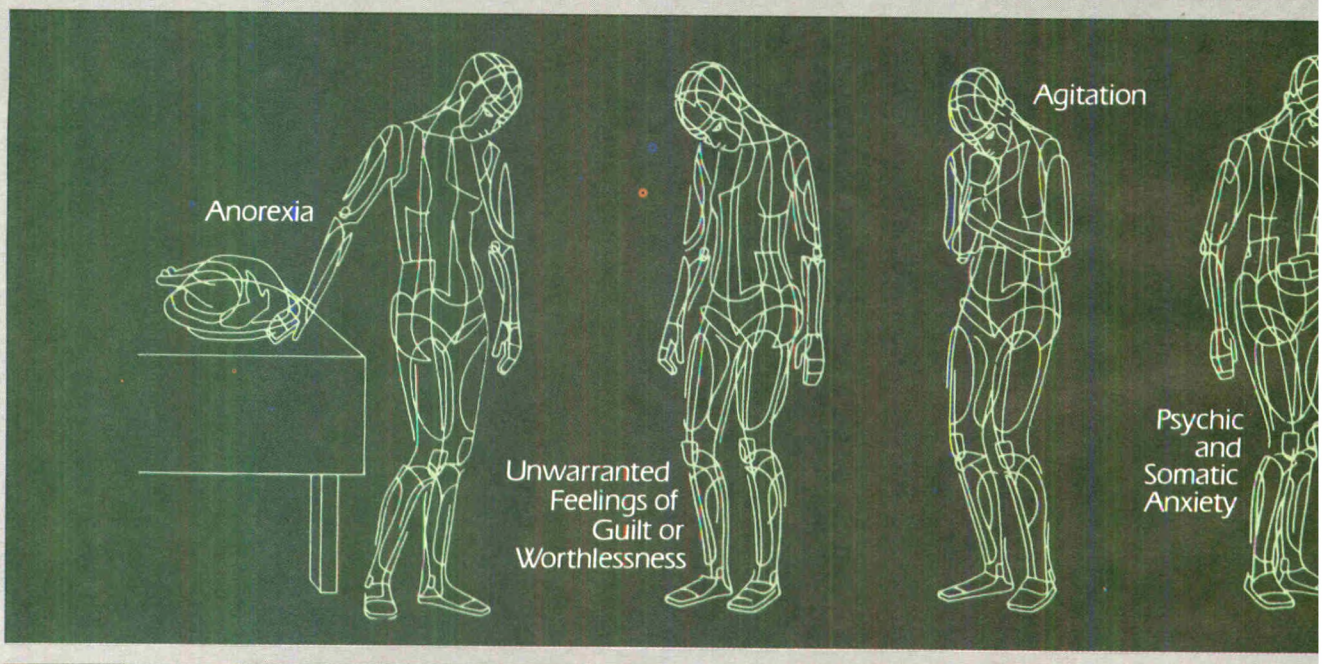
For rapid relief of symptoms in moderate to severe depression and anxiety

In a rigidly controlled nationwide multicenter study, when compared to amitriptyline alone, Limbitrol achieved these superior results...

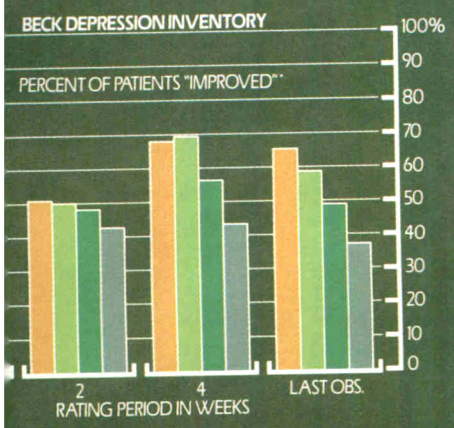
Please see summary of complete product information on last page of this advertisement.

ROCHE


HAMILTON DEPRESSION SCALE
PERCENT OF PATIENTS "IMPROVED"*
100%
90
80
70
60
50
40
30
20
10
0
1
2
4
LAST OBS.
RATING PERIOD IN WEEKS
LIMBITROL
AMITRIPTYLINE
WEEK ONE
*Criterion for "improvement" = score decreased by 1/2 or more from baseline.
LIMBITROL
AMITRIPTYLINE
CHLORDIAZEPOXIDE
PLACEBO
LIMBITROL
AMITRIPTYLINE
WEEK ONE


Anorexia
Unwarranted Feelings of Guilt or Worthlessness
Agitation
Psychic and Somatic Anxiety





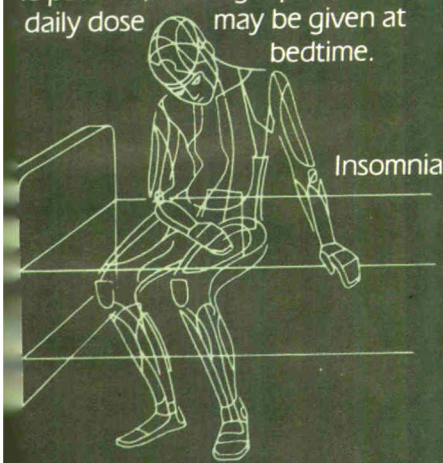
rion for "improvement" =
decreased by 1/3 or
from baseline.

Greater patient improvement

Within the first week—

twice as many patients achieved significant relief—sooner than with amitriptyline alone

omnia, a major target symptom, can relieved sooner by taking advantage he sedative effect of Limbitrol. For ne patients, the larger part or all of daily dose may be given at bedtime.



Better relief of symptoms

Within the first week—

symptom severity was reduced to a greater extent than with amitriptyline alone

As measured by the Hamilton Depression Scale

- insomnia
- psychic anxiety
- somatic anxiety
- agitation
- suicidal ideation
- worthlessness

As measured by the Beck Depression Inventory

- pessimism
- dissatisfaction
- guilt
- social withdrawal
- anorexia

imbitrol®

lets 10-25 each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt)
lets 5-12.5 each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)

r rapid relief of
oderate to severe
pression and anxiety

Greater patient acceptance

- Despite a higher incidence of drowsiness, there was a lower dropout rate due to side effects with Limbitrol than with amitriptyline

(Limbitrol 4%, amitriptyline 13%, chlordiazepoxide 1%)

- The dropout rate due to therapeutic failure was lower with Limbitrol than with amitriptyline

(Limbitrol 2%, amitriptyline 6%, chlordiazepoxide 12%)

Please see summary of complete product information on last page of this advertisement.

ROCHE

How to Initiate and Maintain Therapy

Select dosage strength appropriate for each patient

- Limbitrol 10-25 is recommended for most patients
- Limbitrol 5-12.5 may be indicated for those who do not tolerate higher doses and for elderly patients

Specify daily dosage based on symptom severity

- An initial dosage of three tablets is recommended
- Dosage may be increased to six tablets or decreased to two tablets daily as necessary
- Once a satisfactory response is obtained, evaluate response, and if necessary, reduce dosage to smallest amount needed to maintain remission

Utilize dosage options to best accommodate individual patient needs

- T.I.D. or Q.I.D., familiar regimens best suited for patients who tolerate medication without undue drowsiness
- Two tablets one hour before bedtime and one tablet midday may minimize daytime drowsiness and help relieve a common target symptom—insomnia
- Entire dosage h.s. to take maximum advantage of the sedative effect

How to make each patient an informed patient

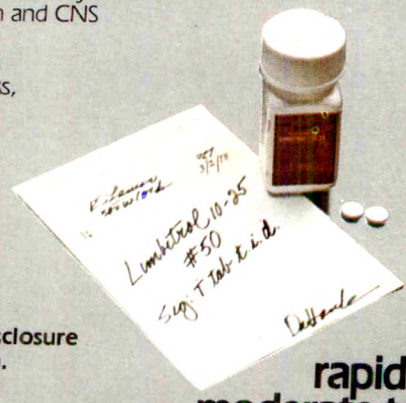
1. Discuss with patients the probability that they will experience drowsiness, especially during the first week.
2. Reassure your patients that drowsiness is one indication that the medication is working and that it may help alleviate their insomnia.
3. Encourage patients to report if drowsiness becomes troublesome so that, if necessary, dosage schedule can be adjusted or the symptom treated.
4. Caution patients about the combined effects with alcohol or other CNS depressants. Let them know that the additive effects may produce a harmful level of sedation and CNS depression.
5. Caution patients about activities requiring complete mental alertness, such as operating machinery or driving a car.
6. Warn pregnant patients and patients of childbearing age that the safety of Limbitrol in pregnancy has not yet been established.

Please see complete product disclosure for other pertinent information.

Limbitrol should not be used under following circumstances

1. Hypersensitivity to benzodiazepines tricyclic antidepressants.
2. Should not be given with an MAO in hibitor. To replace an MAO inhibitor wi Limbitrol, discontinue MAO inhibitor fo minimum of 14 days before cautiously i ing Limbitrol therapy.
3. During the acute recovery phase foll ing myocardial infarction.

Limbitrc



rapid relief
moderate to sev
depression and anxie

LIMBITROL® TABLETS
Tranquilizer–Antidepressant

Before prescribing, please consult complete product information, a summary of which follows:
Indications: Relief of moderate to severe depression associated with moderate to severe anxiety.
Contraindications: Known hypersensitivity to benzodiazepines or tricyclic antidepressants. Do not use with monoamine oxidase (MAO) inhibitors or within 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use; then initiate cautiously, gradually increasing dosage until optimal response is achieved. Contraindicated during acute recovery phase following myocardial infarction.
Warnings: Use with great care in patients with history of urinary retention or angle-closure glaucoma. Severe constipation may occur in patients taking tricyclic antidepressants and anticholinergic-type drugs. Closely supervise cardiovascular patients. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, especially high doses. Myocardial infarction and stroke reported with use of this class of drugs.)

Usage in Pregnancy: **Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Since physical and psychological dependence to chlordiazepoxide have been reported rarely, use caution in administering Limbitrol to addiction-prone individuals or those who might increase dosage. withdrawal symptoms following discontinuation of either component alone have been reported (nausea, headache and malaise for amitriptyline; symptoms [including convulsions] similar to those of barbiturate withdrawal for chlordiazepoxide).
Precautions: Use with caution in patients with a history of seizures, in hyperthyroid patients or those on thyroid medication, and in patients with impaired renal or hepatic function. Because of the possibility of suicide in depressed patients, do not permit easy access to large quantities in these patients. Caution patients about possible combined effects with alcohol and other CNS depressants and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Periodic liver function tests and blood counts are recommended during prolonged treatment. Amitriptyline component may block action of guanethidine or similar antihypertensives. Concomitant use with other psychotropic drugs has not been evaluated: sedative effects may be additive. Discontinue several days before surgery. Limit concomitant administration of ECT to essential treatment. See Warnings for precautions about pregnancy. Limbitrol should not be taken during the nursing period. Not recommended in children under 12.
In the elderly and debilitated, limit to smallest effective dosage to preclude ataxia, oversedation, confusion or anticholinergic effects.
Adverse Reactions: Most frequently reported are those associated with either component alone: drowsiness, dry mouth, constipation, blurred vision, dizziness and bloating. Less frequently occurring reactions include vivid dreams, impotence, tremor, confusion and nasal congestion. Many depressive symptoms including anorexia, fatigue, weakness, restlessness and lethargy have been reported as side effects of both Limbitrol and amitriptyline. Granulocytopenia, jaundice and hepatic dysfunction have been observed rarely.
This list includes adverse reactions not reported with Limbitrol but requiring consideration because they have been reported with one or both components or closely related drugs:
Cardiovascular: Hypotension, hypertension, tachycardia, palpitations, myocardial infarction, arrhythmias, heart block, stroke.
Psychiatric: Euphoria, apprehension, poor concentration, delusions, hallucinations, hypomania and increased or decreased libido.
Neurologic: Incoordination, ataxia, numbness, tingling and paresthesias of the extremities, extrapyramidal symptoms, syncope, changes in EEG patterns.
Anticholinergics: Disturbance of accommodation, paralytic ileus, urinary retention, dilatation of urir tract.
Allergic: Skin rash, urticaria, photosensitization, edema of face and tongue, pruritus.
Hematologic: Bone marrow depression includin agranulocytosis, eosinophilia, purpura, thrombocytopenia.
Gastrointestinal: Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, black tongue.
Endocrine: Testicular swelling and gynecomasti the male, breast enlargement, galactorrhea and minor menstrual irregularities in the female and elevation and lowering of blood sugar levels.
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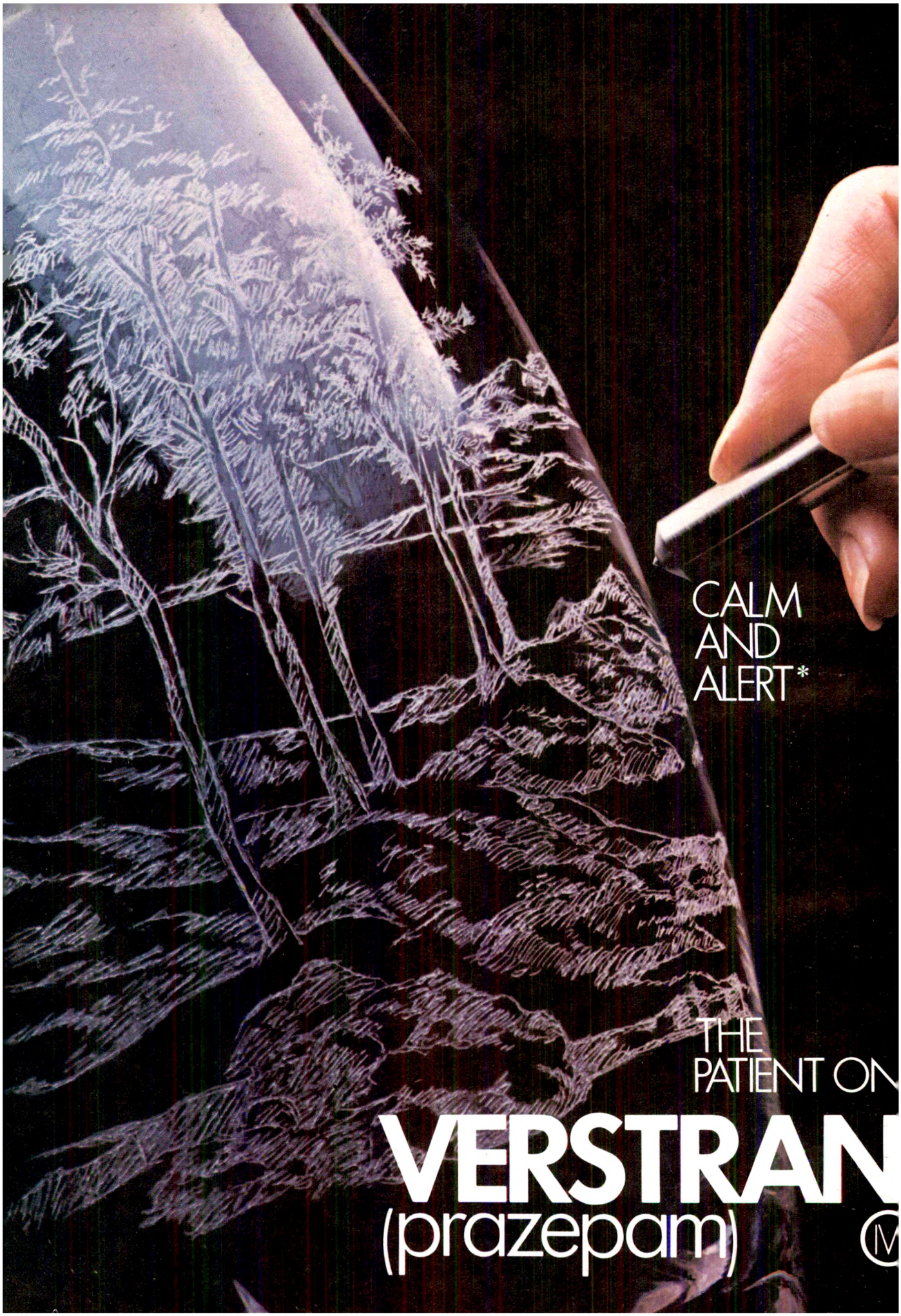
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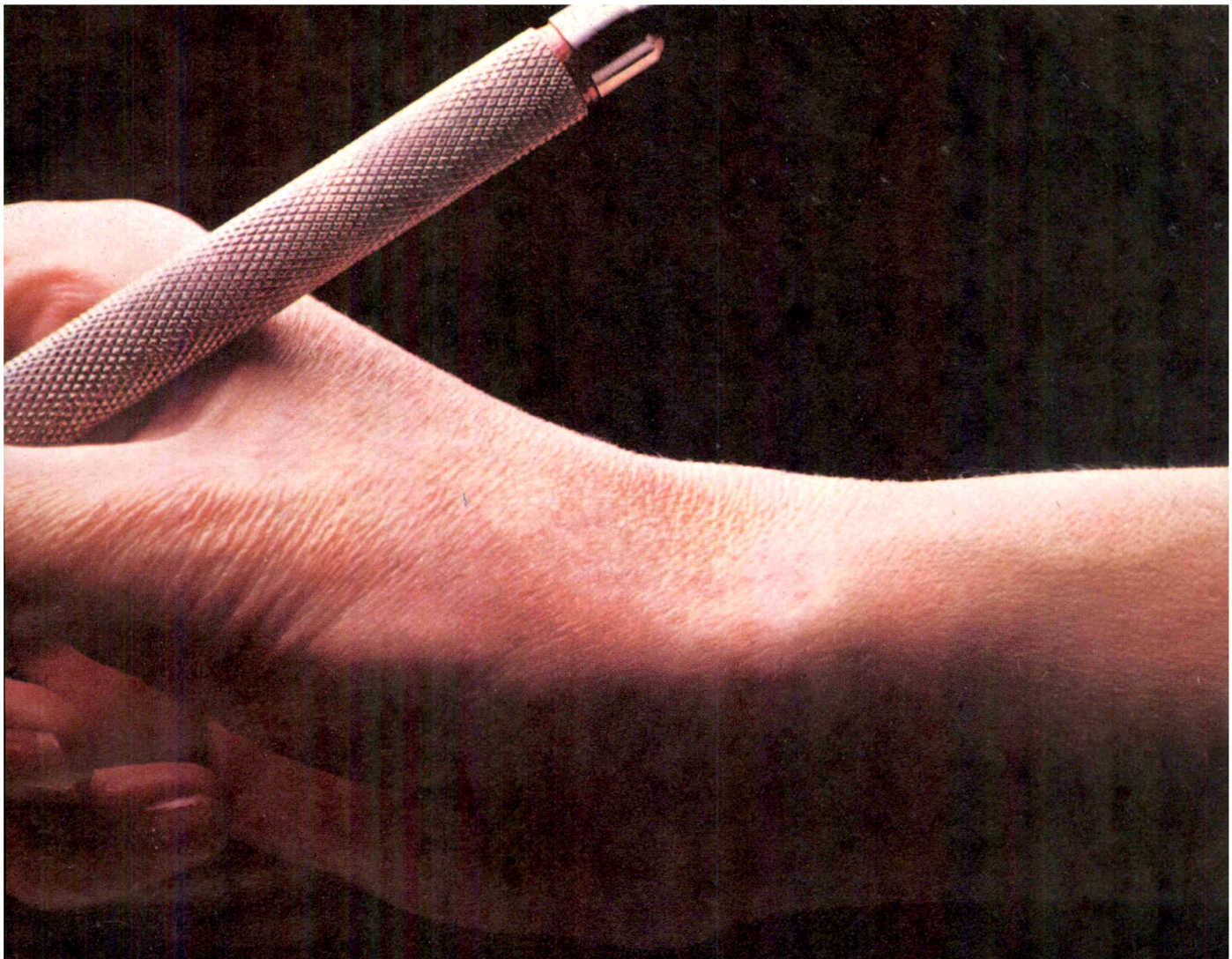
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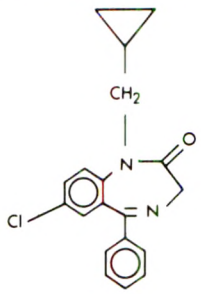
(prazepam)

Ⓝ

VERSTRAN® (prazepam) Ⓝ

Caution: Federal law prohibits dispensing without prescription.

Description: Verstran (prazepam), a benzodiazepine derivative, is identified chemically as 7-chloro-1-(cyclopropylmethyl)-1,3-dihydro-5-phenyl-2H-1,4-benzodiazepin-2-one. The molecular weight is 324.8 and the structural formula is as follows:



Clinical Pharmacology: Studies in normal subjects have shown that Verstran (prazepam) has depressant effects on the central nervous system. Oral administration of single doses as high as 60 mg and divided doses up to 100 mg t.i.d. (300 mg total daily dose) were without toxic effects.

Single oral doses of Verstran in normal subjects produced peak blood levels at 6 hours postadministration, with significant amounts still present after 48 hours. Verstran was slowly absorbed over a prolonged period, rather constant blood levels were maintained, and excretion was prolonged. The mean half-life of prazepam measured in subjects given 10 mg t.i.d. for one week was 63 (±15 s.d.) hours before and 70 (±10 s.d.) hours after multiple dosing — a nonsignificant difference. Human metabolism studies showed that prior to elimination from the body, prazepam is metabolized in large part to 3-hydroxyprazepam and oxazepam.

Indications: Verstran (prazepam) is indicated for the symptomatic relief of anxiety associated with anxiety neurosis, in other psychoneuroses in which anxiety symptoms are prominent features, and as an adjunct in disease states in which anxiety is manifested.

The effectiveness of Verstran (prazepam) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should reassess periodically the usefulness of the drug for the individual patient.

Contraindications: Verstran (prazepam) is contraindicated in patients with a known sensitivity to the drug and in those with acute narrow angle glaucoma.

Warnings: Verstran (prazepam) is not recommended in psychotic states and in those psychiatric disorders in which anxiety is not a prominent feature.

Patients taking Verstran should be cautioned against engaging in hazardous occupations requiring mental alertness, such as operating dangerous machinery, including motor vehicles.

Because Verstran has a central nervous system depressant effect, patients should be advised against the simultaneous use of other CNS-depressant drugs, including phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants. The effects of alcohol may also be increased.

Physical and Psychological Dependence: Withdrawal symptoms similar in character to those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepine drugs. These symptoms include convulsions, tremor, abdominal and muscle cramps, vomiting and sweating. Addiction-prone individuals, such as drug addicts and alcoholics, should be under careful surveillance when receiving benzodiazepines because of the predisposition of such patients to habituation and dependence.

Precautions:

Use in Pregnancy and Lactation:

An increased risk of congenital malformations associated with the use of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) during the first trimester of pregnancy has been suggested in several studies. Verstran (prazepam) a benzodiazepine derivative, has not been studied adequately to determine whether it, too, may be associated with an increased risk of fetal abnormality. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physician about the desirability of discontinuing the drug. In view of their molecular size, Verstran and its metabolites are probably excreted in human milk. Therefore, this drug should not be given to nursing mothers.

In those patients in whom a degree of depression accompanies the anxiety, suicidal tendencies may be present, and protective measures may be required. The least amount of drug that is feasible should be available to the patient at any one time.

Patients taking Verstran for prolonged periods should have blood counts and liver function tests periodically. The usual precautions in treating patients with impaired renal or hepatic function should also be observed. Hepatomegaly and cholestasis were observed in chronic toxicity studies in rats and dogs.

In elderly or debilitated patients, the initial dose should be small, and increments should be made gradually, in accordance with the response of the patient, to preclude ataxia or excessive sedation.

Pediatric Use: Safety and effectiveness in patients below the age of 18 have not been established.

Adverse Reactions: The side effects most frequently reported during double-blind placebo-controlled trials employing a typical 30 mg divided total daily dose and the percent incidence in the Verstran (prazepam) group were: fatigue (11.6%), dizziness (8.7%), weakness (7.7%), drowsiness (6.8%), lightheadedness (6.8%), and ataxia (5.0%). Less frequently reported were: headache, confusion, tremor, vivid dreams, slurred speech, palpitation, stimulation, dry mouth, diaphoresis, and various gastrointestinal complaints. Other side effects included: pruritus, tr
sient skin rashes, swelling of feet, joint pains, vari
genitourinary complaints, blurred vision, and s
cope. Single nightly dose, controlled trials of varia
dosages showed a dose-related incidence of th
same side effects. Transient and reversible aber
tions of liver function tests have been reported,
have slight decreases in blood pressure and increa
in body weight.

These findings are characteristic of benzodiazepi
drugs.

Overdosage: As in the management of overdosa
with any drug, it should be borne in mind that mu
ple agents may have been taken.

If vomiting has not occurred spontaneously,
should be induced. Immediate gastric lavage is a
recommended. General supportive care, includi
frequent monitoring of the vital signs and close obs
vation of the patient, is indicated. Hypotensi
though unlikely, may be controlled with Levophe
(levarterenol bitartrate) or Aramine® (metarami
bitartrate). Caffeine and Sodium Benzoate Injecti
USP, may be used to counteract central nervous syst
depressant effects.

Dosage and Administration: Verstran (prazepam)
administered orally in divided doses. The usual da
dose is 30 mg. The dose should be adjusted gradua
within the range of 20 to 60 mg daily in accordar
with the response of the patient. In elderly or deb
tated patients it is advisable to initiate treatment a
divided daily dose of 10 mg to 15 mg. (See Prec
tions.)

Prazepam may also be administered as a sin
dose daily at bedtime. The recommended starti
nightly dose is 20 mg. The response of the patient
several days' treatment will permit the physician
adjust the dose upward or, occasionally, downwarc
maximize antianxiety effect with a minimum of dc
time drowsiness. The optimum dosage will usua
range from 20 to 40 mg.

Drug Interactions: If Verstran (prazepam) is to
combined with other drugs acting on the central n
vous system, careful consideration should be given
the pharmacology of the agents to be employed. T
actions of the benzodiazepines may be potentiated
barbiturates, narcotics, phenothiazines, monoami
oxidase inhibitors or other antidepressants.

If Verstran is used to treat anxiety associated w
somatic disease states, careful attention must be pc
to possible drug interaction with concomitant medi
tion.

How Supplied: Verstran (prazepam) 10 mg light blu
scored tablets in bottles of 100 (N 0047-0276-51) a
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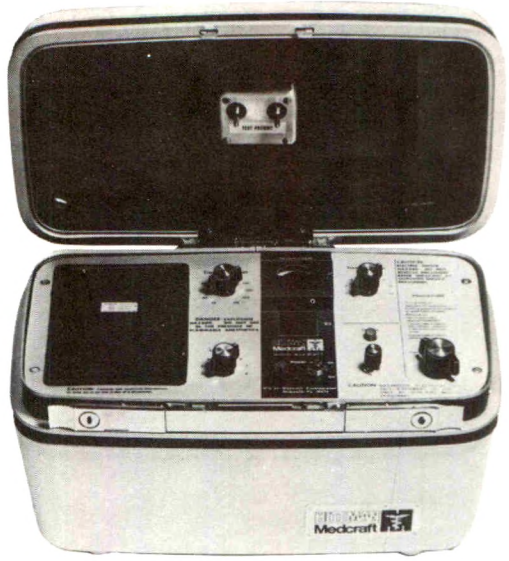
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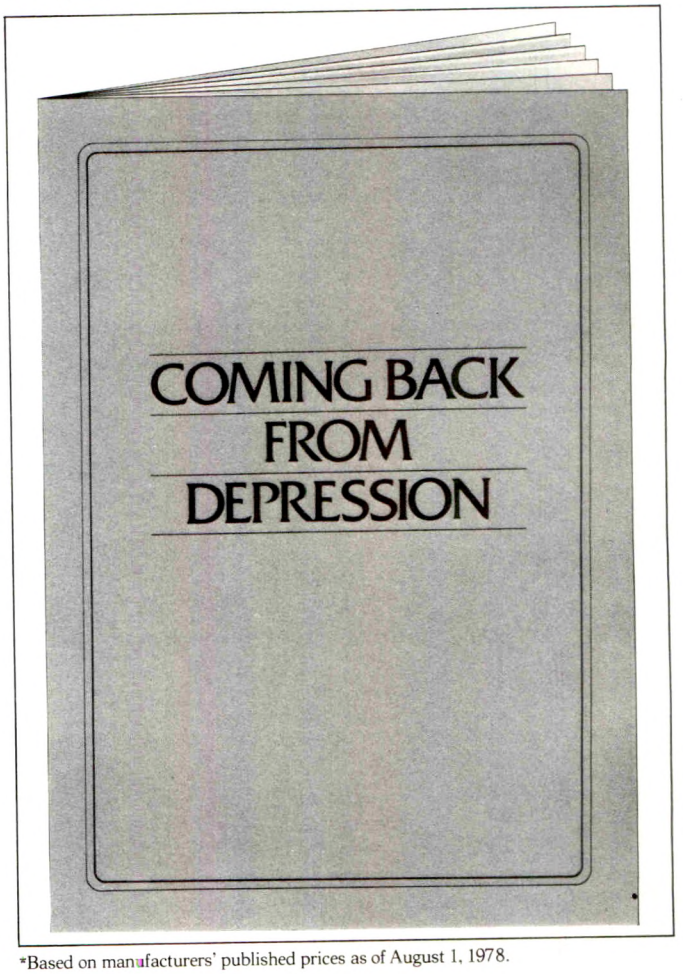
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Please see following page for brief summary.

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Amitid™ (Amitriptyline Hydrochloride Tablets USP)

DESCRIPTION: Amitriptyline hydrochloride is a dibenzocycloheptadiene derivative available for oral administration as FILMLOK® tablets (FILMLOK is a Squibb trademark for veneer-coated tablets).

INDICATIONS: For relief of symptoms of depression alone or accompanied by anxiety. Endogenous depression is more likely to be alleviated than are other depressive states.

CONTRAINDICATIONS: Contraindicated in patients with a history of hypersensitivity to amitriptyline. Do not administer concomitantly with a monamine oxidase (MAO) inhibitor since hyperpyretic crises, severe convulsions, and deaths occurred when tricyclic antidepressants and MAO inhibitors were administered simultaneously. When an MAO inhibitor is to be replaced by amitriptyline, allow 14 days to elapse after discontinuation of the former and then initiate amitriptyline cautiously and gradually increase dosage until optimum response is achieved. Amitriptyline is not recommended for use during the acute recovery phase following myocardial infarction.

WARNINGS: Amitriptyline may block antihypertensive action of guanethidine or similarly acting drugs. Use with caution in patients with history of seizures and (because of atropine-like action of amitriptyline) in patients with history of narrow-angle glaucoma (even average doses may precipitate an attack), increased intraocular pressure, or urinary retention. Closely watch patients with cardiovascular disorders since, in addition to having caused myocardial infarction and stroke, tricyclic antidepressants (including amitriptyline) particularly with high dosage have been reported to produce arrhythmias, sinus tachycardia, and prolongation of conduction time. Close supervision is required when amitriptyline is given to hyperthyroid patients or those on thyroid medication. Amitriptyline may impair mental and/or physical abilities required for performance of hazardous tasks such as operating machinery or driving a motor vehicle. Bear in mind that in patients who may use alcohol excessively the potentiation may increase the danger inherent in any suicide attempt or overdosage.

Usage in Pregnancy: Safe use during pregnancy and lactation has not been established; therefore, in administering the drug to pregnant patients, nursing mothers, or women who may become pregnant, weigh the possible benefits against the possible hazards to the mother and child. Animal reproduction studies have been inconclusive, and clinical experience has been limited.

Usage in Children: At the present time, not recommended for patients under 12 years of age because of lack of experience with use in children.

PRECAUTIONS: Schizophrenic patients may develop increased symptoms of psychosis; patients with paranoid symptomatology may have an exaggeration of such symptoms; manic depressive patients may experience a shift to the manic phase. In these circumstances the dose of amitriptyline may be reduced or a major tranquilizer may be administered concurrently.

Closely supervise and carefully adjust dosage in concomitant use with anticholinergic or sympathomimetic drugs, including combination of epinephrine and local anesthetics. Paralytic ileus may occur with concomitant use of tricyclic antidepressants and anticholinergic-type drugs. Caution is advised if used concurrently with large doses of ethchlorvynol since transient delirium has been reported when one gram of that drug and 75 to 150 mg of amitriptyline HCl were administered. Amitriptyline may enhance response to alcohol and the effects of barbiturates and other CNS depressants. The possibility of suicide in depressed patients remains during treatment and until significant remission occurs. Potentially suicidal patients should not have easy access to large quantities of the drug. Prescriptions should be written for the smallest amount feasible. Limit concurrent administration of this drug and electroshock therapy to patients for whom it is essential since the hazards associated with such therapy may be increased. Discontinue this drug, when possible, several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported. Use amitriptyline with caution in presence of impaired liver function.

ADVERSE REACTIONS: NOTE: A few of the adverse reactions listed below have not been reported with this specific drug, but each of the reactions should be considered when administering amitriptyline because of pharmacological similarities among tricyclic antidepressants.

Cardiovascular: Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, and stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness; tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; and syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, increased intraocular pressure, constipation, paralytic ileus, urinary retention, and dilatation of the urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, and edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, leukopenia, eosinophilia, purpura, and thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, and black tongue. Rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, and elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, and alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache, and malaise. These are not indicative of addiction.

For full prescribing information, consult package insert.

HOW SUPPLIED: Available for oral administration in tablets providing 10, 25, 50, 75, and 100 mg amitriptyline hydrochloride. The 10, 25, and 50 mg tablets are available in bottles of 100 and 1000. The 75 and 100 mg tablets are available in bottles of 100.

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David M. Kaufman, M.D.

Department of Neurology
Montefiore Hospital and Medical Center

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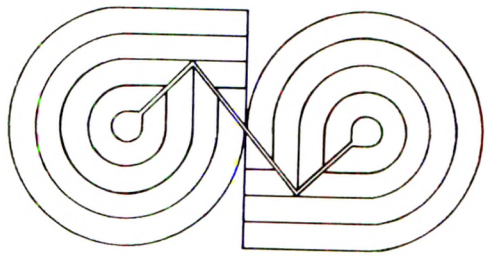
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nfrequent akathisia ınd other disabling :xtrapyramidal effects...

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ased on antipsychotic dosage ranges

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hort-Term Dynamic Psychotherapy

Y JUDD MARMOR, M.D.

hort-term dynamic psychotherapy is rooted in ychoanalytic theory. The author traces the storical background of brief psychotherapy, cusing on the contributions of Freud, Ferenczi, ank, and Alexander and French, and then presents a nthesis of contemporary views. Selection criteria for itients who can benefit from short-term therapy, and e techniques used, are discussed. Similarities to and fferences from crisis intervention techniques are inted out. The trend of the future can be expected to toward shorter-term therapies, but given the esent state of psychiatric knowledge, long-term erapy will still be necessary for many patients.

E ARE LIVING in the midst of a major psychoerapeutic revolution in this second half of the twentih century. New therapies of all kinds have emerged astounding numbers, some scientifically based, oths reverting to the magical, mystical, and religious ots that were the precursors of scientific psychotherpy. At the same time the development of third-party ayers, together with the imminence of some form of ational health insurance, places psychiatrists under essure to find shorter, more broadly applicable, and ore efficient techniques of therapy or risk exclusion om these programs for fiscal reasons. As a conquence, we can anticipate that the briefer techniques group therapy, behavioral therapy, and family therpy will emerge more strongly in the years ahead. The ct is, however, that the vast majority of American sychiatrists are still heavily committed to a one-to-ne model of dynamic psychotherapy (1). Therefore, e greatest need to develop more time-effective approaches is in this area.

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HISTORICAL BACKGROUND

Brief dynamic psychotherapy is rooted in the psychoanalytic tradition. Its fundamental insights and its basic theoretical principles would not have been possible were it not for the ground-breaking discoveries of Sigmund Freud. Actually, some of the earliest psychoanalytic treatments conducted by Freud tended to be of quite short duration. Bruno Walter, the conductor, described in his autobiography (2) a successful six-session therapeutic experience he had with Freud in 1906. Jones (3, pp. 79–80) related that in 1908 Freud was able to get at the psychodynamic roots of the composer Gustav Mahler's potency problem with his wife and relieve it in a single four-hour session. Even Freud's early didactic analyses were almost all of relatively short duration, ranging from several months to about one year (3, pp. 31–32, 161–162). As the goals of psychoanalysis became more ambitious, however, and its theoretical superstructure grew more complex, analytic treatments began to increase in length to such an extent that in his later years Freud pessimistically concluded that some of them were becoming interminable (4).

Many of the early analysts were aware of and troubled by this trend, but the first psychoanalytic pioneer to methodically explore modifications in psychoanalytic technique for the purpose of shortening the length of classical analysis was Sandor Ferenczi, who, around 1918, began to experiment with a technique that he called "active therapy." Ferenczi (5) claimed that in so doing he was merely following Freud's lead. He referred to a paper that Freud had presented at the Budapest International Congress in 1918 (6) in which Freud stated that in certain cases of phobia or obsessional neurosis it is sometimes necessary, in order to advance the therapy, to institute active measures to induce the patient to face the phobia or anxiety. Ferenczi also pointed out that activity as a technique was inherent in the psychoanalytic process because every interpretation that is given constitutes an interference with the patient's psychic transactions of that moment

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and thus facilitates the appearance of thoughts that otherwise might not have become conscious.

Ferenczi experimented with various types of activity in the psychoanalytic situation in an effort to overcome what he called a "stagnation of the analysis." Some of his techniques were restrictive or pressuring (e.g., forbidding masturbation or insisting that patients control certain body movements or other actions Ferenczi deemed to be in the service of "unconscious resistance"). Other interventions were loving or indulgent, based on the theory that the analyst as a parent surrogate was thus making amends for the rejections and traumata that the patient may have suffered from his own parents.

Ferenczi's "reparative" efforts included hugging, kissing, and nonerotic fondling of his patients. When Freud heard of these, he wrote Ferenczi the now famous letter (7) expressing his very dim view of these activities and predicting prophetically that they would eventually lead to greater excesses on the part of other therapists. He wrote,

> A number of independent thinkers in matters of technique will say to themselves: Why stop at a kiss? . . . Bolder ones will come along who will go further. . . . The younger of our colleagues will find it hard to stop at the point they originally intended, and God the Father Ferenczi, gazing at the lively scene he has created will perhaps say to himself: Maybe after all I should have halted in my technique of motherly affection *before* the kiss.

Ferenczi subsequently abandoned these particular techniques, but he never gave up his efforts to develop a more active approach to analytic therapy.

Ferenczi's efforts brought him into early collaboration with Otto Rank, who had been pursuing similar approaches independently. Ferenczi and Rank worked together for a number of years on evolving various modifications of technique, culminating in 1925 in their publication of the slender but seminal volume *The Development of Psychoanalysis* (8). In this volume they pointed out that early in the history of psychoanalysis "splendid cures . . . were effected, sometimes in a few days or weeks" (p. 52) but that as analytic theory and knowledge expanded analyses became longer and longer. They attributed this to a need for "discovering afresh in every single case the psychological and theoretical knowledge derived from analysis" (p. 59) and to turning every analysis into a proving ground for analytic theory. Ferenczi and Rank characterized this as "making the disastrous mistake of neglecting the actual [therapeutic] task for the sake of the psychological interest" (pp. 24–25).

They pointed out that the technique of psychoanalytic treatment, although dependent on psychoanalytic theory, need not and indeed should not remain tied to the "investigative" methods out of which the theory evolved. In this context they criticized an undue preoccupation in therapy with the historical past and emphasized the paramount importance of focusing on the present analytic situation and its transference implications. Anticipating Franz Alexander's concept of the "corrective emotional experience," Ferenczi and Rank asserted that the goal of psychoanalysis "is to substitute by means of the technique, effective factors of experience for intellectual processes" (p. 62). They expressed the conviction that greater understanding of these practical principles of technique would ultimately make it possible to "shorten and simplify psychoanalytic treatment."

In some ways Otto Rank may well be the most important historical forerunner of the brief dynamic psychotherapy movement. His concept of the trauma of birth (9), although biologically and psychodynamically dubious, laid the groundwork for the subsequent recognition of the predominant importance in personality development of the pre-oedipal years, particularly the early mother-child relationship. When Rank published his views, however, the primary emphasis in psychoanalytic theory was on the oedipal period and castration anxieties rather than on the pre-oedipal period and the more basic separation anxieties. Rank's views were vigorously attacked by Freud's psychoanalytic contemporaries because they were correctly perceived as threatening the fulcrum of Freud's theories at that time.

It is unfortunate that the issue of disloyalty to Freud has cast a heavy shadow over the value of Rank's achievements. Looking back from the vantage point of our present knowledge of the importance of pre-oedipal relationships in personality development as well as the nuclear importance of separation and individuation in emotional maturation, we can now perceive that Rank was the prime theoretical precursor of these developments, without in any way denigrating the later creative contributions of people like Rene Spitz (10), Margaret Mahler (11), or John Bowlby (12).

As his ideas evolved, Rank himself began to play down the importance of the birth trauma itself and to recognize that the issue of separation and individuation was really the core problem. He made its working through the central focus of his psychotherapeutic method. It is not surprising, therefore, that Rank was the first analyst to attribute major importance to the setting of a time limit for the analytic process to promote an earlier therapeutic focus on the problem of separation. However, Rank was not the first analyst to experiment with setting a termination date in advance. Freud did this in 1912 in his analysis of the "Rat Man," which he published in 1918 as "From the History of an Infantile Neurosis" (13). Although Freud recognized the usefulness of this technical device, he never made it a cardinal point in his analytic technique. Rank, on the other hand, considered the setting of a time limit central to the therapeutic work and the issue of separation and individuation critical to the process of adaptation and maturation in all of human existence. Thus at a very early point in Rank's therapy the analytic work became centered on the anxieties and conflicts surrounding separation and terminatic

id the entire therapeutic process was foreshortened. recent years Mann has made this technique the fo- l point of his approach to time-limited psychothera- y, but, surprisingly, he made only a brief and pas- ng reference to the work of Rank in his book (14).

It is also interesting, in the light of current develop- nts in brief psychotherapy, to take a second look at nk's concept of "will therapy" (15). Rank empha- ed the importance of mobilizing the patient's "will" the course of therapy and claimed that by so doing e therapeutic process could be facilitated. If we were substitute for the word "will" the more modern m "motivation," we would find that Rank was ying something that has been emphasized by all dern theorists about short-term dynamic psycho- rapy, namely, the overwhelming importance in- hieving a favorable therapeutic outcome of a strong tivation to change on the part of the patient. In this nnection it is noteworthy that four of the seven cri- ia for motivation to change used by Sifneos (16) in lecting patients for brief psychotherapy employ the ncept of willingness (i.e., willingness to actively par- ipate in the treatment situation, willingness to un- rstand oneself, willingness to change, and willing- ss to make reasonable sacrifices in terms of time and s). What is willingness in this context but the ability mobilize one's "will" toward a particular objective?

When, some 20 years after Ferenczi and Rank's ntributions, Alexander and French came out with eir important and seminal volume on psychoanalytic rapy (17), they were frank to admit that their work as "a continuation and realization of ideas first pro- sed by Ferenczi and Rank." The work of Alexander d French stands on its own merits, however. Their lume was the culmination of seven years of research d investigation into the development of shorter ap- oaches to psychotherapy that had been carried out the Chicago Institute of Psychoanalysis.

Alexander, the prime mover in this research, had for ars been puzzled by what he called the "baffling dis- epancy" between the length and intensity of psycho- alytic treatment and the degree of therapeutic suc- ss. He began his investigations by questioning the lidity of the following traditional psychoanalytic gmas: 1) that the depth of therapy is necessarily pro- rtionate to the length of treatment and frequency of terviews, 2) that therapeutic results achieved by a latively small number of interviews are necessarily perficial and temporary but results of more pro- ged therapy are necessarily more stable and pro- und, and 3) that prolongation of an analysis is justi- d on the grounds that the desired therapeutic results e more likely to be achieved than through briefer chniques. None of these traditional assumptions oved to be justified.

In the course of their research, Alexander and his lleagues experimented with the frequency of inter- ews, the optional use of the chair or couch, long or ort interruptions of therapy preparatory to termi- tion, and the combination of psychotherapy with drug or other therapy. They also sought to learn how to control and manipulate the transference relationship to fit the particular psychodynamics of each case. The first and most important principle that evolved from these studies, that of flexibility, seems obvious today, but it was revolutionary in an era dominated by the conviction that the standard psychoanalytic method was the optimum method of therapy for most neurotic patients. Alexander insisted that in psychotherapy as in all medical therapy the physician should adapt his technique to the needs of the patient. "Only the nature of the individual case," he said, "can determine what technique is best suited to bring about the curative processes" (17, p. 26).

Alexander also pointed out that in many instances daily interviews prove to be antitherapeutic because they gratify the patient's dependent needs more than is desirable. He questioned the therapeutic value of promoting regression and argued that it should be "a general principle in all psychotherapy to attempt to check this regressive tendency from the very beginning of the treatment" (17, p.30). This principle was subsequently strongly advocated by Sandor Rado (18).

As one way of heightening the emotional intensity of interviews with patients Alexander recommended consciously manipulating the frequency of visits. He felt this was an effective technique for controlling the transference relationship, limiting regression, preventing the development of overdependency on the therapist, and fostering autonomy. He also advocated interruptions of treatment as another way of increasing the emotional intensity and efficiency of the therapeutic process. Interruptions, by virtue of their ability to test the patient's capacity for self-reliance and for more effectively coping with his life situation, could also be used as preparatory indicators enabling both therapist and patient to arrive at a mutually agreed on termination point.

Like Ferenczi and Rank, Alexander placed great emphasis on the emotional experiences of the patient in relation to the therapist, specifically on what he called the "corrective emotional experience," in which the patient is reexposed under the more favorable circumstances of the transference relationship to emotional situations that he could not handle in the past.

Alexander asserted that in order to know what new emotional experiences are necessary to achieve therapeutic results, the therapist must understand not only the patient's current psychodynamics but also the genetic development of his difficulties. In this respect Alexander's technique differs decisively from that of Rank, who tended to minimize the importance of such genetic understanding. The more precise this understanding, according to Alexander, the more adequately the therapist can provide the proper corrective emotional experience. Therefore, he felt that the therapist's reactions had to be planned on the basis of these dynamic insights.

Although Alexander's views were violently attacked

by most of the classical psychoanalysts of his time, by now the principles he elucidated have become part of the daily working equipment of almost every psychiatrist and psychoanalyst. It is fair to say that Alexander more than any other modern psychoanalyst is responsible for leading the way toward the application of psychoanalytic principles to more active and shorter dynamic psychotherapy. As a consequence of his germinal work, an increasing number of psychoanalysts began to practice more flexibly during the years following World War II. Psychoanalysts began to see their patients less frequently than the traditionally mandated four or five times weekly, often working with them face to face and, in general, entering into more active communicative transactions with them than the more passive classical model required.

In more recent years, as the demand for psychotherapy has widened to encompass greater and greater segments of the population, the pressures to clarify our therapies and sharpen our skills in the application of psychodynamic theory to briefer forms of psychotherapy have continued to increase. Beginning with the 1960s, a number of psychoanalysts both in the United States and abroad have responded to this challenge by picking up where Alexander and French left off. Malan (19, 20), Wolberg (21), Bellak and Small (22), Sifneos (16), Balint and associates (23), Mann (14), and Davanloo (24) are but a few of the individuals who have contributed significantly to the theory and technique of short-term psychotherapy. In the pages that follow I shall attempt to present a synthesis of their views that will reflect the present state of the art in short-term dynamic psychotherapy.

SELECTION CRITERIA

Short-term dynamic psychotherapy rests on two basic substructures, selection and technique. Each of these is of equal importance. Not all patients are equally suitable for short-term dynamic psychotherapy, but a substantial proportion of those who are ordinarily considered suitable for longer-term analytic procedures are equally suitable for the shorter-term approach.

At the very outset of the selection process it is important to underline the necessity for a good history and a psychodynamic diagnosis. By a psychodynamic diagnosis I do not mean placing the patient into a formal nosologic category but, rather, carefully evaluating both the inner and outer forces that contribute to the patient's psychopathology. Inasmuch as short-term dynamic psychotherapy involves an uncovering and searching out of relevant unconscious intrapsychic factors, the therapist looks for qualities in the patient that will indicate his capacity to work effectively in this way. The following are the most important of these qualities: 1) evidences of ego strength (e.g., intelligence, level of educational achievement, sexual adjustment, type of work, ability to assume responsibility), 2) at least one meaningful interperso relationship in the past, indicating a capacity for ba trust, which is essential in the psychotherapeu process, 3) the ability to interact with the therapist the first session (i.e., the capacity to form a positi transference), 4) the ability to think in psychologi terms (i.e., the ability to accept interpretation or, a is sometimes called, the capacity for insight, which usually tested in the initial interview by making a t tative interpretation and evaluating the patient's sponse to it), and 5) the ability to experience feelir (i.e., the degree to which the individual seems to be touch with his own emotions).

The above five qualities are essential factors in selection of patients for any form of dynamic psycl therapy, long-term as well as short-term. Two ad tional selection factors are particularly relevant to a essential for a short-term psychotherapeutic approac The first of these is the existence of a focal conflict. T therapist should be able in the first interview or two, most, to identify a central conflictual problem arou which most of the patient's difficulties revolve. S neos (16) has stated that this focal conflict must oedipal, but this view is not shared by others. For ample, the focal conflict can also be a dependence-dependence conflict, a sibling rivalry situation, o difficulty in coping with object loss.

The other selection factor of prime relevance short-term therapy is the existence of a clear-cut a strong motivation to change. Malan (19, 20) prope emphasized that the issue is not simply one of the tient's wish to get rid of his symptoms but of an ceptance of the fact that in the process some ba change in adaptational patterns may be necessary.

As already noted, with the exception of the last tw all of these selection criteria are not very differ from the criteria that are usually applied in decid whether or not a patient is suitable for classical p choanalytic therapy. However, these selection crite are not necessarily indicative of the depth of exist psychopathology. Most contemporary workers in field agree that short-term dynamic psychotherapy c be used with patients who might be described as re tively "sick" as well as for those with relatively mi problems. Patients with a wide variety of personal disorders and psychoneuroses as well as those w transitional crises may be suitable for short-term namic psychotherapy provided they fulfill the sel tion criteria. The critical issue is not diagnosis so mu as the possession of certain personality attributes p the existence of a focal conflict and a high degree motivation.

TECHNIQUE

In considering the technique of short-term dynan psychotherapy it is first necessary to emphasize t there are certain common denominators in all psycl dynamic therapies. I have described these in de

sewhere (25–27) and shall merely summarize them re: 1) a release of tension (catharsis) in a setting of pe and expectation of help, 2) a constructive pa-nt-therapist relationship (therapeutic alliance) based i both unconscious factors (transference factors) as ell as the real qualities that both patient and therapist ing to their transactions, 3) cognitive learning based i interpretations (insight), 4) operant conditioning sed on overt and/or covert indications of approval or sapproval from the therapist that move the patient in e direction of "mental health" (an additional facet of is operant conditioning is the existence of corrective notional experiences in which the therapist reacts fferently and more constructively to the patient than d significant authority figures in the patient's past), identification with the therapist in which the patient sually unconsciously) models himself after the thera-st, incorporating some of the latter's value systems d/or behavioral patterns, 6) elements of suggestion d persuasion, either covert or overt, which are pres-t in all psychotherapies, 7) some aspects of practice d rehearsal of new adaptive techniques and their neralization, sometimes called "working through" "reality testing," all in a setting of consistent emo-nal support from the empathic therapist.

Given these general factors common to all dynamic erapies, how does short-term dynamic psychothera-differ from other therapies? Four factors that are ecific to the short-term technique can be identified. rst, the patient is always seen sitting up, facing the erapist. Although this factor is essential to short-rm dynamic psychotherapy, it is, of course, present many longer-term dynamic therapies as well. Therefore, it is not as unique as the other three factors are. It important, however, for creating a structure in hich a more active transactional process can occur an would be possible with the patient on the couch.

Second, brief therapy always involves setting a time nit. This is a unique and important element in all ort-term psychotherapies. Setting a time limit is essential for the process, and its importance cannot be veremphasized. Mann (14) has discussed the meaning time in psychotherapy in eloquent philosophical rms. From a purely clinical standpoint, however, ree fundamental consequences can be identified as riving from this technical maneuver. First, the set-ng of a time limit places a central emphasis from the ry beginning of the therapy on the issues of separa-n and individuation. This colors the entire therapeu-process from beginning to end and creates an en-ely different set of expectations than in long-term erapy, where the patient is told at the outset that the ngth of therapy is open-ended, unpredictable, or may on for a year or two or more. Second, not only is e issue of separation and individuation relevant, if t central, to the problem presented by most patients, t putting it in the forefront of the therapeutic tech-que reflects a basic respect for and encouragement of e patient's capacity to be autonomous. This counters e patient's impulse to see himself as helpless, inadequate, and in need of dependent support. Third, the very process of termination constitutes a therapeutic act that tends to encourage the patient's independence and self-confidence. This is not to deny that the initial response of most patients to approaching termination is one of anxiety and often of regression. Nevertheless, the firm and steady insistence on a termination date and the working through of separation anxieties is critical to the process of short-term dynamic psychotherapy.

The issue of how many sessions should be involved is still unsettled among short-term psychotherapists. Mann sets an arbitrary and fixed limit of 12 sessions, but Sifneos, Malan, Wolberg, and Davanloo are rather more flexible, varying the number of visits from 15 to 40, depending on the severity of the patient's problem as well as his ego-adaptive capacities. For most patients, somewhere between 20 and 30 visits seems to be quite satisfactory. It has been my own practice after termination to leave the door open for a follow-up visit after three or six months, if the patient desires. This removes the element of finality from the separation process and softens its impact, but the majority of patients given such an opportunity do not find a need for it. Indeed, they tend to feel better as time goes on.

The third specific feature of short-term dynamic psychotherapy is the persistent focus throughout therapy on the core conflict and the refusal to permit defensive digressions from that central focus. This in turn leads to the fourth major factor, the activity of the therapist, an essential feature of short-term therapy. This activity has two major and significant psychodynamic elements. First, it is a reflection of the therapist's interest in and concern for the patient and the therapist's wish to be helpful, which are important therapeutic factors. Second, insisting on adhering to the central focus and actively discouraging digressions maintains a high level of therapeutic tension and interaction throughout the therapy more effectively than the traditional "abstinence" patterns of classical psychoanalysis. Sifneos called this technique "anxiety provoking," and it often is.

It should be emphasized that such activity does not mean that the therapist is being directive. On the contrary, the therapist follows essentially the nondirective line of traditional psychoanalytic therapy. The activity consists of persistent confrontations and interpretations and, by means of these confrontations, of the discouragement of regression. Whenever possible, Menninger's "triangle of insight" (28) is used in offering cognitive interpretations; that is, the therapist tries to link interpretations to the historical past, to the patient's present life situation and interpersonal relationships, and to the transference relationship in the therapeutic situation. Active transference interpretations and the bringing out of negative feelings as well as positive ones constitute an essential part of the technique of short-term dynamic psychotherapy.

The emphasis on transference interpretation and on the acquisition of insight clearly demonstrates the

close link between short-term dynamic psychotherapy and classical psychoanalytic theory. As I have indicated elsewhere (26), it is questionable that insight in and of itself has the specific therapeutic effect that most psychoanalysts attribute to it, but it nevertheless serves as a powerful reinforcement to all of the other factors that play a role in the psychotherapeutic process.

COMPARISON WITH CRISIS INTERVENTION

The question is often raised as to how short-term therapy differs from crisis intervention. It may be useful in this regard to distinguish emergency care, crisis intervention, and short-term dynamic psychotherapy, which in a sense represent different points on a continuum of psychiatric intervention. Emergency care is concerned with the provision of immediate relief or help to a person who has decompensated in the face of internal or external stress and is totally unable to cope with the situation. In crisis intervention one is dealing with an individual who is in danger of decompensating from internal or external stress and is coping poorly. The goal of crisis intervention is to reduce or remove the stress situation and/or to help the patient deal with it more effectively.[1] In short-term dynamic psychotherapy we are dealing with individuals in conflict, not necessarily in crisis, although a crisis situation may be involved. The goal in short-term dynamic psychotherapy is primarily on modifying the patient's coping abilities and only secondarily on relieving stress.

It is evident from these definitions that there are no sharp lines of demarcation among these modes of approach. Each can merge into the other. Emergency treatment may proceed from the provision of immediate relief to an effort to reduce the precipitating stress situation, and in crisis intervention it is often necessary to help the patient develop more effective coping mechanisms to deal with the presenting stress situation as well as future ones. To the extent that crisis intervention emphasizes the modification of coping mechanisms it moves closer to short-term dynamic psychotherapy.

The differences are primarily in terms of emphasis. Crisis intervention is usually of shorter duration and limited to five or six sessions, and dynamic psychotherapy is usually of longer duration. The primary goal of crisis intervention is the restoration of homeostasis. A secondary goal is to improve the patient's adaptive capacity when necessary. The basic goal of short-term dynamic psychotherapy is to improve the patient's coping abilities. The termination point of crisis intervention is when the crisis is resolved. The ter point of short-term dynamic therapy is not dic the resolution of the crisis. Crisis intervention i a more supportive approach than does short-t namic psychotherapy. It can also be more dire short-term dynamic therapy, the approach i but nondirective. Crisis intervention deals o the here and now; short-term dynamic psych includes the exploration of the past to illumi present. Finally, crisis intervention may invol riety of other techniques (e.g., family therap therapy, and dealing with the social netwo short-term dynamic psychotherapy is esser one-to-one approach. However, it may well be lessons learned from short-term dynamic psyc py will find increasing application in conjoint family, and group therapies as time goes on.

INDICATIONS FOR LONGER-TERM PSYCHOTHERAPY

The foregoing does not imply that longer-t namic psychotherapy will no longer be nece indicated. Given the present state of our kn the substantial number of people now seen in psychotherapy who do not meet the selectio for the short-term approach will continue to longer-term therapeutic relationships. These include individuals with more seriously impai integrative capacities, particularly the mor character disorders, and borderline syndro every psychiatrist knows, some patients ma tenuous symbiotic relationship with a psycho for most of their lives in order to be able to functioning.

THEORETICAL OBJECTIONS

The opposition of traditional psychoanalys short-term therapeutic model has been base preconception that in order to be deep, therap be long. Indeed, when in the course of psycho therapy patients improved quickly their impr was usually derogated as a transference cure o into health. The assumption was that the ther inevitably incomplete and that sooner or late tient would be back. However, this often did cur. The reason for the faulty assumption was misconception of the traditional psychoanaly el, namely, that it was a closed-system mode trast to the open-system model of contempor chiatric thought. The closed model posited th all aspects of the patient's libidinal fixations conscious conflicts were worked through the sooner or later lead to a recurrence of s atology. Open-system thinking recognizes abling a patient to function more effectively in ular area leads not only to heightened self-est

[1]Some crisis theorists divide crisis therapy into two categories: crisis support, which focuses on the patient's current problems and/or relief of symptoms, and crisis intervention, which deals with the patient's adaptive and maladaptive coping mechanisms in the crisis situation. The latter technique clearly merges into short-term dynamic psychotherapy and differs from it primarily in being of shorter duration and crisis oriented.

...lso to positive feedback from the environment. These ...hanges, both from within and from without, can mod-...y the internal psychodynamic system without having ...) work everything through.

The classical method of psychoanalysis arose as a ...illiantly innovative research technique created by ...reud to explore the furthermost corners of the uncon-...ious. For this purpose it has no equal. A good re-...earch technique, however, is not ipso facto a good ...erapeutic method, and Freud's own later pessimism ...out the results of psychoanalysis reflected this ...wareness. Short-term psychodynamic techniques are ...t in any sense a degradation of the "pure gold" of ...sychoanalysis. On the contrary, they constitute in-...ovative techniques and approaches that open the way ... new objectives and potentials for psychoanalytic ...sychotherapy. They constitute a historical trend of ...e first magnitude and point to the direction in which ...e rational psychotherapies of the future will be mov-...g.

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Phenomenology Associated with Depressed Moods in Adolescents

BY SUBHASH C. INAMDAR, M.D., GREGORY SIOMOPOULOS, M.D., MICHAEL OSBORN, M.D., AND EDGARDO C. BIANCHI, M.D.

The authors studied phenomenology associated with depressed moods in 30 adolescents within a semi-structured interview format using defined symptom items. The results suggest that while there is some symptomatic similarity between adolescent depression and depression in adults, there are many differences that warrant more cautious comparison and further study.

THE VERY EXISTENCE of depression in children has been challenged, but its presence in adolescence has often been presumed. Systematic and detailed studies of affective disorders in adolescents are seldom found (1) while studies of those disorders in children are not so rare. Of the several approaches to adolescent depression, one major viewpoint is that depression like that seen in adults is not possible before adolescence and that late adolescence is the specific phase when this adult picture emerges (2). A second viewpoint, which is presumably based on the belief that there are hidden dynamic similarities in adult depression and childhood and adolescent depressive phenomena, uses terms such as "depressive equivalents" (3) and "masked depression" (4) rather than a phenomenologic definition.

Toolan (3) subsumes under the category "depressive equivalents" a wide variety of symptoms: psychosomatic symptoms, eating and sleeping disturbances, boredom and restlessness, delinquent behavior, and promiscuity. Glaser (4) described "masked depression" as including school phobias and learning difficulties.

Poznanski and Zrull (5) criticized the above terms as being sweepingly inclusive and questioned whether "the depressive side of the equation is even observed." Rie (6) criticized the concept of depressive equivalents along similar lines. He noted that a demon strable connection between the concept of depressio and the behavior considered a depressive equivaler had not been established. He stated that "the use c the term depression demands the identification of th essential nature of the disorder which remains ir variant regardless of the variety of manifestations b which it is recognized."

At present we know of only one clinical descriptiv study (1) that has defined the nature of depressive phe nomena in adolescence; most other studies of adoles cents are based on clinical impressions (7) or case re ports (8). Hudgens (1), in a study of psychiatric dis orders in 110 adolescent inpatients (aged 12–19 admitted to the Washington University Medical Cen ter, reported on the symptomatology of 19 adolescent given a definite diagnosis of depression. Fourteen oth er adolescents who did not fulfill the criteria for th definite diagnosis were called "undiagnosed, most lik depression." The research group at Washington Uni versity is active in the area of diagnostic criteria an has initiated attempts to reduce further the ambiguity of diagnostic criteria for psychopathology in adults b suggesting the use of defined symptom items in struc tured psychiatric interviews (9).

It was the purpose of this study to describe wha adolescents experience in the whole range of phenom ena traditionally linked to childhood and adolescen depression. In this way one could hope at the least t delineate depressive phenomena of adolescence for a specific population and at most to define a form o forms of adolescent depression and to then make clini cal comparisons with defined adult entities.

METHOD

Thirty patients aged 12–18 were selected from thos referred to the Inpatient Adolescent Service of th New York University-Bellevue Hospital Center Twenty patients were aged 12–15 years and 10 patient were aged 16–18. The median age was 15.

The cases were selected as follows: The psychiatri staff identified patients they thought had "depresse moods" and the investigators then applied the criteri listed below.

1. Report of depressed mood as a major psycho pathological feature and confirmation of this in th semi-structured interview.

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2. Absence of schizophrenia, mental retardation, ganic brain syndrome, or major medical illnesses rsonality disorders were not excluded).

Weitzel and associates (10) thought a free-form non-stematic approach to the clinical interview increased risk of overlooking important areas of inquiry. A riety of self-assessment scales, rating scales, and uctured interviews have been developed to over-me the problem of unstructured interviews. These struments were all judged to have limitations (9), and y have been used mainly with adults. The addition-problems of modification and use of such in-uments with younger adolescents would be consid-ible.

We used a semi-structured interview format. The in-view began with a brief introduction to the purpose the study. A few open-ended questions were posed out the present state of the patient in order to en-nce responsivity to the exploration of affective tes. The interview then proceeded to a list of ap-oximately 50 items on a schedule that evolved from urvey of the literature on depression in children and olescents. The adolescents were evaluated jointly teams of two psychiatrists (S.I. and G.S., S.I and .O.) who used an interviewer/observer design.

The duration of scale administration ranged from 50 90 minutes, with an average of about 1 hour. The ms were asked in fixed sequence, but the inter-ewer allowed room for elaboration and for probing ibiguous responses. Definitions of symptom items re designed to enable interviewers to decide what nstituted affirmative responses. Most of the major ms were defined on the basis of a glossary of terms ed by Wing and associates in the Present State Ex-ination (11), a structured-interview instrument. ms not used by Wing were included and defined, g., running away and diminished school perform-ce.[1]

We included all possible features of depressive phe-mena previously noted as being present in child-od or adolescent depression. However, we ex-ided phenomena that involved inferences from be-vior which could not be assessed in the interview .g., insatiable affection-seeking [12]), as well as char-teristics drawn from data about children in pro-nged psychoanalytic contact (e.g., "therapist diffi-lty in making sustained contact" or "tendency to gress to oral passivity" [13]).

We attempted to make the schedule as comprehen-ve as possible to avoid missing any significant phe-menologic details. In doing so, we intentionally in-ided partially overlapping categories. Because fur-er elaboration was sought on many items, the terview protocols were rich in detail about patients' bjective experiences.

opies of this schedule and scoring instructions are available on equest from the authors.

RESULTS

Table 1 lists the symptoms commonly associated with depressive moods in this study and their prevalence in a study of adolescent inpatients by Hudgens (1) and a study of adult inpatients by Woodruff and associates (14). These two studies, carried out at different times at the same center, were chosen for comparison because they covered a range of symptomatology similar to that in our study. We included any symptom that occurred in at least 50% of any study group and that could be compared with the present study. Seven items in the present study that had no comparison figures in the other studies were also included. It should be noted that interpretations of the comparisons are limited by the lack of operational definitions of symptoms in the other studies. A symptom was considered present in a patient if there was inter-observer agreement. Specific agreement was high (94%). All of the adolescents selected for this study had depressed moods by virtue of the sample selection criteria, whereas the percentage in the comparison studies was slightly less.

Examination of the table reveals that the inclusion criteria selected a group of patients who had some symptom similarity and who experienced many of the depressive features seen in adults. However, we did not find the following symptoms noted in adults by Woodruff and associates (14): restlessness, pacing and hand wringing, agitation, motor retardation, feelings of persecution, delusions of guilt, hopelessness, and delusions at the time of examination. Affective hallucinations, defined as hallucinations with content congruent with a depressed mood, were seen in two subjects in our study (7%). Except for these two, none of our adolescent subjects had the severe symptomatology seen in the adults studied by Woodruff and associates. Hudgens did not report the presence of these symptoms reported by Woodruff and associates or of two symptoms that could be considered important in a study of adolescents: diminished school performance and running away.

Items related to sexuality, such as promiscuity and diminished libido, while rated, were considered difficult to assess within our study design and therefore were thought to be possibly unreliable for comparison. The subjects were reticent about discussing sex, and determination of diminished libido, which entails the establishment of a baseline of sexual activity, was even more difficult in a group with reportedly irregular sexual contacts.

The interview protocols were rich in such details as precipitating circumstances, length of episodes, and circumstances that could terminate the affective state, so we attempted to formulate a composite clinical picture for this population. We included symptoms that were present in more than 50% of the sample.

These adolescents had experienced depressed moods as a major psychopathological feature for a period of a few months to several years. The date of onset was

TABLE 1
Prevalence (in Percents) of Symptoms Associated with Depression

Symptom Items	Adolescents: Current Study (N=30)	Adolescents: Hudgens (1) (N=19)	Adults: Woodruff and Associates (14) (N=54)
Depressed mood	100	95	93
Loss of interest	77	79	89
Loneliness	53	—	—
Feels unloved	53	—	—
Apathy, boredom	90	—	—
Loss of pleasure	83	—	—
Suicidal ideation	77	84	41
Suicidal acts	60	68	11
Poor concentration	70	84	93
Thoughts slowed	37	—	69
Memory disturbance	30	53	—
Self-depreciation (worthlessness)	57	63	74
Social withdrawal	63	—	—
Irritability	33	42	67
Diminished school performance	73	—	—
Running away	57	—	—
Insomnia (delayed sleep, early waking)	57	74	61
Loss of appetite-weight loss	40	53	78
Subjective anergia (tired all day)	30	58	57
Tired upon awakening	53	—	31
Diminished libido	7	—	50
Somatic symptoms (headaches, abdominal pain, muscle aches, dizziness, nausea, constipation)	37	—	74

difficult to ascertain accurately. Some patients could mark the onset by relating it to an important incident, such as a death in the family at a specific age, but others felt that their depressed moods started so gradually that the time of onset could not be defined precisely. Therefore, although it is important, age at onset was not determined.

It is extremely important to note that most of the adolescents looked sad *only* when they were talking about their sad moods. These moods were not transient, ephemeral states that could be considered normative. Although episodic, they were intense, disabling, and associated with many other symptoms. Despite this, the majority of these patients did not specifically complain of depression as a symptom for which they sought relief. The nature, extent, and chronicity of the depressed moods and associated symptoms were revealed only by systematic inquiry. Depressive moods were precipitated primarily by external events and lasted from a few hours to a day or two. Most subjects viewed their depressive moods as stemming from conflict with a significant other and therefore did not see a need for therapeutic intervention.

The depressed moods of these adolescents were typically associated with apathy and boredom, loss of interest, and loss of pleasure in activities that were usually enjoyable. During their depressed moods many the adolescents were socially withdrawn. These associated symptoms lasted from a few hours to a day two and thus matched the duration of the depressed moods. However, during these periods, a close friend could terminate this self-imposed isolation and allow reengagement in favorite activities. The adolescent often felt lonely and unloved, and this feeling was associated with self-deprecatory ideation and a poor self-image. Inability to concentrate and social withdrawal were reflected in diminished school performance. Suicidal ideation and acts and running away from home could be seen as attempts to deal with all of the chronic problems. Insomnia, defined as delayed sleep or early waking, and tiredness upon awakening were frequently present.

It is important to note that loss of interest, apathy, boredom, loss of pleasure, poor concentration, diminished school performance, and suicidal ideation were present in over 70% of the sample, whereas memory disturbance, irritability, loss of appetite-weight loss, subjective anergia, thought slowing, diminished libido, and somatic symptoms were present in less than 40%.

DISCUSSION

Depression in adolescence has been considered similar to that in adulthood, but the type of adult depression on which the comparison is based has often been unclear. The classification of affective disorders in adults is itself considered to be in disarray (14). Our study suggests that although there are many similarities between the features of adolescent and adult depression there are also many differences, at least in early and mid-adolescence, which warrant more cautious comparison and further study. Thus, our study is not in agreement with Hudgens' conclusion that adults and adolescents are similar with respect to the clinical picture of depression.

"Masked depression" and "depressive equivalents" have not been defined precisely enough to allow comparison, but symptoms that are associated with these diagnostic terms, such as irritability, aggressive behavior, antisocial activity, and promiscuity, were infrequent in the present study. The lack of clarity in the initial clinical picture, including the absence of "observed depression" (i.e., the patients looked sad or cried transiently during the interviews) may have contributed to the origin of the terms "masked depression" and "depressive equivalents." Although the adolescents in this study did not initially look sad, they were found on systematic inquiry to have a broad psychopathological symptom profile, along with the more easily identifiable behavioral components. This initial clinical picture may also be the basis for clinicians' underrecognition of depression in adolescents.

It has been suggested that "prevailing attitudes in Child Psychiatry . . . fail to stress a systematic clinical approach to diagnosis" (15). This is also the case in

olescents: "Myths alluding to the tumultuous nature adolescence and the benign affects of adolescent mptom formation continue to abound" (16), and di-nostic criteria are not adequately defined. The study defined symptoms associated with depressed moods ring adolescence is an approach that could help es-olish diagnostic criteria in follow-up studies for a m, or forms, of depression during adolescence. Pos-le future prospective and retrospective studies uld link child, adolescent, and adult depressive phe-mena and define the "underlying invariant nature" the disorder. We are planning a follow-up study that ll attempt to define diagnostic criteria and to deter-ne the clinical course and natural history of the af-ctive disorder in this sample.

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A special issue of the *American Journal of Psychiatry* will be published in mid-April 1979. This issue, which will be distributed to all subscribers and is in addition to the regular April issue, will consist of a collection of articles on the subjects of alcoholism, depression, or both. Articles will deal with such topics as the treatment of depression with imipramine and ECT; bipolar illness: factors in drinking behavior; physical activity and plasma cyclic AMP in manic-depressive patients; childhood depression; life events at the onset of bipolar affective illness; psychiatric problems in women at alcoholic detoxification; lithium maintenance; efficacy of drugs, and psychotherapy in the treatment of acute depressive episodes; and life events, coping, stress, and depressive symptomatology.

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Mental Health Services for Medicaid Enrollees in a Prepaid Group Practice Plan

BY SIDNEY S. GOLDENSOHN, M.D., AND RAYMOND FINK, PH.D.

The authors present the patterns of utilization of mental health services of a low-income population in a prepaid group practice setting, the Health Insurance Plan of Greater New York (HIP). Over a 6-year period, utilization rates for psychiatric consultations and mental health services by Medicaid enrollees rose to equal or exceed utilization by employed groups. The authors also provide evidence that psychiatric treatment reduces the use of other physician services, especially X-ray and laboratory services.

IN THESE TIMES of growing welfare expenditures and spiraling health care costs, the method of delivery of mental health care to low-income persons has become increasingly important. For more than a decade Medicaid programs have provided financial support for health care services for the poor. During that time modalities of treatment, costs, and effectiveness of services have been studied.

We will describe patterns of utilization of the mental health care provided in a prepaid group practice setting to low-income (Medicaid) enrollees and compare these patterns with those of employed groups. The characteristics of the therapeutic program as they affect accessibility and utilization will be noted. We will also present data from a study conducted for the National Institute of Mental Health on the effect that use of mental health services has had on this low-income population's utilization of nonpsychiatric health services.

This study is based on information derived from about 16,000 Medicaid enrollees from six medical groups in the borough of Queens that are affiliated with the Health Insurance Plan of Greater New York (HIP), a prepaid medical group practice. When mental health treatment was indicated, these enrollees were referred to the Mental Health Service of HIP's Queens-Nassau Center, which has been described elsewhere (1, 2). brief, mental health services are available to HIP su scribers on the basis of a rider to the prepaid heal insurance plan. Approximately 180,000 enrolle bought the psychiatric rider. New York City acquir the rider coverage for about 48,000 of its welfare reci ients. The 1978 annual premium was $9.48 per persc There are no deductibles or copayments, and, course, no premium costs to Medicaid enrollees. broad spectrum of mental health services is offere The basic therapeutic orientation is psychodynam although this orientation may be significantly modifi for low-income enrollees. The goal of treatment is return the patient to a functioning level as quickly possible. Usual in-hospital treatment modalities a provided. Psychotropic drugs, both in and out of t hospital, are supplied to the patient without additior cost.

UTILIZATION OF MENTAL HEALTH SERVICES

In 1970, the second full year in which mental hea services were provided to Medicaid enrollees, servi utilization rates were lower for Medicaid enrolle than for the other two major contractor groups who members are under 65 years of age—federal emplc ees and their families and New York State employe and their families. Since 1970, however, service uti zation rates of Medicaid enrollees have increased, a in 1976 they exceeded the rates of the federal a New York State employees. Rates were 271 servic per 1,000 Medicaid enrollees; 223 per 1,000 federal e rollees; and 199 per 1,000 New York State enrollee

These changes in Medicaid enrollees' utilizati rates reflect increases in both the rate of those enteri the treatment system through initial consultation a the average number of services per treated patient. 1970, the consultation rate was below that of both fe eral and state employees and their families, but it h also increased to rates similar to those of the fede group and in excess of those in the state group (s table 1).

The increases in utilization took place during a pe od of declining Medicaid enrollment. Concurrentl the number of services to HIP Medicaid enrollees l nonpsychiatric physicians also increased. During t period from 1970 to 1976, Medicaid enrollees' avera annual utilization of nonpsychiatric physician servic

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BLE 1
lization Rates (per 1,000 population) by Contractor Group in the Queens-Nassau Mental Health Center

rollees' ntractor Group	1970		1972		1974		1976	
	Consultation	Services	Consultation	Services	Consultation	Services	Consultation	Services
groups under 65 years	12.4	187	15.3	234	13.1	220	13.9	221
:dicaid	10.2	152	17.4	257	15.1	250	14.7	271
deral*	13.6	233	17.7	285	15.3	246	16.4	223
ıte	13.4	176	12.8	225	10.9	195	11.7	199

cludes about 1% in each year 65 years of age or older not covered by Medicare.

)r those under 65 years of age) increased from 149 r 100 enrollees to 361 per 100. In 1970 the ratio of edicaid enrollees' services to non-Medicaid (under years of age) enrollees' services was .60, increasing .85 in 1976. In contrast, as observed above, Medi- id utilization of mental health services in 1976 ex- eded that of non-Medicaid persons eligible for these rvices.

These findings are of particular interest when one nsiders the characteristics of the Medicaid popu- :ion. Medicaid enrollees tend to be less educated an the other subscriber groups; studies (3, 4) have own that those with little education tend to be lower ilizers of mental health services.

It is commonly believed that the low-income person so has a greater tendency to drop out of treatment rly. Our utilization picture, however, is more com- ex. Among those who start treatment, Medicaid pa- nts are more likely than others to end therapy before e sixth visit. Data compiled during the 3-year period om January 1972 through December 1974 show that eatment ended before the sixth visit for 53% of the edicaid enrollees, 43% of federal enrollees, and 35% state enrollees. These data are reported in greater tail elsewhere (5). However, of patients who stay in erapy until the sixth visit, Medicaid patients attend ore sessions than the other groups: the average num- r of sessions per patient per year in 1976 was 18.4 for edicaid patients and 15.4 for employee groups.

'ECIAL CONSIDERATIONS THAT FFECT UTILIZATION BY LOW-INCOME AMILIES

The ability to establish therapist-patient rapport is a quirement for any program aimed at reaching those need of mental health care. Among low-income fam- es, a population that is generally less likely to use alth services, special approaches are required to hieve this goal. For all patients, low income and oth- s, the accessibility of mental health services is clear- essential. In the Queens-Nassau Center easy access attained through unrestricted self-referrals and also rough family physician referrals. Financial consid- ations do not enter the decision to seek therapy; edicaid patients are reimbursed for carfare to the nter. Further, staff are available for urgent or walk- in appointments. Required psychotropic medications are provided at the center, so that needed drugs are obtained without delay.

Once a patient is in contact with the Mental Health Service, the ambience provides further incentive to use the program. The physical setting has little resemblance to the clinic atmosphere so often associated with health care in organized settings. Receptionists are friendly and welcoming. Comfort and understanding are increased by an interracial and bilingual (Spanish) professional staff, since Spanish is the first language of much of our Medicaid population.

Therapeutic orientation is strongly influenced by community mental health principles (6) and techniques. There is emphasis on promotion of functional competence rather than self-actualization and an adherence to a multidetermined view of etiology—psychological, social, biologic, genetic, and educational. Intervention takes place as much as possible within the real-life settings of family, friends, work, school, and community. There is much therapeutic reliance on natural helpers such as spouse, parents, siblings, and peers, and a three-step therapeutic technique of advocacy-mediation (7) is used for and welcomed by many of the underprivileged patients, particularly those who have never been financially self-sufficient.

The Medicaid population can be divided into those who have never been financially self-sufficient and those who have. The latter group are better able to respond to the more usual psychodynamic modalities. In contrast, the former group, often generational welfare families, respond best to concrete help and advocacy-mediation techniques. Since there is overlap, flexibility is required, and we have learned that the traditional therapeutic relationship may still be the treatment of choice for individuals at any socioeconomic level.

EFFECT OF MENTAL HEALTH SERVICE ON UTILIZATION OF OTHER HEALTH CARE SERVICES

The desirability of integrating mental health care and medical care is generally accepted. The benefit of ease of referral has already been noted. Another benefit is the facilitation of close communication between the physician and psychotherapist; the psychiatrist has

for too long been isolated from the primary care physician. Still another benefit—that there will be a reduction in use of nonpsychiatric health services as a result of the use of mental health services—is not as well proved, although the issue has been systematically examined on several occasions. In 1967 Follette and Cummings (8) reported on changes in medical utilization among samples of Kaiser Plan enrollees matched for age, sex, and emotional impairment; one sample (the treated group) had received mental health care from the Kaiser Plan, the other had not. Over a 3-year period following mental health care, the treated group was found to have had fewer health care services (including hospitalization) than the untreated group. In 1968 the Health Insurance Plan of Greater New York (HIP) (9) compared changes in utilization over a 2-year period and observed a tendency toward a reduction in the use of other health services by a group which had received mental health care. In 1969 Group Health Association of Washington, D.C. (10) observed a drop in the use of non-mental health services among patients who had used mental health services. None of these studies was able to obtain a control group of untreated persons who had sought psychotherapy. Nevertheless, there is a widespread impression that mental health services do contribute to a reduction in utilization of other health services.

In 1972, a special study was conducted among a random sample of HIP Medicaid enrollees treated in the Queens-Nassau Mental Health Center to examine the relationship between mental health treatment and utilization of other health services. Like the studies just cited, this study could not generate a randomized control group of patients who had sought psychotherapy and did not receive any; therefore the same reservations in interpretation pertain to this report as to the others.

The Samples

Two random samples of Medicaid enrollees were selected. One, designated as the treatment group, consisted of 263 persons who received one or more mental health services at the Queens-Nassau Mental Health Service of HIP in 1969 or 1970. The second, consisting of 206 persons and designated as the diagnosis group, was a random sample from the same HIP medical groups in Queens for whom an HIP physician indicated on a routine reporting form in 1970 a condition classifiable under the International Classification of Diseases as a mental, emotional, or psychological condition. Nearly all of these were diagnosed by family physicians; none was treated at the Mental Health Service. Medical records covering a 2-year period were reviewed for both groups. For the treatment group, services were counted for a full year preceding the first psychiatric consultation that led to treatment and the full year subsequent to onset of treatment. For the diagnosis sample, services were counted for the full year preceding the date of the diagnosis and the year following the diagnosis. (The physician visit in which the diagnosis was given was not included in the count of services.)

TABLE 2
Sex and Age of Medicaid Diagnosis and Treatment Samples

Variable	Diagnosis Sample (N=141) N	%	Treatment Sample (N=1 N
Sex			
Male	27	19	68
Female	114	81	101
Age			
Under 25	27	19	88
25–44	35	25	51
45 and over	79	56	30

Since the review was limited to those persons f whom information was available for the full 2-year o servation period, the treatment group was reduced 169 persons (64% of the original sample) and the dia nosis group reduced to 141 persons (68% of the ori nal sample). Age and sex characteristics of ea sample were tabulated,

Substantial demographic differences were found the age and sex breakdown between the treatment a diagnosis samples; the treatment group had ma more males and was younger than the diagnosis gro (see table 2). This skew is due to the large number young males with school behavior problems: ma boys with behavioral disorders bypass their fam physicians and are referred directly to the Men Health Service by their schools. The 25–44 year a group provides the sharpest picture of the differenc between the psychiatrically treated and untreat groups, with 56% of the diagnosis group and 18% the treatment group in this category.

Differential Utilization of Services

The two groups were compared on their use of fa ily physicians, specialists, and X-ray and laborato services. In addition to presenting the average numb of services for each group, we also calculated the n change between the year before and the year after, a the net difference between the treatment and diagnos groups. A negative measure indicates a change in t direction of fewer services for the treatment group.

Effect on family doctor services. In comparing fa ily physician utilization, there was a modest drop f the treatment group and a slight rise for the diagnos group. For the treatment group, the average number family physician services in the year before treatme was 3.8 and for the year after the start of treatment was 3.4 services, a drop of 0.4 services. In contra the diagnosis group had an increase of 0.2 services f the year following physician diagnosis. The net d ference for the total sample comparisons, therefore, −.6, indicating a relative decline in family physici services for the treatment group (see table 3). Age a sex differences are also provided; especially notewo

BLE 3
nge in Use of Family Doctor Services

	Mean Before		Mean After		Net Change		
iable	Diagnosis	Treatment	Diagnosis	Treatment	Diagnosis	Treatment	Net Difference
lale	3.9	1.9	3.6	2.3	− .3	.5	+ .8
emale	4.8	5.0	5.1	4.1	+ .4	− .9	−1.3
nder 25	2.5	2.2	3.2	2.2	+ .8	+ .1	− .7
5–44	3.9	4.8	4.9	3.8	+1.1	−1.0	−2.1
5 and over	5.7	6.6	5.3	6.3	− .4	− .3	+ .1

LE 4
nge in Use of Specialist Services

	Mean Before		Mean After		Net Change		
iable	Diagnosis	Treatment	Diagnosis	Treatment	Diagnosis	Treatment	Net Difference
lale	1.0	1.1	1.2	1.1	+.2	0	− .2
emale	1.3	2.7	1.7	2.2	+.4	− .6	−1.0
nder 25	1.3	.9	.8	.9	−.4	0	+ .4
5–44	1.2	2.7	2.1	2.4	+.9	− .3	−1.2
5 and over	1.3	4.3	1.7	3.1	+.4	−1.2	−1.6

BLE 5
nge in Use of X-Ray and Laboratory Services

	Mean Before		Mean After		Net Change		
iable	Diagnosis	Treatment	Diagnosis	Treatment	Diagnosis	Treatment	Net Difference
lale	7.1	4.5	9.4	4.8	+2.3	+ .3	− 2.0
emale	9.4	14.1	11.8	9.7	+2.5	−4.5	− 7.0
nder 25	6.1	4.6	7.0	3.8	+ .9	− .8	− 1.7
5–44	5.4	14.9	14.6	10.9	+9.2	−4.0	−13.2
5 and over	11.5	19.5	11.3	13.4	− .2	−6.1	− 5.9

was the net difference of −2.1 for the 25–44 year group and − 1.3 for women (see table 3), in both es declines in the direction of reduced utilization the treatment sample.

ffect on specialist services. The treatment sample a decrease of .3 specialist services between the ar before and the year after the start of treatment the diagnosis group had an increase of .3 specialist vices; the net difference was therefore a decrease of 5 specialist services. The net difference for the men's treatment group was −1.0, and for the 25–44 r age treatment group −1.2 (see table 4), more evi- ce of reduced utilization of non-mental health serv- s following psychiatric treatment.

ffect on X-ray and laboratory tests. By review of dical records, we were able to count each X-ray and oratory test procedure; each procedure was nted as a service. The net difference between the groups, −5.0 services, was surprisingly large (see le 5).

The marked reduction in X-ray and laboratory services following psychotherapy contrasts with a more modest reduction in other medical services. One possible reason may be that some primary physicians are uncomfortable and frustrated when they are medically responsible for an intensely nervous patient who is not under psychiatric care; as a result, these physicians may tend to temporarily "dismiss" this patient with a referral for an X-ray or laboratory procedure. Another speculative explanation for the large differences in X-ray and laboratory utilization by the two groups may be that the psychiatrically untreated sample had a higher incidence of psychosomatic or conversion symptoms than did the treated sample. Still another possibility is that psychotherapy brings about a reduction in somatic symptomatology. Unfortunately, we do not have the kind of data that could test these hypotheses. Future research that looked at the physical diagnoses of both samples under study could be fruitful; it might identify the kinds of conditions that show the greatest reduction in medical services as a result of mental health care.

DISCUSSION

During the period from 1970 through 1976, utilization rates for new psychiatric consultations and mental health services by Medicaid enrollees rose to equal or exceed utilization by employed groups. This was true in spite of the higher early dropout rate of the Medicaid enrollees.

Comparisons between utilization rates of non-mental health services by Medicaid enrollees treated in the Mental Health Center and Medicaid enrollees diagnosed as having emotional problems but not treated provided evidence that psychiatric treatment reduces the use of other physician services and especially of X-ray and laboratory services. Although these findings are not consistent in every age and sex comparison, they occur with sufficient regularity to support the hypothesis of reduced medical utilization for those receiving treatment for emotional conditions. Thus, while the poor require as many or more mental health services as do middle-income groups, the cost for these services may be somewhat reduced by lower utilization of other services following psychotherapy.

In a household survey among samples of Medicaid enrollees treated at the Mental Health Center and those diagnosed but not treated at the center, 17% of the treated group indicated they had also sought care in other settings, while among the diagnosed but untreated group 11% had sought mental health treatment outside of HIP. Among those who were treated at HIP, satisfaction with the care was high: three out of four said they would seek treatment again in the HIP Center, and two-thirds reported the treatment as being either very or fairly helpful.

We now have 10 years of experience in providing mental health care to the Medicaid population in a prepaid medical group practice setting. On the whole, we believe that this is a feasible method of delivering mental health care to a low-income group. While there are variations in their utilization patterns, over a period of time the differences appear to be minor and ca dealt with appropriately. Innovative therapeutic n agement of the low-income multiproblem family mains a continuing challenge in any delivery syst but treatment in a setting that also includes blue-c and middle-income patients may be a therapeutic p A problem of some magnitude that remains is the stantial attrition rate of Medicaid enrollees from medical plan itself. It is necessary that we deter the causes of the attrition and reverse the trend.

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ne "Moonies": A Psychological Study of Conversion and embership in a Contemporary Religious Sect

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authors undertook this study to enhance chiatric understanding of contemporary rismatic religious sects. After a pilot study, a resentative sample of members of the Unification rch (N = 237) completed a 216-item structured stionnaire. Respondents were below the mean for ıge- and sex-matched group on a psychological eral well-being scale, and they reported ificantly greater neurotic distress before version. The authors discuss correlates of an roved emotional state following conversion and loy attribution theory, drawn from social chology, to put the conversion process into a chiatric perspective.

S STUDY was undertaken to investigate the psychocal state of members of a charismatic religious cult ı regard to the nature of their conversion and coned membership. In recent years there has been cern about the techniques used to effect consions and retain members in certain contemporary gious cults. This has presented a challenge to psyıtry as a discipline, since psychiatrists are frequentalled upon to deal with these matters in both theratic and legal settings. However, psychiatrists are n confronted by issues not readily subsumed under r usual diagnostic and phenomenologic approach. order to shed light on these issues, we elected to roach the Unification Church since this group has n the focus of much concern regarding its effect on verts. The church cooperated because of an exssed interest on the part of its administrative leadıip to demystify the organization in the eyes of the lic by means of an impartial, professional study.

he Unification Church was introduced into the ited States by Korean missionaries in 1961. There are chapters throughout this country, as well as allied branches in Korea, Japan, and western Europe. Members of the church have been referred to as "Moonies" because they are followers of the religious leader Reverend Sun Myung Moon. Their considerable efforts at serving their church have focused attention on the depth of members' commitment. In addition, the efforts of family members to "deprogram" this commitment have raised both legal and psychiatric questions regarding the appropriateness of such undertakings.

METHOD

The project was carried out over a period of 6 months, during which we initially conducted psychiatric interviews and psychological testing of 20 church members to clarify individuals' relationship to the movement.

Questionnaire

On the basis of this and previous work by one of us (1), a 216-item computer-codeable questionnaire was composed and refined by pretesting on church members. Three scales were included in the questionnaire, each composed of a series of items.

1. *Neurotic Distress Scale.* This 8-item scale was used previously for retrospective assessments in relation to stages of membership in a religious sect (1). A score of 1–5 (not at all, slightly, moderately, a lot, and very much) is given to each of a series of statements that reflect neurotic symptoms. Sample items from this scale include, "I felt nervous and tense," "I felt depressed and glum," and "I had trouble controlling my thoughts." Respondents were to choose an answer for each item for each of 4 periods: 1) the most symptomatic period before contact with the church, 2) right before first contact with the church, 3) right after conversion, and 4) the last 2 months.

2. *Religiosity Scale.* This 9-item scale was devised to reflect the intensity of religious commitment to the church. Items dealt with the following: feeling "a close connection to God"; gaining meaning from prayer and meditation; feeling that others "should adopt the same religious beliefs that I do"; and avoiding "thinking about sex" or "getting high from alcohol." Responses were solicited for attitudes for periods 2–4 on the Neu-

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rotic Distress Scale. The choices of answers were those used for the Neurotic Distress Scale.

3. *General Well-Being Schedule*. This 18-item scale was developed to measure psychosocial adjustment in a self-administered assessment of health status (2). Scaled responses to items addressed issues such as, "How have you been feeling in general?" "Have you been feeling emotionally stable and sure of yourself?" and "How happy, satisfied, or pleased have you been with your personal life?" A comparison group was drawn from a study of 1,196 representative adults from Dayton, Ohio (3), with the collaboration of Dr. John Ware. The comparison group (N=305) was balanced with the respondent group for age and sex. The degree of well-being in this schedule is construed as proportional to the reported scale scores. The Neurotic Distress Scale score for the last two-month period and the General Well-Being Schedule score for the last month had a high inverse correlation in this study population ($r=-.64$, $p<.001$).

The questionnaires were administered under our direct supervision to a representative sample of full-time members who lived in the church's residences in a large metropolitan area. Standardized instructions were used in all settings. Respondents were also instructed by senior church figures to give frank answers to the anonymous questionnaire. Because it was an officially sanctioned church activity, full cooperation was elicited. A total of 237 questionnaires completed by native-born Americans were used for data analysis; these were from a total sample that included an additional 70 questionnaires from non-Americans. Because of the mobility of church members in the United States, this sample included members from all regions of the country.

FINDINGS

Respondents' Backgrounds

Most of the members were unmarried (91%), white (89%), and young (mean age=24.7 years). Male members predominated (61%); this is true for the membership of the church nationwide (62%) as reflected in church records. Two-thirds (69%) had been members over 18 months, and the mean duration of membership was almost 3 years (33.5 months). Most (67%) regarded themselves as at least moderately committed to their family's religion before the age of 15. At some point thereafter, one-third (34%) became at least moderately committed to one of the Eastern religious sects, such as the Divine Light Mission, Zen, and Muktananda, and a smaller proportion (19%) to fundamentalist Christian sects. However, fully 90% reported a history of at least some prior commitment to these sects. This figure may be compared with the much smaller proportion (47%) who indicated at least some past commitment to a political party or movement.

Almost half (42%) had attended school on at least a half-time basis during the 6 months before joining the church. Upon joining, however, members tende leave school; only 25% of the members completed lege, although more than half (58%) had begun. dropping-out process began before entering church, since less than one-third (31%) of the resp ents who were students during the 6 months be conversion were actually enrolled full-time.

TABLE 1
Mental Health Scale Scores

Item*	Score Mean	SD	t
Neurotic Distress Scale (study group, N=237)			
Most symptomatic period before contact with church	20.5	6.8	
Right before first contact	18.0	6.0	17.73
Right after conversion	12.6	3.7	1.5
Last 2 months	12.2	3.3	
General Well-Being Schedule			
Study group (N=237)	74.4	17.2	6.24
Comparison group (N=305)	83.4	16.2	

*A product moment correlation was done for the mean scores of the group on the Neurotic Distress Scale (last two months) and the G Well-Being Schedule ($= -.64$, $p<.001$).

Members experienced a fair amount of psychol cal difficulty before joining the church. A sizable tion (39%) felt that they had had serious emoti problems in the past. This had led many to profess al help (30%) and even hospitalization (6%). Thei sponses yielded neurotic distress scores that were nificantly higher before joining than at the time of study (table 1). Even though they reported a declin neurotic distress over the course of membership, t current emotional well-being, reflected in the Gen Well-Being Schedule, was lower than that for matched comparison group. In addition, their prior of drugs was high. One-quarter (23%) said that t had had serious drug problems in the past, and the p tion who had ever used drugs of abuse was higher t for a comparable national sample (for example, us hallucinogens was 45% versus 14%) (4).

Conversion

Conversion usually occurred during a series church-run workshops. For those who volunteere remain, a typical sequence might be a 1-day works followed immediately by a 2-day workshop and th 7-day workshop. The workshop day runs from 8 a.m. to 11:00 p.m. and includes lectures and group cussions related to religious issues, as well as sp and entertainment that are sometimes related to r gious themes. Virtually the entire day, incluc meals, is spent in the immediate company of works and church members. Initially, participants are o not aware that the workshop is associated with church. However, in our interviews members repo no overt coercion or physical deprivation during period. In fact, when they were asked to indicate strongest action taken at any point to discourage th

m leaving the church, 97% indicated that there was more than some disapproval or a little argument. owever, 2 respondents (1%) did report members vertly limiting [their] freedom of movement or trav- but for less than an hour."

The large majority of respondents (91%) gave re- onses that yielded lower neurotic distress scores for e period right after conversion than for the period ht before, with a mean decline of 30%. This is com- rable to a decrease from "moderately" (score 3) to lightly" (score 2) for typical items, such as "I felt pressed and glum." Religiosity was associated with greater decline in symptoms. For example, if sub- ts are ranked according to the degree of improve- nt in distress, the quartile who improved most owed a 128% increase in their reported religiosity ores over the course of conversion, while those who proved least showed a 76% increase (one-tailed t st, t=3.67, p<.01).

Most of the respondents recalled the conversion pe- d in a positive way. A large portion reported having e following feelings "a lot" or "very much" more an usual: "a great deal of respect for another per- n" (67%), "cheered up" (43%), "close or intimate th other people" (48%), and "an appreciation of the ality of God" (76%). For some, however, the experi- ce entailed psychologically disruptive components. me respondents reported having the following expe- nces "a lot" or "very much" more than usual: onflict or unhappiness" (19%), disruption of eating 2%) and sleeping (21%) habits, and feeling they were nder someone else's influence" (12%).

Transcendent experiences were not uncommon dur- g the conversion period, with many respondents re- rting that they experienced the following either retty clearly" or "very intensely": "a special unfa- iliar feeling in my body" (45%), "the presence of meone important to me, even though most people uldn't have seen him" (35%), and "time passing ower or faster than usual in a very special way" 9%). Not uncommon was the type of experience de- ribed by one member who had no prior or sub- quent history of psychiatric disturbance. After a pe- od of prayer at home following a workshop, he saw e face of the Reverend Mr. Moon in front of him and ard him speaking directly, revealing his mission, "as al as we're talking now."

ntinued Membership

"Full-time" membership in the church entails a ma- r life commitment. For example, in the 2 months be- re the survey our respondents, who were full-time embers, spent almost all (mean = 94%) of their nights a Unification Church residence. Commitment to reli- ous principles was also very high; regarding belief, r example, three-quarters (74%) felt a "close con- ction with God" "a lot" or "very much." Specific ohibitions were closely adhered to, and 76% felt that ey should avoid thinking about sex "very much," as posed to only the small minority (11%) who felt this way before joining the church. A large majority of respondents (90%) indicated that they had "pretty clearly" or "very intensely" "seen a special new meaning in life" during daily prayer. There was, in fact, a significant correlation between current scores on the religiosity scale and scores on the General Well-Being Schedule (r=.34, p<.001).

Members' commitment was also reflected in the number of hours worked per week at a regular job, charitable work, or course work. They reported an average of 67 hours, compared with 41 before joining. Street proselytizing and sales was the principal job activity for the majority (52%) of members. This zeal was reflected in most members' strong feeling that people who were not members of the church should "adopt the same religious beliefs that I do" (80%) and "avoid thinking about sex," as they did (86%). It should be noted that the number of hours of work was not significantly correlated with sex, age, duration of membership in the church, or score on the religiosity scale. It was, in fact, positively correlated with the general well-being scores (r=.19, p<.05).

However, the general well-being scores were significantly below the mean for the age- and sex-matched comparison group. As indicated in table 1, there was no significant net change in the mean neurotic distress score from the time right after conversion until the time of the questionnaire, but there was considerable variation in change among individual members (mean change=3.2%±26.3%).

In order to examine this variability, items associated with an increase in the neurotic distress score after conversion were ascertained in two stepwise multiple regression analyses. The two criterion (dependent) variables were 1) the relative increase in the reported neurotic distress score from the 2-month period right after conversion up to the time of questionnaire administration (first analysis), and 2) the actual neurotic distress score for the current 2-month period (second analysis).

The variables tested as predictors (independent variables) were drawn from items related to personal background, prior neurotic distress scores and changes in score, and experiences during conversion. These independent variables were entered into the regression equation as long as they accounted for a sufficient portion of the variance to be significant at the .01 level. The 10 best predictors in each of the regression analyses are listed in table 2 in the order in which they were entered. A subject's increases or decreases in neurotic distress scores were defined relative to the mean change for all members; for example, an increase of 10% in the score was considered as a relative decline if the mean change for all subjects was an increase of 25%.

The first analysis (table 2) provided information on the relative importance of demographic and experiential variables in accounting for variance in the amount of change in neurotic distress scores after the conversion period. Variables associated with an in-

TABLE 2
Results of Stepwise Multiple Regression Analyses

Item	Time Period	R	R^2	Simple r	Beta Weig
First analysis: predictors of an increase in neurotic distress score after conversion period					
Decline in work satisfaction	After the conversion period	.31	.09	.31	.1
Decline in theistic belief	After the conversion period	.37	.13	.30	.3
Relative increase in Neurotic Distress Score	Over the course of conversion	.40	.16	−.20	−.4
Higher Neurotic Distress Score	Immediately before conversion	.47	.22	−.11	−.3
Reported pressure from members to remain in church	Most ever	.50	.25	.15	.1
Decline in perceived interpersonal support	Over the course of conversion	.52	.27	.12	.1
Degree of religious commitment	Immediately before conversion	.53	.28	−.01	−.3
Less increase in theistic belief	Over the course of conversion	.55	.30	−.08	.2
Female gender*		.56	.32	.12	.1
Increased commitment to alcohol prohibition	Over the course of conversion	.57	.33	.02	.1
Second analysis: predictors of higher neurotic distress scores at the time of questionnaire administration					
Neurotic Distress Score	Immediately before conversion	.44	.19	.44	.6
Relative increase in Neurotic Distress Score	Over the course of conversion	.57	.32	.04	.4
Decline in theistic belief	After the conversion period	.62	.39	.23	.3
Less reported increase in interpersonal support	Over the course of conversion	.64	.41	.08	.1
Level of parental education	Highest achieved	.65	.43	.20	.1
Reported pressure from members to remain in church	Most ever	.66	.44	.22	.1
Highest Neurotic Distress Score ever	Before conversion	.67	.45	.36	.1
Decline in perceived interpersonal support	Since the period after conversion	.67	.45	.05	.0
Overall religious commitment	Immediately before conversion	.68	.46	−.07	−.3
Relative decrease in theistic belief	Over the course of conversion	.69	.48	.00	.2

*Versus male gender.

crease included a decline in theistic belief and work satisfaction after conversion, along with less religious commitment before conversion. In addition, subjects reporting a higher neurotic distress score before conversion and a relative increase in neurotic distress score over the course of conversion were more likely to experience an increase in the neurotic distress score during subsequent membership.

A higher score at the time of the questionnaire was associated with the following constellation: less religious commitment right before joining and a decrease in theistic belief over the course of membership (table 2). However, one-third of the variance in the current neurotic distress score was accounted for by a high score before conversion and a relative increase in the score over the course of conversion.

DISCUSSION

In studying such a sect, questions may be raised as to the reliability of survey responses. Objective information on current circumstances can probably be taken at face value, given the instructions for frankness from both the church organization and the research team, although subjective reports, particularly retrospective ones, may be vulnerable to unintended distortion (5). It should be noted, however, that self-assessments of mental state on the General Well-Bei Schedule were not higher than those for the co parison group, even though enthusiasm might have l to an overstatement of good feelings. Findings are, a plicable only to converts who retained membersh since those who had dropped out would most lik have presented some critical differences in their sponses. With these points in mind, the following sues bear emphasis.

The average convert apparently experienced en tional distress before joining. The scores reported the Neurotic Distress Scale for the period before c version were considerably higher than those for period after conversion, or for the present. In additi current scores reflected somewhat greater distr than those of a like-aged comparison group. The lik hood of a higher incidence of psychiatric illness in t population is indicated by the high percentage of th who had sought prior professional help and hospit ization for emotional problems.

Affiliation with the Unification Church apparen provided considerable and sustained relief from n rotic distress. Although improvement was ubiquito a greater religious commitment was reported by th who indicated the most improvement. Similar i provements have been reported for affiliation amc members of Eastern religious cults (1, 3) and those Erhard Seminars Training, a hortatory self-impro

ent group (6). In addition, the anxiety-reducing as-
cts of religious experience have also been noted to
ay a role as a suicide preventive (7).

Continued membership also appeared to support a
rtain stability in psychological status, as seen in the
milarity between the mean scores for neurotic dis-
ess both right after conversion and at the time of the
rvey. After an average of almost 3 years, most sub-
cts still reported a diminished level of distress. How-
ver, an increase in distress over the course of mem-
rship was reported by some subjects, such as those
ho reported a decline in work satisfaction and reli-
ous commitment over the course of membership.
lembers who reported greater improvement in neu-
tic distress over the course of conversion tended to
turn to their previous levels of distress over the
urse of membership.

The nature of religious conversion may be discussed
ith recourse to the findings presented. Lifton has dis-
ssed noncoercive "personal change," in contrast to
thought reform [brainwashing]" (8). He quoted Wil-
am James, saying that "conversion is in its essence a
rmal adolescent phenomenon, incidental to the pas-
ge from the child's small universe to the wider in-
llectual and spiritual life of maturity" (8, p. 469). It
ay be noted that many of our respondents were in-
ed in such "passage" prior to joining. This was re-
cted in their generally premature movement away
om the school setting and their interest in Eastern
d fundamentalist religious groups. In addition to this
ansition in commitment, many were apparently ex-
riencing considerable emotional distress, leaving
em open to an alternative perspective that would
ovide relief.

The issue of brainwashing has been raised regarding
ntemporary cults. In discussing brainwashing and
ought reform, Lifton described changes in world-
iew forceably effected in military captives. This
rves to highlight some similarities to as well as dif-
rences from the conversion context described here.
elevant to both situations is the importance of some
ntrol over the milieu of communication. The Unifi-
ation Church workshops entail a series of carefully
ructured experiences that give increasingly more ex-
licit messages for commitment. On the other hand,
ose present at the workshops had voluntarily pre-
nted themselves. They were more likely to be open
a new perspective than the brainwashed, physically
erced subjects who were unwilling captives at the
itset. The brainwashed captives described by Lifton
ere typically abused and coerced into compliance,
mething not reported by our subjects.

Attribution theory (9) presents a model for decision
aking that is drawn from social psychology and
osely related to the theory of cognitive dissonance
). It points to the tendency of individuals to uncon-
iously seek "plausible explanations for their feel-
gs among whatever explanations are available in an
vironment" (10, p. 323), that is, to attribute causa-
on based on the available cognitive input. Attribution theory has recently been applied to the study of religious conversion. Its operation may be observed when there is a disequilibration of a person's subjective state, such as was recalled regarding the conversion period by respondents in relation to their feelings and their perceived interpersonal milieu. With the cognitive input during this period determined by the church members directing the workshop, the participants may be implicitly encouraged to attribute their altered experience to a new explanatory set. The potential relief of neurotic distress experienced over the course of religious affiliation may indeed play an implicit role in this process.

This description serves to highlight some aspects of cognition, affect, and communication in relation to conversion. The converts' needs for social affiliation would certainly influence responses to a setting where conversion may occur. We have dealt with this latter issue elsewhere at greater length, using data from another sect (11).

Conversion might be compared to the crystallizing role of the experience of significance sometimes found at the onset of delusion formation, in the face of emotional disruption on a much more severe and pathological level. Indeed, most respondents (71%) did recall that there was one point at which their conviction to join coalesced. However, a salient difference is the autistic origin of the experience of significance, as compared with attribution during the conversion experience, which is consensually validated within the context of the religious group.

After the initial conversion, life experiences and activities gain meaning within the elaborated beliefs of the attribution set. For example, it has been pointed out that for American faith-healing subjects, within their "assumptive world view" their personality structure and magical beliefs are not abnormal, but rather are part of a coping system that provides ego integration (12). Reflections of this are seen in the positive relationship between the respondents' diminished state of distress and the intensity of their religious commitment, as well as their willingness to increase work hours, with greater work hours correlated with greater emotional well-being.

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Effects of Interpreters on the Evaluation of Psychopathology in Non-English-Speaking Patients

Y LUIS R. MARCOS, M.D., MED.SC.D.

Non-English-speaking patients in need of psychiatric services are usually evaluated with the help of an interpreter. Discussions with psychiatrists and lay hospital interpreters who had experience in these interviews and content analysis of eight audiotaped interpreter-mediated psychiatric interviews suggested that clinically relevant interpreter-related distortions could lead to misevaluation of the patient's mental status. The author notes that pre- and post-interview meetings of clinicians and interpreters have been found useful in minimizing these distortions.

N NEW YORK CITY, patients whose primary language is not English constitute a significant proportion of the psychiatric population. A number of these patients speak enough English to allow some communication in this language. The problems involved in the psychiatric evaluation and treatment of this bilingual population have been the subject of recent experimental and clinical studies (1–5). However, many patients' competence in the English language is so low that communication can be established only with the help of an interpreter.

At Gouverneur Hospital and Bellevue Psychiatric Hospital, two New York City institutions affiliated with New York University, an average of 30 psychiatric interpreter-interview evaluations are conducted daily. In both hospitals the two most commonly spoken languages apart from English are Spanish and Chinese (i.e., Cantonese and Toisonese). Neither hospital has official interpreters (to my knowledge this is true of all psychiatric institutions) so any available individual who can speak the patient's language may be asked to interpret in evaluations of the non-English-speaking patient. The interpreter may be another patient, a patient's friend or relative, or a member of the hospital staff.

To my knowledge, the effect of the interpreter-mediated interview on the assessment of psychopathology has not been studied systematically, and an exhaustive literature review revealed only a brief description of the interview's process (6) and one clinical report (7). In that report, Sabin reviewed two cases of suicide by Spanish-speaking patients who were evaluated and treated by English-speaking psychiatrists with the aid of an interpreter. Because the evaluating clinicians had made the same clinical error independently of one another, Sabin ruled out clinician effect as an explanation for the outcome and concluded that the patients' emotional suffering and despair may have been selectively underestimated in the process of interpretation. His report was a retrospective study based on a review of the patients' psychiatric records, so it is difficult to evaluate the extent to which the clinicians' written reports contributed to their conclusions.

A review of the general literature on interpretation and translation did not offer any particular information applicable to the special practice of psychiatric interviewing through an interpreter. Most authors in this field, however, concur that faithfulness is the fundamental object of interpretation (8). My purpose in this paper is to explore the process of interpreter-interview assessment of psychopathology and to identify the major distortions that arise in this common practice. I will present specific recommendations to aid English-speaking clinicians who examine patients through an interpreter in minimizing errors.

METHOD

Two consecutive procedures were planned. The first included open discussions with psychiatrists and lay hospital interpreters who had experience in interpreter-interview evaluations. The second procedure, the main focus of the study, consisted of content analysis of audiotaped psychiatric interpreter-interview evaluations. For the first procedure, eight exclusively English-speaking psychiatrists and six bilingual hospital employees with extensive experience in interpreter interviews participated in unstructured discussions designed to obtain their impressions about problems in interpreter-interview examinations. In these dis-

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cussions particular emphasis was placed on the following questions:

1. What are the general problems involved in the process of evaluating psychopathology through an interpreter?
2. What areas of mental status examination are more apt to be distorted by this procedure?
3. What are the ideal characteristics of interpreters who translate in a psychiatric interview?
4. What type of patients tend to have more difficulty adapting to this interview procedure?

For the second procedure, a total of eight psychiatric evaluations of non-English-speaking patients conducted through an interpreter were audiotaped for content analysis. The patients' native languages were Chinese or Spanish. They were seeking psychiatric help for a variety of emotional difficulties: two patients were diagnosed as suffering from involutional depression, two from depressive neurosis, and four from chronic undifferentiated schizophrenia. Their average age was 50 years. The patients had previously expressed willingness to volunteer for the study. An English-speaking psychiatrist conducted the interviews with the help of an interpreter. The English language questions were identical and were presented in the same order. The questions are the items of the Psychiatric Evaluation Form Interview (9), a distillate of the common probes used in psychiatric evaluations.

Three different types of lay interpreters who had no knowledge of the aims of the study participated in this procedure. Four interviews were interpreted by a psychiatric nurse with 10 years of experience in clinical psychiatry; two by a nurse's aide who was psychiatrically unsophisticated and worked in a different department, and two by the patients' relatives. All interpreters were proficient bilinguals, selected because they represented the type of individual commonly asked to participate as an interpreter in hospitals.

In the analysis of the recorded material, one English-Spanish and one English-Chinese bilingual psychiatrist listened to the respective audiotapes and compared the English portion with the Spanish or Chinese portion to assess their clinical equivalence. Specifically, these clinicians compared 1) the interviewing psychiatrist's questions with the interpreter's translation of them to the patient, and 2) the patient's answers with the interpreter's translation of them to the interviewing psychiatrist.

The clinicians who listened to the tapes were familiar with the conclusions of the first procedure (i.e., discussions with psychiatrists and lay hospital interpreters experienced in interpreter-mediated evaluations) and used those conclusions as the starting point in their comparative analysis. The rating clinicians were allowed to listen to the tapes as many times as they wished. Because this study was exploratory, no specific hypotheses were tested. The primary aim of this analysis was to identify patterns of distortions associated with the interpreter-interviewer procedure and with the different types of interpreters.

RESULTS

Psychiatrists' and Lay Interpreters' Views

General problems of interpreter-interv tions. All the psychiatrists who participate cussion agreed on frequent practical prob ated with the use of volunteer interpreter: preters often consider this practice "toc responsibility," an imposition, and a bot terruption of their regular job activities. T trists emphasized distortions stemming fr preter's attitude toward either the patient cian (e.g., an interpreter's overidentificat patient leading him or her to challenge th suggestions to the patient). Dynamics such terfere with the faithfulness of the interp create tension during the interview.

Most of the interpreters agreed that th embarrassed or anxious about some of th questions to the patient. These questions with sex, financial matters, and the explor cidal or homicidal tendencies. All of the felt overwhelmed by the responsibility o translators. Both psychiatrists and lay int pressed significant concern for the prote confidentiality of the patient's communic

Mental status evaluation through an Psychiatrists indicated that the assessme aspects of thinking and of affect (specifical al withdrawal and blunted affect) had a h bility of being distorted by the interpret dure. Also, certain ambivalent attitudes o the patient were difficult to evaluate th preters. Part of the problem appeared to translatable paralinguistic and vocal cue available to the clinician.

Characteristics of a good interpreter. believed that competence in both languag familiarity with the patient's culture we quirements. It was also suggested that the knowledge of clinical psychiatry could be minishing distortions, particularly in the a the formal aspect of thinking and affect. thought that as a rule patients' relatives o not make good interpreters because they l tivity.

Patients and the interpreter-interview With regard to the vulnerability of patients go these interviews, there was agreement t paranoid and very anxious patients usuall culty coping with the interpreter-intervie Some psychiatrists, however, suggested th patients who react positively to this prac they recognize in it extra interest and att

Analysis of the Recorded Interviews

The content analysis of the recorded in terview evaluations revealed a variety o that, in many instances, confirmed th pressed by the psychiatrists and lay in

eir previous discussions. The findings will be orgaed according to three major types.

1. Distortions associated with the interpreter's lantage competence and translation skills. Although, in neral, the interpreters were proficient in the source d target languages, various types of clinically relent errors of translation were detected. These includ omissions, additions, substitutions, and conensations. Such distortions appear to be related both insufficient language competence and to deficient inrpretative skills. Examples are given below.

Clinician to Chinese-speaking patient: "What kind of moods have you been in recently?"
Interpreter to patient: "How have you been feeling?"
Patient's response: "No, I don't have any more pain, my stomach is now fine, and I can eat much better since I take the medication."
Interpreter to clinician: "He says that he feels fine, no problems."

Clinician to Spanish-speaking patient: "Do you feel sad or blue, do you feel that life is not worthwhile sometimes?"
Interpreter to patient: "The doctor wants to know if you feel sad and if you like your life."
Patient's response: "No, yes, I know that my children need me, I cannot give up, I prefer not to think about it."
Interpreter to clinician: "She says that no, she says that she loves her children and that her children need her."

These examples were extracted from recorded interews conducted through psychiatrically unsophistited interpreters. In both cases it is obvious that the inician did not get an accurate impression of the paents' responses because of a combination of transtion errors such as omissions, condensations, and bstitutions. In the first case, the interpreter failed to mmunicate to the clinician that the patient had foused his response on his improved physical condition ther than his mood. In the second example, the paent's ambivalent answer about her feeling and life as not conveyed to the clinician. In fact, the intereter's translation indicated signs of tangential thinkg more than anything else.

Several distortions were also detected when the inrpreters attempted to translate patients' long anvers. Usually, interpreters' attempts to summarize swers resulted in a loss of important information.

2. Distortions associated with the interpreter's lack of ychiatric knowledge. The most frequent distortion in is category consisted of the interpreters' "normalation" of patients' thought disorders, such as circumstantiality, tangential thinking, loose associations, d blocking. It was obvious that interpreters often ied to "make sense" of the patients' disorganized atements, which prevented the clinician from detectg these important aspects of the mental status. The llowing verbatim example is self-explanatory:

Clinician to Spanish-speaking patient: "What about worries, do you have many worries?"
Interpreter to patient: "Is it there anything that bothers you?"
Patient's response: "I know, I know that God is with me, I'm not afraid, they cannot get me [pause] I'm wearing these new pants and I feel protected, I feel good, I don't get headaches anymore."
Interpreter to clinician: "He says that he is not afraid, he feels good, he doesn't have headaches anymore."

Also, as suggested in the discussions with psychiatrists and lay interpreters, the quality and depth of the patient's affect could not be assessed reliably through the interpreter. This may be related to the fact that interpreters tend to limit their translation to *what* the patient says, neglecting *how* the patient says it.

3. Distortions associated with the interpreter's attitudes. Interpreters who were relatives of the patient tended either to minimize or emphasize psychopathology. For example, the daughter of a patient clearly emphasized the patient's psychopathology while expressing to the clinician her belief that her mother should be hospitalized. In another instance the interpreter expressed to the patient his own negative feelings about the medication the clinician had just suggested. Because this was done in the patient's language, the clinician did not notice it. Similarly, in another interview the clinician was exploring the possibility of a tubal ligation with a patient, and the interpreter openly expressed her disagreement with this procedure and suggested that the patient not go along with it.

Another common observation was that relatives serving as interpreters answered the clinician's questions to the patient without actually asking the patient. For instance, the son of a patient was asked to inquire about his father's possible suicidal ideation. Without asking his father, he insisted on a negative answer.

DISCUSSION

The above findings suggest that clinicians evaluating non-English-speaking patients through an interpreter are confronted with consistent, clinically relevant, interpreter-related distortions which may give rise to important misconceptions about the patient's mental status. In addition to the relative untranslatability of some communicative components (e.g., the formal aspect of speech, expression of affect, and paralinguistic aspects), three major sources of interpretative distortions have been identified: 1) deficient linguistic and/or translation skills of the interpreter, 2) interpreters' lack of psychiatric sophistication, and 3) interpreters' attitudes toward either the patient or the clinician.

It would be optimal if there were enough competent psychiatric interpreters so that English-speaking clinicians could reliably evaluate non-English-speaking patients. Failing this, it is important that clinicians be made aware of the interpreter effect on the translation

of psychopathology. At this point some recommendations can be made to clinicians who must use the interpreter interview.

First, it is advisable that clinician and interpreter meet before the interview to discuss the goals of the evaluation, the focal areas to be assessed, and any particularly sensitive topic that may have to be explored (e g., suicidal or homicidal ideation, hospitalization, family relations). At this time, the clinician should make sure that the interpreter has an acceptable level of competence in the two languages. It is also important to explore the interpreter's attitude toward the interview and the patient, particularly when the interpreter is a relative or acquaintance of the patient or has expressed some preconceived ideas about the patient's problems. The clinician should also discuss confidentiality and emphasize the significant role of the interpreter as an objective intermediary in the evaluation process.

The clinician should make an extra effort to clarify for psychiatrically unsophisticated interpreters the differences between formal and content aspects of the patient's verbalizations (e.g., how the patient says things versus what the patient says). The need for textual translation should be emphasized, as well as the danger involved in attempting to "make sense" of the patient's statements. Interpreters should also be encouraged to ask for clarification from the patient or the clinician at any time during the interview.

Similarly, a postinterview meeting sho ranged between the clinician and the inte this session the clinician should look for clar both the interview material and the dynan interaction. Further, the interpreter shoul ample opportunity to verbalize and proces ings that may have been aroused during the i

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ong-Term Effects of Traumatic War-Related Events on Sleep

Y PERETZ LAVIE, PH.D., ALBERT HEFEZ, M.D., GILA HALPERIN, M.SC., AND DANIEL ENOCH, M.D.

even patients who had combat neuroses resulting om the 1973 Yom Kippur War and complained of ep disturbances were studied in a sleep laboratory. eep-onset insomniacs, dream-interruption somniacs, and pseudoinsomniacs were fferentiated on the basis of electrophysiologic cordings. Compared with normal controls who tively participated in the Yom Kippur War, patients owed significantly longer sleep latencies, lower ep efficiency indices, lower percentage of REM ep, and longer REM latencies.

HE OCCURRENCE of sleep disturbances in the initial ages of the combat neurosis syndrome has been early established. Bartemeier and associates, in a de- led description of combat neurosis, wrote, "There almost unanimous agreement that the first symp- ns of the failure to maintain psychological equilibri- n are increasing irritability and disturbances of ep" (1, p. 374). Sleep disturbances included diffi- lties falling asleep and staying asleep, battle dreams, d recurrent terrifying dreams and nightmares. aigie (2), on the basis of 2 years' experience in the iddle East during World War II, listed insomnia as e fourth most common complaint of combat neurosis tients.

Less is known about the long-term effects of trau- atic war-related events on sleep. A group of 57 veter- s, studied 15 years after their combat experiences , showed a high incidence of insomnia and night ter- rs. In a follow-up study of 92 veterans of the Viet am War, it was found that their combat experience ntinued to disturb them long after they returned to vilian life—in some cases in the form of severe sleep sturbances (4). Another interesting finding emerged a follow-up study of survivors of marine explosions; ore of them complained of sleep disturbances 3.5–4.5 ars after the disaster than immediately following it (5). There is one report of a study of combat neurosis in a sleep laboratory in which electrophysiologic recording techniques were used (6). The authors reported that "in striking contrast to patients' reports of frequent nightmares prior to the study, only two clear nightmares were reported during the many nights of recordings" (pp. 30–31). This statement contradicts the nonlaboratory findings, which emphasized the role of nightmares and frequent battle dreams as the primary cause of sleep disturbances in combat neurosis patients. No other sleep disturbances were mentioned in that report.

In the present paper, we report sleep laboratory data on combat neurosis patients from the 1973 Yom Kippur War who were referred to our sleep laboratory with complaints of persistent sleep and/or emotional disturbances. An age-matched control group of active service veterans of the same war, who had been exposed to similar stresses but survived without psychological breakdown, was also studied.

METHOD

Subjects

Eleven patients, aged 24–33 years (mean=26.54, SD=3.35) were referred to the sleep laboratory 2–2.5 years after their traumatic experience. All of them were hospitalized during or shortly after the war for treatment of combat neurosis. Hospitalization periods ranged from 2 to 12 weeks, during which patients were given pharmacologic as well as psychotherapeutic treatment. None had a record of previous hospitalization or psychiatric treatments, and all claimed to have had normal sleep before the war. Nine of the 11 had been hospitalized after exposure to especially acute traumatic events, which in 5 cases were associated with physical injury. Patient 10, for instance, functioned normally for a week as a tank commander in the Sinai desert. Then his vehicle took a direct hit by an air-to-ground missile and he was thrown from the tank and suffered a head concussion. Emotional disturbances appeared while he was hospitalized for medical treatment, and psychiatric treatment was initiated.

In 2 patients neurotic symptoms appeared gradually over periods of weeks or months. Patient 3, for instance, served on the Golan Heights in a front-line technical unit that operated under stressful conditions. Five or 6 weeks after the cease-fire he began to exhibit neurotic symptoms—sleep disturbances, irritability,

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depression, etc.—and was hospitalized in a psychiatric treatment center. All of the patients studied have received psychiatric treatment since the war.

In the psychiatric interviews before the sleep recordings, the most common complaints were depression, irritability, uncontrolled fits of rage, headaches, pain in various parts of the body, and sleep disturbances. The latter consisted of difficulty falling asleep, fragmented sleep, and frequent nightmares; in all cases, however, there had been considerable improvement since the war. Ten of the 11 patients had not been taking drugs for more than 2 weeks prior to the study. One patient used 5 mg of nitrazepam (Numbon) once or twice a week and no firm withdrawal period could be established, but since the sleep recordings showed no drug effects his data were included in the analysis.

Nine men aged 24–27 years were recruited from the Technion student body as controls. They were required to continue their normal everyday lives during the course of the study.

Sleep Recording Procedures

Patients and controls reported to the laboratory about an hour before their regular bedtimes and, except for 2 subjects whose observation continued for only 2 nights, slept in the laboratory 3 or 4 consecutive nights at a time. Totals of 34 and 27 nights were available for patients and controls, respectively. All participants were fitted with electrodes for recording EEGs, electro-oculograms (EOGs), electromyograms (EMGs), ECGs, and respiration (measured with a nostril thermistor and a respiratory belt); tibialis EMG was also recorded in some patients. All recordings were done on a Beckman type R411 dynograph according to established procedure (7). All sleep records were scored according to international criteria (7) in 30-second epochs. Data were averaged over all laboratory nights, excluding the first night, which was considered as adaptation to the laboratory. Data were averaged for the 2 subjects who were studied for 2 nights because no differences were found between the 2 nights.

The following parameters were extracted from each record: total time in bed; total sleep time; sleep latency; REM latency; percentages of sleep stages 1, 2, 3–4 (combined), REM, and wake time plus movement time after sleep onset; number of REM periods; length of first three REM periods; and length of the first and second sleep cycles measured from the beginning of one REM period to the beginning of the next one. Statistical significance of the differences between the 2 groups was assessed by two-tailed t tests.

RESULTS

Significant differences between patients and controls were found in REM latency ($p<.02$), REM percentage ($p<.002$), and length of the third REM period ($p<.04$). Total sleep time was shorter and sleep efficiency indices lower in patients than in contr ($p<.05$) (see table 1). According to the sleep efficier index, the patients fall into 2 subgroups (table 2): tients 1–5 had indices of 0.89 or more, sleep latenc of less than 15 minutes (mean=8 minutes), and l than 7% wake time after sleep onset plus movem time. Patients 6–11 had indices below 0.85, with mu longer sleep onset latencies (mean=41 minutes), a exhibited two types of sleep disturbances. In 2 of t latter group (patients 10 and 11), the low index v primarily due to a large increase in wake time a sleep onset, accumulated in multiple mid-sleep aw enings. The remaining patients in the subgroup exh ited long sleep latencies with few mid-sleep aw enings. The hypnograms in figure 1 demonstrate t different types of sleep disturbances and indicate a that most mid-sleep awakenings in patient 10 cluste around REM periods or at expected times of REM riods. A similar temporal pattern of mid-sleep aw enings was observed in patient 11. These repetitive terruptions of REM sleep suggest that patients 10 a 11 suffered from dream-interruption insomnia (8). F ther evidence is provided by the fact that they were only ones to report war-related nightmares on aw ening from REM sleep in the laboratory. Moreov most REM interruptions were preceded by increa heart rate. Similar relationships between autonor arousal and REM nightmares were described by Fi er and associates (9).

TABLE 1
Electrophysiologic Sleep Measurements for Patients and Controls

Measurement	Patients (N=11) Mean	Patients (N=11) SD	Controls (N=9) Mean	Controls (N=9) SD	
Sleep latency (minutes)	28.7	26.2	19.6	16.2	n
Sleep efficiency index	84.9	9.5	91.1	3.2	.(
Total sleep time (minutes)	345.4	50.8	377.4	23.6	.(
REM latency (minutes)	99.9	34.5	68.6	15.2	.(
Sleep stages (percents)					
Wake and movement	6.5	6.5	4.5	2.8	n
1	2.4	1.3	2.1	1.74	n
2	44.7	6.6	42.2	8.1	n
3 and 4	29.3	8.7	28.4	4.9	n
REM	16.8	2.7	22.6	4.5	.(
Number of REM periods	3.4	.5	3.7	.6	n
Length of REM periods (minutes)					
R1	11.2	6.2	13.6	7.6	n
R2	19.4	6.7	20.2	8.1	n
R3	26.1	12.4	36.1	7.6	.(
Length of sleep cycles (minutes)**					
T1	90.6	24	94	12	r
T2	113.2	27.5	98.2	22.9	r

*One-tailed t test.
**Measured from beginning of one REM period to beginning of the next

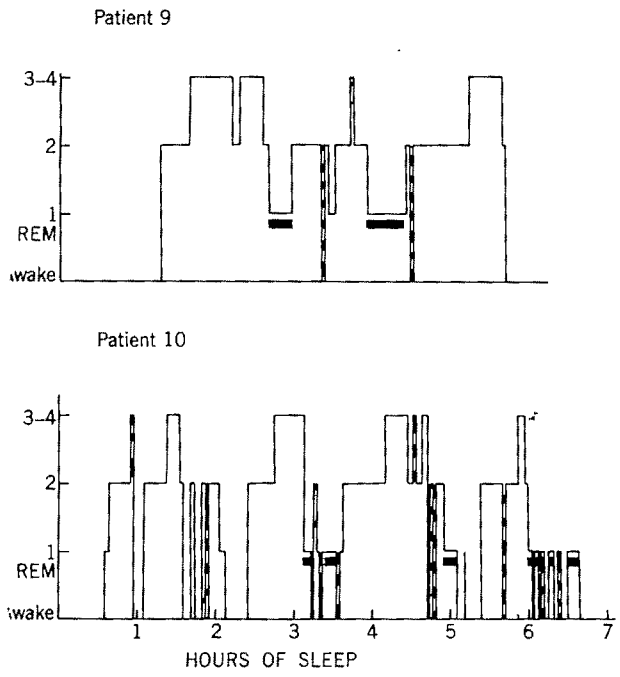
Additional Findings

In addition to changes in sleep architecture, so other observations in the patients' sleep data are wc

3LE 2
ctrophysiologic Sleep Data for Patients

ient	Sleep Efficiency Index	Sleep Latency (minutes)	Sleep Stages (percents)				
			Wake and Movement	1	2	3 and 4	REM
1	95.4	3.8	2.0	0.7	36.0	43.3	17.3
2	94.8	3.5	1.6	2.6	42.7	30.6	22.2
3	93.0	10.0	6.8	0.0	42.5	39.3	14.0
4	89.3	10.3	5.5	2.3	57.3	19.0	15.0
5	89.2	12.7	2.8	5.2	50.1	26.9	14.7
6	87.2	37.5	2.8	1.7	52.2	26.8	16.1
7	85.6	30.0	3.6	2.2	48.6	27.5	13.8
8	77.5	77.7	5.3	1.9	36.1	39.6	18.3
9	77.1	71.7	1.6	1.5	42.8	32.9	20.5
10	67.8	42.5	20.0	4.4	43.4	16.9	15.1
11	75.9	16.5	18.8	2.0	40.4	20.4	17.9
tal							
Mean	84.8	28.7	6.5	2.4	44.7	29.3	16.8
SD	9.5	26.2	6.5	1.3	6.6	8.7	2.7

URE 1
ep Hypnograms of Sleep-Onset Insomnia (Patient 9) and Dream-
rruption Insomnia (Patient 10)



ting. Patient 3 manifested an interesting variant of eep disturbances. In the preliminary interview he ade no mention of actual sleep disturbances but complained of severe fatigue and dizziness in the morning iat often persisted throughout the day. Sleep recordgs revealed an apparently normal amount of sleep, ith an index of 93.0, but there were an unusual numer of gross body movements, averaging 0.20/minute ver his 3 recording nights. On the third night, after he entioned occasionally waking up with "aching legs," bialis EMG recordings were taken and revealed myonic jerking of the legs at a rate of about 1/minute uring the first 3 hours of sleep. These leg movements did not interfere with the sleep process, which was predominantly of stages 3 and 4. Two other patients revealed similar rates of body movement, and both had indices higher than 0.88.

In 4 patients, the recordings indicated modifications toward a more aroused sleep. Patients 10 and 11, who had multiple mid-REM awakenings, also exhibited during sleep stage 2 and REM sleep frequent trains of alpha activity that persisted for 2–5 seconds and differed from the alpha-delta sleep pattern observed earlier in psychiatric patients (10). Similar alpha trains were observed in a third patient with a sleep-onset insomnia; a fourth patient demonstrated a dramatic increase in the rate of K complexes during sleep stage 2: 2.72, 1.60, 2.59, and 2.59 per minute for the 4 recording nights, respectively, compared with the mean rate of 1.21/minute reported for normal individuals of similar age (11).

None of the controls exhibited similar sleep modifications. A detailed comparative study of some of these episodic sleep phenomena between patients and controls is currently being done in our laboratory.

DISCUSSION

Our investigation demonstrated that exposure to traumatic events which lead to psychological breakdown has long-term effects on sleep; these could be detected in the laboratory 2 to 2.5 years after the trauma. Like insomnia in general the combat neurosis version studied here was a combination of sleep-onset insomnia, dream-interruption insomnia, nocturnal myoclonus, and pseudoinsomnia. It is not certain, however, whether all 5 patients whose sleep was adequate were actually pseudoinsomniacs. The high rate of nocturnal body movements observed in 3 of these patients (who complained of fatigue on awakening in the morning, usually persisting throughout the day) suggests that classification as a nonrestorative sleep syndrome is more appropriate in this case. The in-

crease in nocturnal motor activity may represent a basic disturbance of the sleep process that is not reflected in changes in sleep architecture. Unaware of their increase in motor behavior, the patients misattributed their morning feeling to increased sleep latency and mid-sleep awakenings. Takahashi and associates (12) also reported increased rates of body movements in pseudoinsomniacs. It should be emphasized, however, that based on patients' complaints in prerecording interviews, patients 1–5 could not be differentiated from patients 6–11.

The reduced REM percentages and increased REM latencies in this sample are surprising, since most patients complained of emotional dreams and of occasional nightmares. The possibility that the reduced REM time was an artifact of short sleep periods or was secondary to depression can be ruled out, since patients and controls with matched sleep periods differed significantly in REM time, and patients free of any depressive symptoms showed reduced REM time. Although we have no immediate explanation for the reduced REM time on the basis of existing literature data, it might be a means of forestalling highly emotional dreams. Except for 2 patients with dream-interruption insomnia, none of the subjects reported nightmares in the laboratory; all of them spoke of almost nightly nightmares immediately following the trauma, and some described forcing themselves to stay awake to avoid recurrent terrifying dreams. If this is the case, dynamic changes in REM sleep parameters should be found at different times after the trauma, and patients who have completely recovered should have norm near normal amounts of REM sleep. These quest are still open.

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ersonality, Sexual Adjustment, and Brain Lesions in Patients ith Conversion Symptoms

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clarify the interrelationships of conversion nptoms, hysterical personality, sexual adjustment, d cerebral organic disorders the authors studied 89 tients with classic motor conversion symptoms oup L) and compared a subgroup of 24 of these tients (group H) with 24 matched control patients oup C). They found hysterical personality in 19% of ɔup L, 21% of group H, and none of group C and ssive-immature-dependent personality in 19% of ɔup L, 21% of group H, and none of group C. Forty-ht percent of group L, 50% of group H, and 58% of ɔup C had some cerebral disorder. Patients with nversion symptoms who also had hysterical and ssive-immature-dependent personalities were ɔecially likely to have sexual abnormalities.

PARTICULAR, DRAMATIC TYPE of personality has ditionally been associated with hysterical symptoms. aepelin (1) and Lewis (2) have provided vivid deriptions of this type. Chodoff and Lyons (3), hower, pointed out that the "so-called hysterical consrsion reactions do not occur solely by any means in tients who present the characteristics of the hysical personality." De Alarcon (4) criticized Chodoff d Lyons and others who reached similar conclusions the grounds that their studies were retrospective. e Alarcon observed, "It may be safe to assume that obably all patients with symptoms of hysteria have me features of what we call hysterical personality." e demonstrated that in 14 papers on hysteria, includg that of Chodoff and Lyons, the following characristics were repeatedly mentioned for the hysterical rsonality: histrionic behavior, egocentricity, emoɔnal lability, dependency, excitability, seductivess, suggestibility, and childishness. Lazare and asciates (5) further demonstrated that features of the sterical personality, as defined in the literature, can discriminated in psychiatric patients. There is thus od evidence that hysterical personality has definable features that may have demonstrable psychogenetic characteristics.

Conversion symptoms were explained by Breuer and Freud (6) as the consequence of unconscious sexual conflict. Wartime experiences led to the view that conflicts other than sexual ones might be responsible for such symptoms (7). There have been very few reports recording the frequency of impairment of sexual relationships in patients with hysteria. Purtell and associates (8) studied 50 patients with hysteria and reported that 86% of them showed sexual indifference and 73% had no pleasure from sexual activity. They also stated that 98% of their women patients with hysteria had some kind of sexual symptom and that the most common was indifference.

The definition of hysteria of Purtell and associates, as well as that of Perley and Guze (9) and that of Winokur and Leonard (10), conforms to Guze's definition (11), which involves patients who are a different population from those who have conversion symptoms only. It was the latter who were the subjects of the classical observations of Charcot (12) and Breuer and Freud (6) and in whom a link between the symptom and sexual repression was first implied. Further, although it is generally accepted that fear of sexuality is one factor in the hysterical personality, there are few hard data to support this view apart from some experimental observations in students (13–15).

Other work (16–18) has suggested that cerebral organic disease has a role in causing hysterical symptoms. The interrelationships of conversion symptoms, hysterical personality, sexual adjustment, and cerebral organic disorders remains unsettled. In this paper we record evidence regarding the link between personality and sexual maladjustment in 89 patients with conversion symptoms. We compare a subgroup of patients with conversion symptoms with matched psychiatric patient control subjects. We also examine the relationship between conversion symptoms and personality and the presence of cerebral organic disorders.

METHOD

The 89 patients with conversion symptoms (group L) reported on here comprise a series of personal cases seen by one of us (H.M.) at the National Hospitals for Nervous Diseases at Queen Square and at Maida

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Vale between September 1968 and December 1971. Details of the selection of these patients as well as details of the selection of the control group of psychiatric patients matched for age and sex with 24 of the 89 index cases were given in 1975 by Merskey and Buhrich (18). The subgroup of 24 patients with conversion symptoms matched for age and sex to the control group is referred to as group H, and the control group is referred to as group C.

Forty-eight percent of group L, 50% of group H, and 58% of group C had some cerebral lesion or disorder affecting cerebral function. The most common of these was epilepsy (23 in group L, 5 in group H, and 8 in group C). Others included multiple sclerosis, cerebrovascular disease, tumor, head injury, encephalitis, and dementia. The high proportion of patients in group C with physical illness is partly explained by the fact that the Department of Psychiatry at the National Hospitals provides psychiatric service to neurological patients. However, this factor does not entirely account for the high proportion of organically ill patients in group L and group H because many of the patients in these groups might have been expected to have been referred for differential diagnosis of somatic symptoms.

During the course of examination of these patients the personality characteristics of each one as well as information about sexual adjustment were recorded. Patients were regarded as having a hysterical personality on the basis of attention-seeking, histrionic, and emotionally labile attitudes, with or without seductiveness. Frigidity or sexual maladjustment were not included in the criteria for hysterical personality because they were being compared with hysterical personality traits. Obsessional personalities were defined as those who adopted meticulous patterns of behavior, usually with minor checking habits. Passive-immature-dependent personalities were defined as those who showed childish, immature attitudes, a lack of independent behavior in social and personal relationships, and overt dependence on others in their family and immediate environment. These gradings were essentially the systematic personal judgments of one of us (H.M.). Other characteristics graded were the anxious, worrying personality type, psychopathic personality, and a variety of mild character deviations.

Sixteen patients showed no evidence of any definite personality deviation; these patients are referred to in the study as "normal." The group labeled hysterical were compared with the subgroups labeled, respectively, obsessional, passive-immature-dependent, and "normal."

The patients' sexual behavior was graded into the five following categories:

Category 1: frigidity includes all patients who said they never fully enjoyed sexual intercourse or that they had refused it despite reasonable opportunity and lack of moral objection.

Category 2: life-long abstention refers to patients over the age of 40 who had lived an inhibited life, having refused opportunities for heterosexual relatic ships, marriage, or both.

Category 3: current problem includes patients wl had been sexually normal but who expressed a curre problem of loss of interest, loss of enjoyment, or imp tence.

Category 4: normal includes unmarried patients u der the age of 40 who were interested in heterosexu contacts, enjoyed sexual intercourse, had hete sexual masturbation fantasies, or dated; it also i cludes married patients who enjoyed intercourse.

Category 5: insufficient information includes p tients for whom relevant information was scanty b cause of their circumstances or who were reticent providing information.

We recognize that these criteria are conservati and may have reduced the percentages of abnormal recorded.

TABLE 1
Personality Types in Groups L, H, and C*

Personality Type	Group L (N=89) N	Group L (N=89) %	Group H (N=24) N	Group H (N=24) %	Group C (N=24 N
Hysterical	17	19	5	21	0
Passive-immature-dependent	17	19	5	21	0
Obsessional	7	8	1	4	7
"Normal"	16	18	3	12	6
Others	32	36	10	42	11

*The difference between groups H and C was significant (χ^2=15.5467; p<.0

TABLE 2
Sexual Behavior Ratings in Groups L, H, and C*

Sexual Behavior Rating	Group L (N=89) N	Group L (N=89) %	Group H (N=24) N	Group H (N=24) %	Group C (N=24 N
Normal**	52	58	13	54	20
Abnormal***	28	32	9	38	3
Insufficient information	9	10	2	8	1

*The difference between groups H and C for abnormal versus normal ratings was significant (Fisher's exact probability test, p=.0487).
**Category 4.
***Categories 1-3.

RESULTS

Of 89 patients with conversion symptoms (group I 16 (17%) were men and 73 (83%) were women. T number of patients with hysterical, obsessional, pa sive-immature-dependent, and "normal" personal styles in group L and in the 24 control patients in gro C are shown in table 1. Results are also given for t subgroup of patients with conversion sympto (group H) compared with the control group.

There was a significant excess of hysterical perso ality and passive-immature-dependent personality the group of patients with conversion hysteria co

BLE 3
rsonality Types and Sexual Adjustment Ratings in Group L*

	Sexual Adjustment Rating					
	Normal**		Abnormal***		Insufficient Information	
rsonality Type	N	%	N	%	N	%
sterical (N=17)	5	29	10	59	2	12
ssive-immature-lependent (N=17)	8	47	8	47	1	6
sessional (N=7)	5	71	2	29	0	
lormal" (N=16)	12	75	3	19	1	6

The difference between hysterical and "normal" personality types was significant (Fisher exact probability test, p=.025). The difference between hysterical and obsessional plus "normal" personality types was also significant (χ^2=6.645; df=1, p=.01). The other comparisons were not significant.
Category 4.
Categories 1-3.

BLE 4
ganic Cerebral Disorders and Personality Types in Group L*

	Organic Disorder Present		Organic Disorder Absent	
rsonality Type	N	%	N	%
sterical (N=17)	6	35	11	65
ssive-immature-lependent (N=17)	7	41	10	59
sessional (N=7)	4	57	3	43
lormal" (N=16)	5	31	11	69

ie differences were not significant.

red with control patients (see table 1). One male and females in group L had hysterical personality (age nge 17–65, mean 36.9), 1 male and 16 females had ssive-immature-dependent personality (age range –56, mean 38.4); and 1 male and 6 females had obssional personalities (age range 15–60, mean 33.4). ven males and 9 females (age range 16–60, mean .9) were considered "normal."

The results of our ratings of the sexual behavior of e total sample are shown in table 2. This table shows e number and percentage of patients reporting nor-al (category 4) and abnormal (categories 1–3) sexual justment. Thirty-two percent of the patients with nversion hysteria had abnormal sexual adjustment. ie difference between groups H and C was signifi-nt (see table 2).

Ratings of sexual behavior in relation to personality yles are shown in table 3. The sexual maladjustment patients in the group with hysterical personality was gnificantly greater than that of patients in the "nor-al" group (see table 3). There was a greater in-dence of reported sexual abnormality in the passive-mature-dependent group than in the "normal" oup, but this difference did not reach significant lev-s. There was also a trend toward greater sexual ab-rmality in the hysterical group compared with the sessional group (59% and 29%, respectively).

In the hysterical personality group 7 of 10 of the patients reporting abnormal sexual adjustment reported life-long frigidity; 2 of 10 were life-long abstainers. In the passive-immature-dependent group 7 of 8 reported life-long frigidity. In both groups there was 1 patient with a current problem. In the obsessional group 1 patient had current problems and 1 reported frigidity. In the "normal" group 1 patient reported life-long frigidity and 2 reported a current problem. The higher incidence of sexual abnormality in patients with conversion hysteria (see table 2) is attributable to frigidity, and this is particularly related to passive-immature-dependent and hysterical personality patterns.

We examined the presence of chronic organic disorders affecting the CNS in relation to both personality style and sexual behavior. Details of the organic illnesses were provided in 1975 by Merskey and Buhrich (18). As already mentioned, the largest number of patients with such disorders had epilepsy. The 23 patients with epilepsy were grouped with another 16 patients who had organic CNS disorders affecting the upper brain stem or higher levels. Twenty-four of these 39 patients reported normal sexual adjustment, and 10 reported abnormal sexual adjustment. Among 40 patients with no organic pathology, 19 had normal sexual adjustment and 17 had abnormal sexual adjustment. The trend for organically sick patients to have better sexual adjustment was not significant. There was also no significant difference between patients with epilepsy and others with organic lesions or between those with definite temporal lobe epilepsy and the rest of the epileptic subjects. The distribution of personality patterns in those with epilepsy was similar to the whole group with conversion symptoms.

Table 4 shows the incidence of organic pathology in the different personality groups. Obsessional patients were more likely than hysterical patients to have an organic lesion, but the numbers are few and the trend is weak.

DISCUSSION

Of 89 patients with conversion symptoms, 19% had a hysterical personality style. This figure approximates that cited by Chodoff and Lyons (3) (3 of 17 of their series) and that of Ljungberg (19), who cited a figure of 21% for his series, a figure that Slater (16) also cited for his patients. Thus, conversion hysteria is not uniformly related to a hysterical personality style. However, our figures show that the incidence of hysterical personality in patients with conversion symptoms is considerably higher than in a control population. This supports the contention that the hysterical personality style is more likely to lead to conversion reactions, although they need not be expected in every patient with hysterical personality.

We have also found an equal incidence of passive-immature-dependent personality in patients with conversion symptoms. This finding and the fact that there is a much smaller proportion of obsessional patients in

the group with conversion symptoms compared with control subjects suggest that patients with conversion symptoms have personality characteristics that differ from those of other psychiatric patients.

Analysis of the sexual adjustment of the patients with conversion reactions indicates that 58% of the total group with conversion symptoms and 54% of the subgroup of these patients reported normal adjustment. This is significantly different from the 83% of normally adjusted patients in the control population of psychiatric patients. The comparative increase in the incidence of abnormal sexual adjustment in conversion hysteria in this series does not appear to be related to the presence of organic factors. It is also far less than that cited by Purtell and associates (8) (86%). With broader criteria we might have found a larger incidence of sexual abnormality, but we believe that the population of Purtell and associates was different from ours and that ours is perhaps the first controlled demonstration of sexual pathology in patients suffering from conversion hysteria. This facet of abnormal behavior is not confined to patients with hysterical personality. Patients with passive-immature-dependent personalities also had a high incidence of sexual maladjustment. Both groups had frigidity as the common abnormality. The characteristics of the passive-immature-dependent personality style are equivalent to those designated "oral" by Lazare and associates (5), who reported that "dependency, passivity, self-doubt, fear of sexuality appear in the oral factor."

The fact that 58% of our patients reported normal sexual adjustment suggests that the etiology of conversion symptoms is not invariably a "substitute for sexual gratification," as would be predicted by Freudian libido theory (20). We would therefore agree to some extent with Chodoff (21) that

> however these disorders are labeled, miscarried sexuality probably does not play the major role in their psychogenesis, for our epoch at least. It appears that failed human needs and strivings other than the sexual ones are responsible for the strange symptoms and desperate lifestyles that have been called hysterical.

Personality disorders and sexual maladjustment remain important correlates of conversion symptoms. In addition, organic lesions were slightly more frequent in patients with conversion symptoms and obsessional personality. Thus, the genesis of conversion symptoms is multifactorial. In settled conditions in developed countries they are infrequent except in certain special settings, e.g., a neurological clinic (18). They will be more likely to occur in association with the hysterical personality, sexual maladjustment, or chronic brain lesions.

The further theoretical significance of these differe causes still remains to be evaluated, although Wh lock (17) has pointed out that no theory of hyste which fails to take into account both psychological a organic causes can be adequate. We can conclude th personality factors, sexual maladjustment, situatio problems, and organic cerebral disease all play su stantial roles that are sometimes complementary a sometimes independent in the production of co version symptoms.

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sychiatric Services in Soviet General Outpatient Clinics

Y BORIS M. SEGAL, M.D.

e author believes that the first level of psychiatric re in the U.S.S.R.—the general polyclinics utpatient clinics) and the medical-sanitary units— ve not been adequately studied. He describes this el of care, which is considered useful and ogressive in that the clinics are supposed to make ychiatric care available to the population, lighten e load placed on psychiatric facilities, and improve llow-up therapy. However, he also points out the ortcomings in this first level of psychiatric care in e U.S.S.R., including the bureaucratic nature of the stem and the uneven quality of service the clinics nder to various social groups in urban and rural eas.

ESTERN WRITERS (1–17) as well as Soviet writers 3–29) have described the concepts, structure, and livery system of psychiatric services in the U.S.S.R. owever, the majority of these works analyzed the acities of psychiatric dispensaries (community mental alth centers) and mental hospitals—the second and ird levels of psychiatric service. The first level of ch medical aid has not yet been adequately studied. According to official Soviet sources (18–29), social- d health care in the U.S.S.R. has two important funcns: the curative-prophylactic and the sanitary-hyenic. The curative-prophylactic aspect of medical aid the responsibility of district and special polyclinics utpatient clinics) and of psychiatric, venereological, ncer, and tuberculosis dispensaries as well as varis kinds of hospitals. There are also medical-sanitary nters (health units) and a special network of medical cilities for children that includes polyclinics, hospils, and consultation clinics. There are also consultan clinics for women.

IMARY CARE FACILITIES

neral Polyclinics

The first level of general medical care in urban areas the district (*raion*) polyclinic (outpatient clinic). In large cities these facilities usually serve approximately 40,000 residents. Service is available only to individuals registered in a given district. Consultation in the polyclinic is available with internists and with such specialists as surgeons, neuropathologists (or psychoneurologists, neurologists with some training in psychiatry), ophthalmologists, and ear specialists. There are also therapy rooms for surgery, physiotherapy, and other forms of treatment. The entire population of a district is divided into smaller districts (*uchastki*), and each of these smaller districts is served by a physician who not only receives patients during appointed hours but who also makes house calls to patients unable to get to a medical office. Such a district in a large city may contain 3,000–4,000 inhabitants. Polyclinic physicians prescribe treatment for patients, send them to a hospital if necessary, care for them after they are discharged from the hospital, arrange for consultations with specialists, sign releases from work, and direct patients to a pension commission.

The task of the polyclinic is not only therapeutic but also preventive. The polyclinic must actively seek patients who need treatment; it must act to prevent illness among the population of its precinct. Once or twice a year it must conduct examinations of patients with suspected or confirmed serious disorders. Most of the polyclinics do not have regularly employed psychiatrists. According to official data, consultations with patients who exhibit signs of such so-called border conditions as neuroses or psychosomatic disorders are conducted by a neuropathologist or psychoneurologist. The local district physician—an internist—and the neuropathologist are supposed to locate mentally ill patients within their area and direct them to the district psychiatric health center (dispensary). Certain large district polyclinics require a psychiatrist from the district psychiatric health center to hold periodic consultations, thus facilitating the discovery of early and mild forms of mental illness.

The larger *raion* polyclinic keeps a file on every patient discharged from a psychiatric hospital, thus enabling the physician in a small district facility to maintain contact with local psychiatrists if necessary.

Other Facilities

The medical-sanitary centers have the same functions as do polyclinics, but their activity is directly connected to a specific enterprise or a factory. When these centers were organized after the revolution, their task was to improve medical services for workers.

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Large medical-sanitary centers have qualified personnel, special hospitals, and sanatoriums at their disposal. Normally, no psychiatrists are employed in these facilities, but large medical sanitary centers have a psychiatric unit, sometimes on a part-time basis. Neuropathologists also work in these facilities, and they are supposed to conduct therapeutic as well as preventive work.

There is also an extensive network of departmental medico-sanitary centers and clinics. Many ministries, the academies, the KGB, and "creative" unions (unions of writers, composers, architects, and others) have special "closed" clinics serving only employees and their families. The many military research institutes or so-called numbered enterprises have their own "closed" medical-sanitary centers and hospitals.

Rural Areas

The first links in the chain of medical psychiatric care in rural areas are the psychoneurologist who works in the district polyclinic, the rural precinct physicians, and the feldshers. Rural physicians or feldshers send patients who have recently become ill or whose condition has become more acute to the district polyclinic or regional psychiatric health outpatient clinic. This facility, along with the regional interdistrict psychiatric hospital, is supposed to provide systematic care, periodically sending patients for consultations with psychatrists in the district. According to official literature (19, 20, 27–29), when a consulting psychiatrist arrives in a rural district he or she studies patients, checks the status of the register, notes corrections that must be introduced in the files of the regional outpatient clinic (e.g., a change in diagnosis), and recommends treatment and work arrangements. The same sort of instructive-consultative work is to be done on a higher level by the psychiatrist in the district polyclinic attached to the office of the psychoneurologist.

Rural district physicians are supposed to do more than just study the condition of the mentally ill and to hospitalize them. They are also to provide follow-up therapy for patients who have been discharged from the hospital that will enable them to return to work. Soviet authors (27–29) have recommended outpatient treatment in rural areas for a large number of patients with epilepsy and organic diseases of the brain, for alcoholics, and for mentally retarded patients. Precinct doctors are supposed to provide care to schizophrenic patients in remission, to patients with residual paranoid ideas, to those with acute but short-term psychotic disorders of a toxic-infectious nature, those with postseizure conditions, and those with an acute alcoholic abstinence syndrome (27–29).

In sum, the official data (18–29) describe a well-organized and universal system of preliminary outpatient care for mental patients in the U.S.S.R. However, the first level of mental health services corresponds to the hierarchical structure of Soviet society rather than to this model.

PROBLEMS IN PRIMARY CARE

The medical facilities for the elite in Soviet soci offer the most favorable conditions. The "close polyclinics for the privileged are well equipped and located in attractive and modern buildings. The sp cialists employed by these establishments are selec with special care. The number of patients in these c ics is much smaller than in the general polyclinics, the attending physician and psychoneurologist (or p chiatrist) spends a considerable amount of time exa ining patients. Preventive and psychotherapeuti work in these institutions corresponds to the model scribed above. Patients are regularly reexamined ev if they do not request medical care. Consultations available with prominent specialists. Patients in ne of additional care are directed to research institutio departmental sanatoriums, rest homes, and spec hospitals.

The higher party and government officials are serv by polyclinics of the special administration of the M istry of Health. These closely guarded "Kremli polyclinics enjoy the most privileged position. The s aries of the physicians and staff in these facilities 50% above the norm, and the staff are given a numb of privileges. Their relations with the patients strictly controlled by the administration. Patients offered all of the modern methods of examination a treatment and all the services of a family doctor.

Large cities also have an extensive network of f and accessible district polyclinics and medical-sa tary centers. In general, this system provides the ur population with more or less adequate medical se ice. The main shortcoming of the system—one tha peculiar to all medical care in the U.S.S.R.—is the for bureaucratic nature of the work. The psych neurologist, like the physician in a polyclinic, is ti and dissatisfied with overwork and extremely low p (the general salary for this work is approximately 1 150 rubles or $130–$200 a month). The physic spends a major part of his or her workday filling various documents; almost no time is left for exam ing and talking with patients.

Patients correctly complain that appointments in polyclinic often result in nothing more than receivin work release form. The atmosphere of polyclinics usually tense. There are long lines of people waiting see doctors. It is very difficult to meet with hig qualified psychiatrists. Many patients try to rece private treatment even though this is officially forb den.

Local psychoneurologists in small districts are in especially difficult position; they view their work exhausting and thankless. They do not provide p ventive treatment, but they make house calls. C sultations with psychiatrists in district polyclinics rare; there is little or no prophylactic or therapeu work conducted among patients with neuroses or w psychosomatic and sexual disorders, although su patients account for a substantial number of appo

ents. These patients receive only mild tranquilizers d are advised to "not be nervous." In fact, there are) facilities for such patients. They are usually not nt to psychiatric outpatient clinics because they are)t sick enough. They are told, "They're crazy there. 'hat do you want to go there for?" If those seeking :lp do manage to get an appointment with a psychia- .st, most frequently the result is just a short chat and prescription for tranquilizers. For patients with more rious neurotic disorders there are psychotherapy oups in some psychiatric dispensaries. Psychotic pa- nts are sent to psychiatric dispensaries.

The availability of psychiatric service in small cities d especially in villages presents a problem that is far om being solved. There are usually one or two poly- nics in small cities but no psychiatrist and no neuro- thologist. To obtain an appointment with these spe- alists one usually has to travel some distance to the gional center, which is often located several hundred lometers away. Because of poor travel connections, lack of good roads (especially in the spring and fall hen weather conditions make it almost impossible to t anywhere), and the lack of privately owned autos, e journey to a medical center can be very difficult. elatives of patients with psychomotor agitation en- unter the most distressing problems. Families are ten forced to make great financial sacrifices to obtain car and get the patient to the regional center. Villages nerally have no ambulances for psychiatric cases, d the police refuse to transport the mentally ill.

Doctors do not accept patients from other regions ecause medical practice in the U.S.S.R. requires at patients be treated according to their place of sidence, that is, where they are registered by the po- ce. Unless they have made previous agreements with eir superiors, local doctors are categorically for- dden to issue their patients permits to travel to rge cities where there are more qualified psychia- ists and where there is usually a better organized stem of health service. In spite of all these bar- ers many patients travel without permits to Moscow, eningrad, Kiev, and other large cities. On arriving in ny one of these centers, they are sent to the Ministry f Health and to other official departments, where they ceive assignments with recommendations that they e examined and treated according to their place of sidence. Only in certain instances does the Ministry ermit a patient from the provinces to be examined in n outpatient clinic of the capital, and very rarely is ch a patient hospitalized in a big city. This usually ppens only if the patient's relatives have made sev- al complaints to higher officials and Party depart- ents. Sometimes, in desperation, patients and their elatives refuse to travel to their place of residence. hey demand justice, creating a public disturbance to ring attention to their case.

Thus, complex bureaucratic obstacles hamper the rst level of mental service in the U.S.S.R. An extensive stem of barriers prevents the population from taking dvantage of socialized medical care. The many mil- ions of rural inhabitants receive the least favorable treatment and are in the most difficult position. (One must take into consideration that in 1970 44% of the Soviet population lived in rural areas; an additional 47,500,000 people lived in small cities.) In addition, the supply of medicine, which is generally poor throughout the U.S.S.R., is especially inadequate in rural pharmacies, where there are no psychopharmacological remedies. All these problems create a dissatisfaction with the system of medical care—especially among the lower strata of the population.

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The *Journal* will continue this year its policy of early review of annual meeting papers. Final versions of papers that are ready before the Chicago meeting will be accepted for review. Do *not* submit your paper early if you anticipate substantive revisions after discussion at the meeting. Manuscripts should be submitted in triplicate, double-spaced throughout, and prepared in conformance with *Journal* style specifications, including length limitations. Papers written in "speech" format should not be submitted.

Please label the paper "Annual Meeting 1979, Final Version" and include on the title page the address and phone number of the corresponding author and the number of words, tables, and figures. The cover letter should incorporate the copyright transfer statement described in "Information for Contributors."

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If your paper will not be ready for review before the meeting but will be in final form at that time, bring three copies to Chicago and submit them to the session Secretary (submitting copies to the Press Office *does not* satisfy this requirement). Again, papers that are not ready for review should not be given to the Secretary.

If you have questions about this or other *Journal* policies, please stop by the *American Journal of Psychiatry* staff office during the meeting, and staff members will be pleased to assist you.

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RIEF COMMUNICATIONS

orazepam, a New Benzodiazepine Derivative, in the Treatment f Anxiety: A Double-Blind Clinical Evaluation

C LAYTON MCCURDY, M.D.

nety-five adult outpatients suffering from moderate severe anxiety were randomly assigned to treatment th lorazepam or placebo in a double-blind four-week dy. Patients were evaluated at pretreatment and er two and four weeks of treatment using three ing scales. Lorazepam, at an average daily dose of mg twice daily, was highly effective in relieving xiety as documented by clinically and statistically nificant differences over placebo in most of the ms on all rating scales at all rating periods. No rious adverse effects were reported, and there were adverse interactions with other medications, cluding digitalis and aspirin.

HERE HAS LONG been public and medical concern th the alleviation of excessive anxiety. This interest s resulted in extensive research to determine the uses of anxiety, design methods for evaluating it, d develop therapies for its treatment. Drugs, in par- ular the benzodiazepine derivatives, have proven to a successful therapeutic modality. These com- unds have been under continuing development and finement since the 1960s (1).

Lorazepam, or 7-chloro-5-(*o*-chlorophenyl)-1,3-di- dro-3-hydroxy-2H-1,4-benzodiazepin-2-one, one of e newer benzodiazepine compounds, is generally ell tolerated, has a low biological toxicity (2), and oduces few side effects (3–5). On a weight basis, lorazepam is a more active anxiolytic than diazepam (1 mg versus 5 mg, respectively). While this drug is relatively new to the United States, it has been widely used in other parts of the world, particularly Europe.

The following double-blind study was conducted to assess the efficacy and safety of lorazepam in treating anxiety.

METHOD

Ninety-five patients, 35 men and 60 women ranging in age from 18 to 65 years, participated in the study. These patients were referred from a community mental health center and an Alcoholics Anonymous group. They represented a broad range of socioeconomic backgrounds.

Selection criteria required that all patients have ratings of at least moderately severe anxiety as well as three related symptoms on the Hamilton Anxiety Scale (6). They were also required to have a global rating of at least moderate illness. Patients excluded from the study consisted of those with signs of psychosis or a history of psychosis, acute or chronic brain syndrome, mental deficiency, acute reactive or involutional depression, suicidal tendencies, and drug addiction. Patients with a sensitivity to benzodiazepines were also excluded, as were pregnant patients and those with a history of myocardial infarction within 3 months of the study. Patients suffering from alcoholism were included in the study if they met the above criteria and had been sober for 60 days. Informed consent was obtained before patients were accepted into the study group. Patients were permitted no psychoactive medication for at least 4 days before the study began.

The study design was fully randomized and was double-blind. The total duration of the study was 4 weeks; patients were evaluated at pretreatment and after 2 and 4 weeks of treatment. Patients received either

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1-mg scored tablets of lorazepam or matching placebo, administered on a twice-daily schedule. All but 1 patient started on a regimen of 3 tablets per day, 1 in the morning and 2 at bedtime. If there was a need to reduce the dosage, the morning dose was eliminated first. After the first follow-up visit (at 2 weeks), the daily dosage was continued, reduced, or increased to a maximum of 6 tablets per day, maintaining the twice-daily regimen.

The 95 participants were randomly divided into 2 groups. Forty-seven patients received lorazepam and 48 received placebo. Efficacy and safety were evaluated after 2 and 4 weeks of treatment and at a final evaluation, which by protocol was defined as the last evaluation for any patient who participated in the study for at least 4 days. Although 16 patients (5 in the lorazepam group and 11 in the placebo group) failed to complete the full 4-week course of treatment, 87 (43 in the lorazepam group and 44 in the placebo group) were available for final evaluation.

The efficacy of medication was measured using a global rating scale, the physician-administered Hamilton Anxiety Rating Scale (6), and the self-administered Lipman-Rickels 35-Item Self-Rating Scale (7). The global assessment of the severity of illness used a 4-point scale, while the degree of change since the start of the study was judged on a 7-point scale. The Hamilton Anxiety Rating Scale measured drug effectiveness through changes in total scores in 13 symptom categories, and in psychic and somatic clusters. Intensity of symptoms was rated on a 5-point scale (1=absent, 2=mild, 3=moderate, 4=severe, and 5=very severe). The Lipman-Rickels scale was used to determine therapeutic effectiveness.

At each visit patients were asked whether there were any side effects and, if so, their nature and intensity. In order to avoid prompting the patients, the questions were asked without resort to a checklist. All adverse side effects were recorded regardless of the patient's ultimate inclusion in or exclusion from the efficacy evaluation.

One-way analysis of variance, chi-square test of independence, and Fisher exact probability test were used to determine whether the 2 treatment groups were initially balanced and homogeneous in terms of severity of illness and demographic characteristics. Because of their subjective, ordinal nature the rating scale data were evaluated by means of nonparametric statistical tests. The Kruskal-Wallis test was applied to scores from all three rating scales to compare drug efficacy between treatment groups. Fisher's exact probability test was applied to changes in the global ratings after pretreatment to detect differences between groups.

RESULTS

There were no significant differences between the 2 treatment groups in age, weight, race, sex, duration of illness, or initial severity of illness on the global, Hamilton, and self-rating scales. Four patients from each group were excluded from the final efficacy evaluations because rating scale data were incomplete or not obtained, leaving 43 in the lorazepam group and 44 in the placebo group available for final evaluation.

Dosage

Total daily dosages of lorazepam ranged from 2 to 6 mg, with a mean of 3.2 mg; the mean treatment period was 26.5 days. Dosages in the placebo group ranged from 3 to 6 tablets per day, with a mean of 3.4; the mean treatment period was 24.6 days. At the end of 2 weeks 38% of the patients were receiving an increased dosage of lorazepam (i.e., over 3 mg per day), and 51% required an increased dosage of placebo.

Clinical Efficacy Ratios

According to published standards for evaluating psychotropic drugs (8), a drug can be regarded as demonstrating clinically significant efficacy if the percentage of patients improved with the drug divided by the percentage improved with placebo is at least 1.5. Clinical efficacy ratios may be determined on the basis of the percent of patients improved, percent reduction in mean scores on rating scales, or both. Both types of ratios were computed in this study.

Table 1 presents the overall improvement rates (percent of patients improved) for the three rating scales at the final evaluation. There was a statistically significant difference between lorazepam and placebo on all three scales. The ratios for patient improvement were similar to the ratios for percent reduction in mean scores on rating scales. The results of both methods for measuring improvement therefore satisfied the efficacy criteria outlined above.

Global Rating Scale

Intergroup analysis (Kruskal-Wallis test) indicated that lorazepam was significantly more active than placebo at all evaluation periods. Improvement in global severity with lorazepam was significant at 2 and 4 weeks ($p<.05$).

Hamilton Anxiety Rating Scale

Analysis between groups showed that lorazepam was significantly superior to placebo in the following assessments at 2 weeks and the final evaluation: anxious mood, tension, insomnia, genitourinary symptoms, behavior at the interview, psychic cluster, and total Hamilton score. At week 4 lorazepam was significantly superior to placebo in the assessments of anxious mood, psychic cluster, and total Hamilton score. Table 2 presents the scores at pretreatment and at 2 and 4 weeks for key items and clusters of the Hamilton Anxiety Rating Scale; the greater reductions with lorazepam are apparent.

BLE 1
rcent of Patients Improved and Clinical Efficacy Ratios at the Final
aluation

m	Global Rating Scale	Hamilton Total Score	35-Item Scale Mean Item Score
rcent of patients improved			
Lorazepam	93*	72**	65*
Placebo	52	46	30
nical efficiency ratio	1.8	1.6	2.2

<.01, Fisher exact probability test.
<.05.

BLE 2
an Scores at Pretreatment, 2 Weeks, 4 Weeks, and Final Evalua-
n for Key Items and Clusters of the Hamilton Anxiety Rating Scale

m	Pretreatment	Week 2	Week 4	Final Evaluation
xious mood				
Lorazepam	3.26	2.60*	2.29*	2.31*
Placebo	3.11	2.84	2.57	2.64
nsion				
Lorazepam	3.28	2.51*	2.15	2.19**
Placebo	3.23	2.82	2.43	2.52
chic cluster				
Lorazepam	19.63	15.56*	13.76**	13.91*
Placebo	18.77	16.98	14.95	15.34
tal score				
Lorazepam	31.91	25.93*	22.81**	22.96*
Placebo	30.93	27.66	24.38	25.14

<.01 (Kruskal-Wallis analysis).
<.05.

Item Self-Rating Scale

Intergroup analysis (Kruskal-Wallis test) of the ans of clusters, factors, and mean 35-item scores owed significant differences in favor of lorazepam at time periods for somatization, anxiety cluster, and an item score (table 3), and at week 4 and the final aluation for interpersonal sensitivity, anger-hostili- , and general neurotic factor.

fety of Medication

Nine patients reported side effects from medica- n—7 in the lorazepam group and 2 in the placebo oup. In the lorazepam group 4 patients (8.5%) re- rted sedation; 2 (4.3%), psychomotor agitation and itability; 2, nausea; 1 (2.1%), insomia; and 1, urticar-

One of the placebo patients (2.1%) reported psy- omotor agitation and irritability; 1 had urticaria; and eported shortness of breath. Two of the 7 lorazepam tients who reported adverse effects discontinued atment as a result (1 patient had repeated dizziness, rred vision, nausea, and irritability; the other had icaria). The symptoms of the 5 remaining lorazepam tients either disappeared with continued therapy or re controlled with dosage adjustment. In the pla- bo group 1 patient tolerated the symptoms (psycho- tor agitation and shortness of breath), and the oth- , who had urticaria, discontinued treatment.

TABLE 3
Mean Scores at Pretreatment, Week 2, Week 4, and Final Evaluation for Key Items and Clusters of the 35-Item Self-Rating Scale

Item	Pretreatment	Week 2	Week 4	Final Evaluation
Somatization				
Lorazepam	2.04	1.73*	1.53*	1.55*
Placebo	1.97	1.89	1.79	1.83
Anxiety cluster				
Lorazepam	2.38	1.98**	1.69**	1.72*
Placebo	2.38	2.18	2.03	2.03
Mean 35-item score				
Lorazepam	2.27	1.93**	1.67**	1.70*
Placebo	2.32	2.12	1.95	2.01

*p<.01 (Kruskal-Wallis analysis).
**p<.05.

Five patients in the lorazepam group (10.6%) and 11 in the placebo group (22.5%) discontinued treatment. As previously mentioned, 2 patients taking lorazepam discontinued because of adverse effects. The remaining 3 failed to return to continue the study. All 5 of these patients were receiving 3 mg per day at that time. One of the patients in the placebo group discontinued therapy because of adverse effects, and 3 discontinued because of unsatisfactory response to treatment. The remaining 7 dropped out for reasons unrelated to treatment. The dropout rates of the 2 treatment groups did not differ significantly.

Only 6 patients received concomitant medications during the study; 5 of these medications were permitted by protocol (conjugated estrogens, digitalis, norethindrone/mestranol, aspirin, and ethynodiol diacetate/mestranol). One patient received a narcotic-containing mixture, unacceptable by protocol criteria, and was therefore dropped from efficacy evaluation.

DISCUSSION

The superiority of the benzodiazepine class of compounds over its predecessors for the treatment of anxiety is well known. In the present study the relatively small amounts of lorazepam that were used (an average total daily dosage of 3.2 mg on a twice-daily regimen) were much more effective in treating anxiety than the usual doses of earlier benzodiazepines. The need to take medication only twice daily—in the morning and at night—was an obvious convenience to the patients.

The results of this 4-week double-blind study of 95 psychoneurotic patients indicate that lorazepam given twice daily in a mean dose of approximately 3.2 mg per day is significantly more effective than placebo in relieving the great majority of symptoms associated with anxiety. This conclusion is based on intergroup analysis of all 3 rating scales (2 physician-rated and 1 patient-rated scale) at 2 and 4 weeks and the final evaluation. No serious side effects were reported. In addition, there were no adverse interactions between

lorazepam and acceptable concomitant medications including aspirin, digitalis, and hormonal preparations.

Lorazepam is rapidly absorbed and has no psychoactive metabolites. The half-life permits a flexible dosage regimen and rapid attainment of steady-state blood levels, following which there is no accumulation of the active drug (9). Patient acceptability was high with this drug. It appears that lorazepam is an effective, safe, and acceptable antianxiety medication.

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Childbirth-Related Psychosis and Familial Symbiotic Conflict

BY RICHARD M. KETAI, M.D., AND MARVIN A. BRANDWIN, PH.D.

The authors present 3 cases of childbirth-related psychosis in young women to illustrate an interpersonal psychodynamic pattern that may contribute to such reactions. In each instance the woman is the submissive partner in a symbiotic relationship with her husband and/or mother. The baby's presence threatens the symbiotic equilibrium of these relationships and leads to the dominant partner's overt or covert aggression against the woman, which precipitates the psychosis. A personal or family history of psychosis may increase the woman's vulnerability to becoming psychotic in this situation.

CHILDBIRTH-RELATED PSYCHOSES are usually considered to be acute manifestations of underlying schizophrenic or manic-depressive illness (1), unique mainly in their temporal association with pregnancy and childbirth. They are similar to psychotic episodes occurring at other times in that biological and psychological p dispositions interact with situational stresses to det mine the specific form and timing of the reaction.

Studies of childbirth-related psychoses have cused on the biological and psychological vulnerab ities of the mother. The pathological effects of stress experienced by the new or expectant father have al received attention. Little consideration has been g en, however, to the role of interpersonal family d namics as a precipitant of these psychoses. The f lowing three case histories of patients recently seen our inpatient unit illustrate a pattern of marital or fa ily interaction that may, we believe, significantly i crease the woman's vulnerability to psychotic rea tion.

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CASE REPORTS

Case 1. Ms. A, a 29-year-old graduate student who h been married 5 years, became symptomatic 1 day after t birth of her first child. The night before delivery her husba confessed to having had an affair during the pregnancy. Af delivery Ms. A became agitated, sleepless, aggressively demanding, and suspicious. She accused the nursing and me cal staff of plotting to harm her baby, who she now believ had God-like powers. Her acute disturbance and inability care for her baby necessitated psychiatric hospitalizati Initially a difficult management problem because of aggr sively manipulative manic behavior, she responded well

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eatment with chlorpromazine and lithium. The affective na-re of her postpartum psychosis was consistent with a fam- history of mania.

Ms. A and her husband were an intensely symbiotic uple. The marriage was marked by extreme togetherness, reflected in Mr. A's statement, "We spend almost 24 urs a day with each other, including our classes; we have ver been separated." During the hospitalization they mir-red each other's attitudes to an extreme degree, maintain- a hostile united front against the hospital staff. Closer servation, however, revealed a competitiveness tinged th aggression underlying their intense mutual dependency. r. A particularly tended to be critical and demeaning when s own self-esteem was threatened. He remained unable to mprehend that his confession of adultery might have been ensitive. In contrast, during treatment Ms. A commented t she had been deeply hurt by his statement, especially h regard to its timing.

Case 2. Ms. B, a 26-year-old woman in her fifth month of gnancy, was admitted for her third psychiatric hospitali-tion after she became depressed and delusional. After an sode of auditory hallucinations and mild paranoid delu-ns when she was 17, Ms. B functioned adequately until delivery of her first child after 2 years of marriage when was 24. The marriage had been opposed by Ms. B's ther, who had an intrusively close relationship with her ghter and a mutually antagonistic relationship with her n-in-law. The marital interaction was marked by extreme erdependence but marred by increasing arguments during pregnancy.

Three weeks postpartum Ms. B came to the emergency om in an agitated state, believing her baby was controlled the devil. Her husband strongly opposed hospitalization, wing any separation as a threat to the marriage. During ensuing 3 weeks their discord escalated, however, and spite the increasing severity of Ms. B's disturbance Mr. B ted that he was considering leaving the marriage. One ening Ms. B expressed fears of hurting the baby, but her sband left to stay overnight with friends. The next day she pressed the same fears whereupon he left home on an er-d. While he was away she strangled the child and at-mpted suicide by drug overdose. Ms. B's baby died, but survived and was committed to a state hospital where remained for a year. Two months after discharge she came pregnant again by Mr. B.

Despite the similarity of Ms. B's current psychotic symp-ms to those of the previous episode, her husband once in resisted inpatient treatment efforts. Shortly after her nission Mr. B persuaded her to sign out against medical vice, stating that he felt that he would "vanish" without . She became progressively more psychotic and had to be dmitted because of intense suicidal impulses. Throughout ther hospitalization Mr. B opposed the treatment pro-m. Ms. B vacillated between responsiveness to the ward ieu and withdrawal from staff interactions in collusion h her husband's attitudes. However, she responded well nedication and, with concerted staff support, had an un-ntful delivery and postdelivery period.

Case 3. Ms. C was a 21-year-old woman who was admitted eeks postpartum because of an acute psychosis that be- 3 days after delivery. According to her mother, Ms. C been told by her husband in the delivery room that he venereal disease. She then became progressively dis-turbed, showing sleep and eating disturbance, confusion, fear of dying, ideas of reference, and delusional thoughts of her baby being Christ or the devil. This was her second psychiatric hospitalization, the first occurring when she was 17 for what was described in the record as a hysterical psychosis. Ms. C attributed her illness to her struggle "to get away from my mom." Her developmental history provided clear evidence of an intensely ambivalent symbiotic bond with her mother. After discharge she functioned marginally as a "street person" until, despite strong parental disapproval, she married an itinerant rock-and-roll musician who she felt would "rescue" her. Before the marriage, to "prove" her love to him, Ms. C severely stabbed herself in the abdomen. Her parents accused Mr. C of the stabbing, citing the incident as evidence of his being a dangerous person and as justification for their persistent efforts to break up the marriage. Ms. C vacillated between a protective symbiotic involvement with her husband and a more ambivalent symbiotic bond with her mother. During much of the marriage she lived with her parents rather than with her husband, including the final 4 months of pregnancy.

Upon admission Ms. C was agitated, was behaving bizarrely, and was having visual and auditory hallucinations. She expressed fears that her husband would harm her or that he and the baby were in danger. Improvement was gradual and marked by sudden regressive shifts, often precipitated by visits from her mother, who was intrusive and controlling of both the patient and ward staff. Ms. C's mother considered her unfit for motherhood and sought to adopt the baby. She accused her son-in-law of being a danger to her daughter, a concern reflected in the patient's own delusional preoccupations. For therapeutic reasons it was necessary to restrict the mother's visits. In contrast, Mr. C seldom visited, broke scheduled appointments, and became increasingly more difficult for the patient or staff to contact.

DISCUSSION

Pregnancy and childbirth challenge both wife and husband not only with new realistic concerns but with previously unresolved intrapsychic and interpersonal issues rekindled by expectant parenthood. For some this stress may have positive consequences by serving to promote more mature resolutions of conflict. For others the hazards are greater than the available coping resources and regression ensues.

From 2% to 8% of female admissions to psychiatric hospitals are for postpartum illnesses (1). Various psychogenic contributions that have been implicated include the woman's intolerance of the nurturant demands of the child because of her own unfulfilled narcissistic or oral dependency needs, rejection of the mothering role because of inadequate or hostile identification with her mother, reactivation of unconscious psychic conflicts such as oedipal or sibling rivalries, and weakness of personality integration leading to ego identity diffusion in reaction to pregnancy-induced bodily changes (1–3). The pathogenic effects of such psychodynamic factors are compounded by the somatic-physiological changes of pregnancy and her genetic-constitutional predispositions to psychiatric illness.

Studies of men with psychopathologic reactions as-

sociated with new or expectant fatherhood suggest a number of dynamic factors as significant precipitants (4–8). These include increased financial responsibilities, triggering of latent homosexual conflicts by restriction of heterosexual opportunities during the prenatal period, rearousal of unacceptable childhood anger toward parents or siblings, reactivation of unresolved oedipal conflict, and frustration of dependency demands as the wife's attention is diverted to the baby's needs. Towne and Afterman (5) found a characteristic personality trait of chronic, primitive dependency needs among men with psychoses related to parenthood. These men sensed the child as a rival for dependency gratification. Inevitably, their own dependency demands could not be met, and they frequently responded aggressively and destructively toward their wives during and after pregnancy. Even among less disturbed men pregnancy-related stresses may arouse irritability, neglect, belittling, or other forms of aggression. For the psychotically predisposed wife already struggling with motherhood conflicts the husband's abuse or nonsupport may well tip the balance toward psychotic decompensation.

We believe that the presence of a mutual symbiosis with the husband or a parent (usually the mother) increases the woman's vulnerability to postpartum psychosis. The need for symbiotic attachment implies failure of developmental tasks of individuation-separation and establishment of a stable ego identity. These are the core problem areas likely to be stimulated by the psychological and biological pressures of pregnancy.

As the symbiotic relationship may itself be a defense against conflict awareness, the need to resist disruption of a primary defensive structure may provoke an exaggerated reaction to the threat of dissolution of symbiotic homeostasis. This seems applicable to Mr. A and Mr. B. Mr. B's fear that he would "vanish" if separated from his wife reflects his dependence on the symbiotic bond in preserving his own personality integration. In each instance the husband's aggressive behaviors, which were, paradoxically, communication of his need to maintain the prepregnancy marital status, forced the wife into the dilemma of choosing between husband and child. This bind, especially difficult because of the wife's own symbiotic needs and related conflicts, was crucial in precipitating the psychotic reactions in both women, and in one instance proved fatal to the child.

In the third case the main symbiotic bond was between Ms. C and her mother. Ms. C's effort to break this ambivalent attachment by marriage was successfully thwarted by her mother's open and effective resistance, especially when Ms. C became pregnant. The pregnancy was probably perceived by Ms. C's mother as a threat in that it strengthened the husband's position. Her hostility toward Mr. C can also be considered as aggression toward her daughter, who was a pawn in the struggle between her husband and her mother. After childbirth the baby, too, became a pawn. Mr. C's reaction, which expressed both self-protective and hostile motives, was a gradual wit drawal of support from his wife.

All three cases illustrate a common pattern: 1) pre ence of a symbiotic relationship, with the pregna woman as the submissive partner, 2) experience of t pregnancy by the dominant partner as a threat to t symbiotic equilibrium, 3) communication of the pa ner's distress by overt or covert aggression against t woman, and 4) precipitation of a psychotic reaction the woman related to intensification of her own un solved individuation-separation conflicts. In each i stance acute decompensation occurred immediate following an explicit hostile act.

Similar marital dynamics have been observed Martin (9) as potentiating hysterical psychoses times of stress in women with oral-narcissistic, hy terical characters. He relates these reactions to the tachment to the hated but need-satisfying husba when he becomes more destructively unsupporti during family crises. The etiologic role of chron overtly hostile marital interaction in childbirth-relat psychotic reactions of women with primitive, narc sistic personality organization has also been not (10). Specific to our cases is the pseudoharmonious r ture of the marital bond until disrupted by pregnanc The women acknowledged their husband's hostili by direct complaint or in delusional content, or while psychotic. As the psychosis cleared they we resistant to discussion of marital discord, as if attem ing to re-establish the previous protective symbio bond. The symbiotic partner in these cases remain unable to recognize any of his or her contribution the woman's illness.

In this paper we have focused on the role of sy biotic family conflict as a determinant of postpartu psychotic reactions. Consideration of such marital a family dynamics may have significant prophylac value. We recommend that prompt supportive int vention be made when the marital situation shows presence of high-risk circumstances for the pregn woman. Such concern is indicated when the woman involved in an excessively interdependent but cover ambivalent marital relationship, is subject to increas hostile behavior or loss of support from a husband d tressed by the pregnancy, and has a personal or fam history of psychotic illness.

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Required Educational Objectives in Community Psychiatry

Y JAMES H. SHORE, M.D., J. DAVID KINZIE, M.D., AND JOSEPH D. BLOOM, M.D.

ommunity psychiatry is an essential requirement of pproved residency training. The authors present a rmat of required educational objectives for this perience. They feel that recent attempts to limit sychiatric consultation to the consultation-liaison odel are restrictive and incomplete. In their view of e psychiatrist's role, community consultation chniques are essential skills for the psychiatric ractitioner.

DEBATE CONTINUES in psychiatric training concerning educational priorities for community psychiatry. This debate has focused on the content of the residency training curriculum, the impact of the community mental health movement on psychiatric practice, and the renewed identity of psychiatrists with the medical model. In a widely quoted article on the educational objectives of a psychiatric residency program, Yager and Pasnau (1) did not mention the inclusion of community psychiatry training. In a recent discussion of psychiatric consultation, Lipowski (2) argues that the preparation of psychiatrists for the consultant role should emphasize a consultation-liaison model instead of community mental health consultation.

The current edition of the *Directory of Accredited Residencies* (3) outlines the essentials of an approved residency:

The curriculum must include sufficient material from the social and behavioral sciences (such as psychology, anthropology, sociology) to help the resident understand the importance of economic, ethnic, social and cultural factors in mental illness. The clinical portion of the curriculum must provide experience in both community and forensic psychiatry. The clinical training in community psychiatry should include experience in a community mental health center such as a school, court, or police department. Training in forensic psychiatry must involve more than solely didactic instruction. It should include supervised clinical experiences such as consultative work with judges, attorneys, police, probation officers, and other professionals in the legal field, and, whenever possible, actual experience in court room testimony.

This requirement is reinforced by criteria developed by the Psychiatric Education Branch at NIMH for evaluating psychiatric residency programs (4). Their guidelines for site visitors encourage the program evaluators to give attention to a balanced curriculum that should include core areas which cover the principles of community mental health and the practice of consultation work.

EDUCATIONAL OBJECTIVES

This report presents the formal educational objectives in community psychiatry that have been developed since 1973 at the University of Oregon Medical School for a community psychiatry training program. Community psychiatry is a half-time, six-month experience in the third postgraduate year, plus a six-month fourth year elective. Each resident must have met the knowledge and skill requirements of the previous two years' educational objectives. Direct patient contact in diagnosis and treatment of major mental illnesses are the precommunity training goals after a medical internship has been completed. The curriculum in community psychiatry includes all activities of indirect services that would be performed by a psychiatrist in a

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community setting. These involve consultation, interdisciplinary team participation, administration, community educational tasks, supervision of coprofessionals, and forensic psychiatry.

By the end of the resident's rotation in community psychiatry, we expect him or her to be able to perform the following tasks: 1) to enter a community mental health delivery system with a clear understanding of the psychiatrist's role; 2) to negotiate a consultation contract; 3) to work effectively as an interdisciplinary team member in a community mental health program for direct patient services; 4) to work effectively as an interdisciplinary team member in a community mental health program for indirect service or mental health consultation; 5) to conduct a mental health consultation with a community agency; 6) to initiate basic mental health program planning strategies; 7) to work through a process of consultation termination; 8) to evaluate his own role in a community setting with regard to its indirect versus direct service aspects and to demonstrate the use of different models of consultation; and 9) to conduct a precommitment evaluation and court examinations under Oregon's new commitment statute.

By the end of the rotation in community psychiatry, we expect the resident to demonstrate knowledge in the following areas: 1) history of the community mental health movement; 2) basic concepts in social, transcultural psychiatry, and psychiatric epidemiology, including the service delivery system to specific ethnic groups and disadvantaged minorities; 3) general principles of crisis intervention, other types of intervention, and methods of prevention; 4) the role of social, cultural, and family stress in mental health and adaptation; 5) the theory and practice of different models of consultation and the ability to identify the characteristics of direct and indirect service; 6) the structure of community mental health delivery systems in Oregon; and 7) the general principles of forensic psychiatry with particular focus on the Oregon commitment process.

The field experience and the supervision are designed to meet skill objectives. The content of the community psychiatry seminar, designed to meet knowledge objectives, includes the following areas: 1) an introduction to the basic concepts of community psychiatry, historical review, epidemiology, concepts of prevention, and interdisciplinary relationships; 2) the consultation process, describing the models and techniques of community mental health consultation; and 3) specific categories in community psychiatry, program development, program evaluation, administrative psychiatry, and forensic psychiatry.

Evaluation of the educational objectives will be assessed by a formal report both by the community site supervisor and faculty supervisor. In addition, psychiatric residents will be given a mini-Board examination at the end of their third year of community psychiatry rotation, which will include written examination following the ABPN National Board format and a one-hour oral examination. The oral examination will involve two examiners—a psychiatrist and a nonphysician mental health professional. This will evaluate the trainee's ability to apply his knowledge to the implementation of the consultant's role and his understanding of interdisciplinary community mental health issues.

DISCUSSION

Although no set of educational objectives in community psychiatry would be acceptable to all training programs, those we have presented here represent a broad focus that includes principles of public health, the consultation process, the importance of interdisciplinary partnerships, and sociocultural determinants of behavior. The inclusion of forensic psychiatry in the community curriculum seems appropriate since most residency programs will be unable to develop a separate educational program for forensic psychiatry and since most psychiatric activities that involve law take place in community settings.

Oregon's program (5) was planned jointly by the Oregon Mental Health Division and the University of Oregon School of Medicine. The organizational structure reflects an emphasis on state-wide training opportunities throughout the community mental health delivery system. Five distinct groups worked in a collaborative effort to develop a proposal for community psychiatry training. These groups include the Oregon Mental Health Division, Oregon State Hospital, community mental health center directors, and medical school administration, and department of psychiatry faculty of the University of Oregon School of Medicine, and it was felt that a diverse representation of mental health interests would assure state-wide participation, relevant priorities for training placement, and continued community support. More than 40 potential training sites have been developed throughout the state; 24 are located in Oregon's county-state mental health programs, and 5 additional sites are in transcultural settings that involve training in mental health programs for American Indians, Chicanos, or blacks. Senior residents may elect to train in alcohol and drug center mental health education, or in administrative and forensic psychiatry.

Categorical funding from NIMH for postresidency community training (6) is no longer available, and most residency programs must turn to state and county partnerships for appropriate training liaisons. Numerous models of these partnerships have recently been described (7, 8). Fink (9) has described a community psychiatry curriculum in Virginia in association with a single community mental health center and subsequent problems of resident response to the community placement. In Arizona Beigel and associates (10) offer senior residents an opportunity to participate as ex officio community board members. In Massachusetts Sabin and Sharfstein (11) developed a communi

aining program in a psychiatric hospital that had be-ome a community mental health center with responsi-lity for catchment area services.

Psychiatric educators face a special challenge in acing residents in community settings. The identity isis and culture shock (12–14) encountered by many sychiatric residents working in community centers ay reinforce maladaptive habits, a defensive reliance n the medical model, and a negative reaction for par-ipation in any nondirect service community mental alth activity. To facilitate the resident's adjustment Oregon, the program director emphasized clear ructuring of the training situation and continual sistance from supervisors in the conceptualiza-n of the community psychiatrist's role. Supervisors ressed the doctor's unique role as a member of an terdisciplinary health team with a patient-centered cus. Supervision was scheduled weekly with both mmunity program staff and the department's pro-am director. This met the frequent criticism from sidents who in previous community experiences had mplained of poorly structured and inadequately su-rvised training.

Zwerling (15) recently reviewed the impact of the mmunity mental health movement on psychiatric actice and training. He discussed three basic con-pts that will have a profound impact on traditional eory and practice. These concepts must be taken in-account in curriculum design for psychiatric resi-ncy programs—catchment area planning, programs r prevention, and understanding of behavior that in-ıdes multiple sociocultural determinants. However, agree that "one of the formidable problems that es psychiatric education is the need to protect ainst a diffusion of training so that our trainees do t end up learning less and less about more and ore" (15, p. 262). In working toward that goal we ve stressed the psychiatrist's unique role as a physi-an in the interdisciplinary community mental health am.

In our opinion Lipowski's recent attempt to define sychiatric-therapeutic consultation" (consultation consultation-liaison psychiatry) as the primary con-ltation model for psychiatrists (2) is both restrictive d incomplete. We agree that there are two major odels of consultation in the field of psychiatry and at they differ significantly in their goals, participants, ttings, and methods. Lipowski concludes that the sychiatric-therapeutic model of consultation entails ovision of optimal care for the individual patient hile the community mental health model involves provision of assistance to mental health care givers. Community mental health consultation need not exclude, as Lipowski suggested, a patient-centered focus as a primary goal in the working alliance. We believe that both methods of consultation, the in-hospital consultation-liaison model and the community mental health consultation model, are essential skills for the psychiatric practitioner. We propose that both methods should be taught and the psychiatric-therapeutic model be included in another part of our residency curriculum. The Oregon program is designed to give all psychiatric residents a required intensive experience in community psychiatry that will prepare them to meet the pluralistic and changing role of the psychiatric practitioner.

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Comparative Efficacy of Tofisopam and Placebo

BY HAROLD L. GOLDBERG, M.D., AND RICHARD J. FINNERTY, PH.D.

Tofisopam is a new agent marketed in Europe as a minor tranquilizer. The authors conducted a 4-week double-blind trial of this drug compared with placebo in 57 outpatients with anxiety and depression. They found that according to physician ratings and patient self-ratings tofisopam was an effective anxiolytic agent for subjects with anxiety and depression. Twenty-one percent of the patients receiving tofisopam and 10% of those receiving placebo reported side effects. The drug was especially effective in the treatment of somatic difficulties.

TOFISOPAM is a new agent developed by a European pharmaceutical firm. It bears a structural resemblance to diazepam (Valium) and oxazepam (Serax). Although clinically a benzodiazepine, tofisopam differs from the diazepam-like minor tranquilizers in the position of the nitrogen atoms (see figure 1). This structural difference manifests itself in a pharmacological and clinical profile that sets it apart from the minor tranquilizers currently in use in the United States.

Tofisopam has been investigated in approximately 1,200 patients in 29 trials in Europe. The European investigators found that tofisopam has the therapeutic characteristics of a minor tranquilizer and also has a mild stimulatory effect. It produced no substantial muscle relaxation, ataxia, loss of concentration, fatigue, or drowsiness. It is currently on the market in France and Hungary.

Szobor's open study of 20 patients (1) found that best results were obtained in vegetative neuroses, depression, and anxiety. According to a 1971 report by Danel in the files of Diamant Laboratories in France, in an open study of 47 patients with anxiety and depression a rapid marked anxiolytic effect was found in 23 patients given a daily dose of 200–300 mg/day of tofisopam. These patients also showed a reduction in psychosomatic complaints. Danel also reported that in an open study by Fouks tofisopam was given to 61 patients for 6 months. The patients who had hyperactivity and anxiety with psychosomatic disturbanc responded particularly well to the drug. Danel also r ported that Herbeuval found that 29 of 41 patients w psychoneurotic disturbances such as anxiety and d fuse neurovegetative symptoms responded favorat in an open study to 100 mg/day of tofisopam in 3 days. Patients with anxiety accompanied by neurov etative symptoms responded best. Finally, Juhasz a Tringer (2) evaluated 32 patients with various forms chronic neuroses and neurotic personality disord given 150 mg/day of tofisopam. Clinical observati using the Symptom Distress Checklist (HSCL) (3) r vealed that patients with somatic symptoms respond best to the drug. Normalization of diminished sex drive occurred in 5 of these 32 patients.

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METHOD

Study Design

The study reported here was undertaken to evalu the anxiolytic and antidepressant effects of tofisop and placebo in the treatment of patients with cc current symptoms of anxiety and depression. Subje satisfying the selection criteria were given either r fisopam or a similar-appearing placebo on a doub blind basis. Evaluations were conducted before tre ment and potentially at 1, 2, and 4 weeks after init tion of the study. Subjects could be terminated fr the study for reasons consistent with high standards patient care: lack of therapeutic response and the as ciated need for intervention, emergence of substant side effects, and remission of symptoms. Inform consent was obtained from all of the participating s jects.

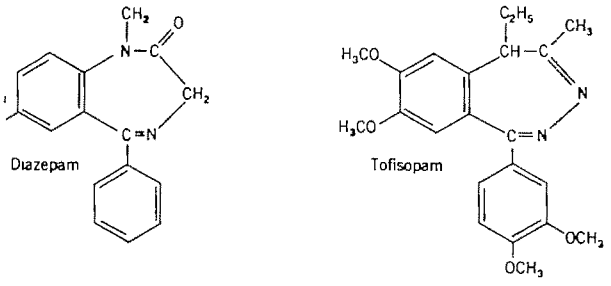
Population

A total of 57 outpatients were admitted to the stu All of the patients were at least moderately ill: 42. were moderately ill, 38.6% were moderately sever ill, 15.8% were severely ill, and 3.5% were extrem severely ill. Twenty-one percent of the patients w men and 79% women. The average age was 37 years.

Subjects were admitted to the study if their sco on the depression scale of Raskin and associates and the Covi Rating Scale for Anxiety (5) were grea than 13 and neither score was less than 5. The me total for the depression scale was 9.7, and the anxi mean was 11.2. Subjects were also required to h total Hamilton Anxiety Scale (6) scores greater th

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GURE 1
ructure of Diazepam and Tofisopam



. The mean pretreatment Hamilton anxiety total ore was 24.48.

In addition, a depression checklist, patient and fam- history inventory, and the Klein procedure for the agnosis of endogenomorphic depression (7) were mpleted by the physician at intake. Baseline analy- of background characteristics revealed that the ran- mization of the total patient population into the two atment groups clearly produced two patient sam- es that were not distinguishable from each other.

perimental Measures

At each measurement period the treating physician ted each subject on a global scale of severity of ill- ss with seven categories from not ill to extremely verely ill. Measures of therapeutic effect ranged om very much improved to very much worse. Pa- nts rated their experience on a similar scale. Pa- nts who failed to complete the 4-week study were signed a final rating from either the final disposition hedule or the previous rating depending on circum- ances and the opportunity for physician interview.

The Hamilton Rating Scale for Depression (8) was ministered at intake and at week 4 of the study. Un- rtunately, patients who failed to complete the 4 eeks could not be consistently interviewed to obtain al ratings, and posttreatment scores are available ly for completers. The Hamilton Anxiety Scale (6) d Symptom Check List (SCL-56) (3) were adminis- red before treatment and at 1, 2, and 4 weeks on edication. Endpoint ratings were assigned to sub- cts who did not complete the 4 weeks of study. The L-56 was scored to produce the eight composite rat- gs suggested by the assessment manual of NIMH's ew Clinical Drug Evaluation Unit (NCDEU) (9): so- atic complaints, cognitive difficulties, irritability- stility, depression, fear-anxiety, hostility, irritabil- -sensitivity, anxiety.

bject Attrition

Seven patients receiving active medication and 12 acebo recipients were terminated from the study be- e the 4-week completion date. Only 3 subjects owed enough improvement to justify their being ex- sed from treatment.

RESULTS

Twenty-one percent of the patients receiving tofisopam reported the emergence of side effects during drug treatment; 10% of the placebo recipients also indicated some difficulties. The total number of complaints was nearly equal across treatment groups. One patient receiving tofisopam had an immediate and severe emergence of dizziness, syncope, excitement, and confusion that did not moderate after 7 days. This patient was excused from the study. The other patient who dropped out of the study because of side effects had more moderate levels of difficulty, but the side effects (blurred vision and dry mouth) persisted for nearly 2 weeks.

After the first week of treatment no patients had dropped out of the study and no difference was observed between tofisopam and placebo. By week 2 tofisopam was more effective than placebo, but 9 patients had left the study. By week 4, 13 placebo subjects had dropped out of the study. Most of these dropouts were nonresponders, and the week 4 distribution of ratings is greatly influenced by their elimination. The final global illness ratings, not confounded by subject attrition, showed tofisopam to be significantly more effective than placebo (see table 1).

Both the physicians' and the patients' ratings of therapeutic effect were not significantly different for treatment groups (chi-square analysis was used; $p=.05$ was accepted as significant). However, a consideration of the total distribution indicates that if ratings of a little improvement were combined with ratings of nonimprovement, the distributions would be equal to the global illness ratings listed in table 1 and would show tofisopam significantly more effective than placebo.

Analyses of outcome measures rated at each of the time intervals may have been confounded by subject attrition. At week 1 the sample was still complete and the analyses were not biased by the dropout of subjects. At week 2 the subjects with negative responses to tofisopam, as well as some placebo nonresponders, had dropped out of the study, but the ratings at week 2 were definitely biased in favor of tofisopam. By week 4 a large number of placebo recipients had dropped out because of a worsening of illness due to the absence of active medication. Most of the remaining placebo subjects were placebo responders or placebo recipients who had not become sufficiently more ill to warrant intervention. The week 4 data were biased in favor of placebo. Our presentation and discussion of outcome measures will focus on week 1 and endpoint data collections to avoid the confounding of results by sample attrition.

Although an exhaustive analysis of the pretreatment variables revealed no differences between treatment groups, in all of the analyses reported below the posttreatment scores have been adjusted for pretreatment level.

The Hamilton depression scale scores were ob-

TABLE 1
Physicians' Global Illness Ratings*

	Week 1		Week 2		Week 4		Endpoint	
Illness Rating	Tofisopam	Placebo	Tofisopam	Placebo	Tofisopam	Placebo	Tofisopam	Plac
No illness to mildly ill	8	8	17	6	18	9	20	1
Moderately to severely ill	20	21	6	19	4	7	8	1
Total	28	29	23	25	22	16	28	2

*The difference between tofisopam and placebo in the first week was nonsignificant (χ^2=1.0). For the second week it was significant (χ^2=11.96; p<.001). Fo fourth week it was nonsignificant (χ^2=2.94). At endpoint it was significant (χ^2=7.80 p<.01).

tained only before treatment and at week 4. The analyses of these outcome measures involved comparisons of most of the tofisopam subjects (22 of 28) with fewer placebo subjects (16 of 29). The placebo subjects may have comprised a large number of placebo responders. The Hamilton depression total score still showed significantly less illness for tofisopam subjects (analysis of variance covaried for pretreatment ratings; p=.05). The individual items of middle (p<.05) and late (p<.01) insomnia, somatic anxiety (p<.05), general somatic symptoms (p<.05), hypochondriasis (p<.01), and paranoid symptoms (p=.05), although rarely present, were all significantly more relieved for tofisopam subjects. Although the Hamilton depression scale certainly shows significant improvement, this does not necessarily indicate improvement in depression because none of the depression items showed any significant improvement and the reason for significance of the total scale relates to the somatic items listed above. These findings, despite the bias of attrition, suggest that tofisopam may be an unusually effective treatment for patients whose principal conflicts are manifested in somatic concerns.

The Hamilton Anxiety Scale scores were obtained at each rating interval. At the end of week 1 no significant differences were observed between treatment groups according to analysis of variance covaried for pretreatment ratings. The final ratings showed tofisopam significantly more effective in reducing total symptomatology (p<.04) and the cluster of items termed "somatic" (p<.03), which includes insomnia, muscular somatic, sensory somatic, cardiovascular symptoms, respiratory symptoms, gastrointestinal symptoms, genito-urinary symptoms, and automonic symptoms. One other single rating, tension, showed a nonsignificant trend (p=.08) in favor of tofisopam.

The SCL-56 measures are best expressed in terms of the cluster scores. The analysis of these at the end of the first week showed significantly greater improvement for tofisopam on the somatic and anxiety cluster (p=.03 for both) and a trend in favor of tofisopam (p=.06) for the depression and fear-anxiety cluster. The endpoint ratings showed a significant difference (p=.05) in favor of tofisopam on the irritable-sensitive cluster. Four other clusters were nearly statistically significant: somatic (p=.06), depression (p=.07), fear-anxiety (p=.08), and anxiety (p=.09).

The final ratings showed a large number of individual SCL-56 items for which the patients' self-ratings clearly showed more marked response to tofisop than to placebo. Thirteen individual items were sig cantly less prevalent in tofisopam subjects and anot 8 items showed strong tendencies to significa (p<.10).

DISCUSSION

It is clear from the analyses above that tofisop was an effective anxiolytic agent in the treatment these subjects with mixed anxiety and depressi with a unique advantage for patients with som complaints. An analysis of the side effects did not dicate that tofisopam had a high degree of risk for tient safety. The number of patients who dropped of the study because of these side effects (N=2) w somewhat less than expected for an active psych tropic agent. The physician and patient ratings of gl al effect agreed with each other and with the hypot sis that tofisopam is effective. The total Hamilton pression and anxiety rating scale scores show significantly less posttreatment symptomatology tofisopam subjects. The individual Hamilton anxi items indicated that tofisopam was most effective the treatment of somatic difficulties. The patients' s ratings of symptomatology showed significant effe for tofisopam after the first week of treatment, physicians' ratings did not show such rapid impro ment. The self-rating clusters had only one signific difference (irritability-sensitivity), but four ot trends would have achieved statistical significance somewhat larger sample (N=80) with the same dis bution had been analyzed.

Further analyses of the efficacy of tofisopam co benefit from the results of this study. It would be v interesting to see if the interaction between tofisop and a high degree of somatic complaints would cont ue in a larger series and would be distinct from effect of a "standard" medication on patients w anxiety and depression.

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:osmetic Surgery as an Antecedent of Life Change

Y MYRON L. BELFER, M.D., JOHN B. MULLIKEN, M.D., AND THOMAS C. COCHRAN, JR., M.D.

f 42 patients who underwent cosmetic surgery, 4 ıtients, who gave no indication of an impending life ıange before surgery, obtained a legal separation or vorce 3–6 months later. These 4 patients had had rong parental domination and hostile relationships ith younger siblings; they had a need for success on eir own terms and for the rapid closure of potential onflict. The authors suggest that such patients are ndergoing significant identity changes that become onscious only after cosmetic surgery, which tests out eir shift from passivity and withdrawal to activity nd participation.

LTHOUGH CERTAIN PATIENTS worry plastic surgeons nd psychiatric consultants because of the possible resence of psychopathology, which could make such n operation inadvisable, most patients seek cosmetic ırgery to alter a feature or features that cause self-onsciousness, unwelcome attention, or adverse com-ent; to gain social acceptance; or to increase their pportunity for employment (1, 2). There is another :oup of patients, however, previously undescribed, who desire a concrete change in their appearance through surgery in preparation for a significant life change. The psychodynamics and psychiatric evaluation of this subset of patients are described in this report.

We studied 42 patients who requested rhinoplasty, abdominoplasty, or augmentation or reduction mammoplasty; each had pre-operative psychiatric evaluations and were followed for 1 to 2 years post-operatively. Within 3 to 6 months after surgery, 4 of the patients obtained a legal separation or divorce without any prior indication that a life change was being planned or evidence of an intervening psychological crisis with an impact on known family dynamics.

These 4 patients present a strikingly uniform psychodynamic pattern. The early life of each was marked by social isolation, hostile relationships with younger siblings, and an early need to opt for security and closure. The patients lacked close parental ties and sought through education to maximize their potential, gain security, and prepare themselves for separation from their families. We did not note any specific patterns of life disruption. These patients' peer relationships were circumscribed and at the time of evaluation for surgery none of them had long-standing friendships of current significance; yet by formal psychiatric criteria none of them would be described as schizoid.

These patients had achieved significant success in their work lives and were currently considered valuable professionally. They did not express the sense of inadequacy described by Meyer and associates (3). Their level of achievement surpassed that of other family members and their spouses.

As for all patients for whom there is concern about their desire for cosmetic surgery, a psychiatric evaluation was performed to determine the patients' ex-

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pectations about the surgery, the meaning of the expected change in appearance for their personal and social lives, and their spouses' feelings regarding the surgery (3, 4). The requested operations did not have any relationship to a psychologically significant bodily deformity of a parent. In cases where the patients' occupation involved public exposure, we determined whether or not the patients had shared their plans for surgery with their colleagues.

In giving psychiatric clearance for surgery, we used conservative criteria (5), which emphasized a lack of 1) discernible significant psychopathology, 2) unrealistic surgical expectations, and 3) plans for precipitous, unrealistic life changes such as divorce or job change. None of these patients had received psychotherapy or had a history of significant psychopathology.

CASE REPORTS

Case 1. Mr. A, a 37-year-old married management consultant, sought a rhinoplasty at age 35 when he was at a high point in his career. He was referred for pre-operative psychiatric consultation because of the general concern about adult males who request rhinoplasty.

Mr. A was the older of 2 male children, and his early life had been marked by parental unavailability and lack of affective interchange. He saw his father as an angry, unloving, aggressive person who was dominated by his wife. He saw his mother as a passive-aggressive woman. As far back as he could remember Mr. A recalled feeling a sense of distance from his parents, a persistent desire to "escape," and a fear of parental retaliation. He had had few outside interests other than his studies, few friends, and did not date until college. Mr. A received a doctorate degree in physics but to achieve financial success chose a career in management, where he was successful. He married the first woman he dated, a woman who made few demands and who by virtue of her training could be financially independent. Retrospectively, Mr. A saw his marriage as a calculated decision, made at the right time with the right person, and one that caused the least disruption with his family.

Mr. A had a significant objective nasal deformity but a more obvious divergent strabismus. He said that at that point in his life he was feeling better about himself, generally took more pride in himself, and wanted a rhinoplasty to achieve a more pleasing profile. He had previously lost 13.6 kg through diet and exercise and had begun to dress fashionably. There was no evidence of overt marital discord, but Mr. A said that his wife "lacked the vision of the future" which he had achieved as he became successful.

Postoperatively Mr. A expressed immediate satisfaction with his rhinoplasty. Two months later he called the psychiatric consultant for an appointment and said that he was considering a separation from his wife. There had not been any intervening psychological crisis. The response to the rhinoplasty at work, socially, and within his family had been entirely satisfactory; the patient, however, felt that now his wife would be unable to match his lifestyle, and when he confronted her with changes that he wished she would make, she refused, which led to their eventual separation.

Case 2. Ms. B, a 24-year-old married dental hygienist, had had an augmentation mammoplasty when she was 22 and a year later requested a cosmetic rhinoplasty. Psychiatric consultation was sought because of the minimal objective deformity and the fact that this was the second request for plastic surgery within a year.

Ms. B was the oldest of 4 girls. In her early life emphasis had been placed on family and loving one's own family, yet Ms. B recalled a lack of affective contact and much covert communication among family members. Her family considered the role of the woman to be stereotypically passive and compliant.

Ms. B met her husband in grammar school and married him to be with him while he was in the service. Over the following 6 years Ms. B received an associate degree and became a dental hygienist. In this job, and because she was in a metropolitan area, she found herself exposed to a wider variety of people and experiences than previously. She found her husband resistant to these new experiences, and increasing tension developed. Ms. B resolved to correct what she felt to be inadequacies in her self-image, ostensibly for her own sense of well-being.

Six months after a successful minimal rhinoplasty Ms. B contacted the consulting psychiatrist to say that she was separating from her husband. She placed emphasis on her failure to get her husband to change. She described him as resistant to change, distant, and uncommunicative. Ms. B said she wanted the opportunity to explore interests and social contacts that her lifestyle with her husband had not permitted.

DISCUSSION

The 4 patients in the study group evidenced strong parental domination, a need to succeed on their own terms, and a need for rapid closure of potentially conflictual situations. Diagnostically, such patients may be seen as having significant concerns with their identity that only come to consciousness and lead to life changes after cosmetic surgery. The operation serves as the fulfillment of some seemingly less significant goal, i.e., changing an aspect of their body, but, as discussed by Reich (1), the initial reason a patient requests cosmetic surgery may not be the true reason he or she fears that the operation may be refused, and it has been noted that the real, less conscious motivation may become evident only after surgery. Meyer and associates (3) elaborated on the same patients' wish to shift from passivity and withdrawal to activity and participation, which is signified by the surgical intervention.

The cosmetic surgical procedure in these cases can be seen as a manipulation, performed unknowingly by the surgeon, which serves to consolidate the patient's identity and test out, in a derivative manner, the patient's acceptability and competence in an active role. The patients were pleased with their surgical outcome and less consciously saw this as permission to pursue active change in other spheres of their lives, namely separation or divorce. The psychiatric consultant should be alert to the possibility of marital disruption when a patient requests cosmetic surgery and his or her history includes difficulty with affective expression, a marked difference in the level of achieve

nt from that of his or her spouse, a need for closure, d the sense that the patient is asking the surgeon to odify his or her appearance in conjunction with a ent professional, personal, or social success.

The consultant seeing such patients must pursue the ective gaps in these patients, inquire carefully into possibility that the requested surgical operation resents a displaced concern (6), and assess the de- e to which surgery offers the patients the opportu- y to escape their passivity. Psychotherapy may per- the exploration of the less conscious motivations ding up to cosmetic surgery and permit the decision make a life change to take place more openly.

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he Impact of Political Process on Hospital Psychiatry

GEORGE L. HOGBEN, M.D., TECLA CRITELLI, M.D., AND RALPH HOFFMAN, M.D.

litical forces motivated the New York State partment of Health to withhold payment for dicaid-financed inpatients on therapeutic passes. e elimination of therapeutic passes had many gative effects on patient care. The authors believe s experience demonstrates that political factors can erwhelm standard clinical practice and reasoned alth planning to force irrational change on health e delivery.

LITICAL EVENTS HAVE produced remarkable anges in psychiatry. Altered attitudes on the part of alth authorities have led to dramatic reductions in federal budget for training psychiatrists. Economic tagflation" has contributed to shorter hospitalizations for many patients with acute psychiatric conditions. Prolonged hospitalization for chronic disorders has been much less available since passage of the Community Mental Health Centers Act of 1963. Funding for psychiatric research has been drastically curtailed by these economic conditions.

Many professionals believe these political processes are adversely affecting psychiatric patient care. However, data establishing a negative impact of political events on patient care have not been forthcoming. In fact, benefits from short-term versus long-term hospitalization have been demonstrated (1). These results suggest that political forces which reduce the amount of time available for inpatient therapy may benefit some patients.

However, there are many aspects of patient care that have been adversely affected by political forces. For example, psychiatrists have relied on therapeutic passes (temporary authorized absences from the hospital) in treating hospitalized patients. Most therapeutic passes are granted for short periods during the day. Recently, New York State health officials denied reimbursement to hospitals for time spent on therapeutic passes by Medicaid-financed patients. Many hospitals eliminated therapeutic passes in response to this directive. This paper outlines the political process that led to the Medicaid reimbursement guidelines and describes the impact on the patients and staff in a short-term psychiatric unit of the elimination of therapeutic passes.

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POLITICAL PROCESS

Our description of the political process that led to the decision to end reimbursement to Medicaid patients on passes is based on reports that appeared in the *New York Times* in October and November of 1976.

After it came close to bankruptcy, New York City was forced to adopt austerity measures to satisfy its fiscal rescuers, New York State and several large monetary institutions. A projected budget deficit of $100 million in the Health and Hospitals Corporation threatened the success of the austerity program. The root of the problem seemed to be 5,000 unused beds in the municipal, voluntary, and private hospital system (2).

The situation was highly sensitive because many different people, state and city agencies, and jobs were involved. The Health and Hospitals Corporation had already effected major cutbacks in its own system and did not favor additional measures. The voluntary and private sector was also reluctant to reduce bed capacity.

Political maneuvering over the budget between the mayor of New York City and the governor of New York State sifted down through the network of city and state agencies that have overlapping responsibilities. The State Department of Health and the Bureau of Audit and Control both criticized the city's inability to check Medicaid abuses (2, 3). The City Department of Health and the Human Resources Administration countered by charging that the state agencies had not cooperated by tightening regulations.

On Oct. 28, 1976, the *Times* reported that state auditors had uncovered city procedures to fill beds with Medicaid patients in an effort to reduce deficits (4). State officials warned that Medicaid would not pay for unnecessary hospitalizations.

On Nov. 9, 1976, the *Times* reported that state auditors had "discovered" the practice of therapeutic passes for psychiatric patients (5). The state agencies said they intended to review the granting of therapeutic passes. Over the next five weeks the state agencies quietly designed a policy to deny hospitals reimbursement for Medicaid-financed patients during the time they were on therapeutic passes. The policy was based on the definitions of an inpatient in the Code of Federal Regulations (6) and the State Medical Handbook (7), which govern the administration of medical assistance programs. These administrative codes define an inpatient as one who receives room, board, and professional services in a hospital on a "continuous 24 hour basis." Hospital Memorandum 76-128 (8) from the New York State Department of Health instructed hospitals "not to bill Medicaid for periods while patients are not actually in the hospital." The memorandum threatened that hospitals violating the directive would be referred to the Attorney General for fraud investigation.

Hospital administrators were informed of the new policy in mid-December 1976. The unit chiefs of Mount Sinai Hospital were notified of the policy a instructed to implement it as soon as possible. The p icy was implemented by the unit we will describe Jan. 1, 1977.[1]

SETTING

From Jan. 1 to March 30, 1977, the 3 months af the termination of therapeutic passes, observations individual patients' courses of hospitalization a changes in the milieu and staff responsibilities were corded. These observations were made during co munity and small-group meetings, therapeutic team ports, and staff meetings.

The 23-bed general psychiatry unit is one of f adult and child inpatient psychiatry units at Mo Sinai Hospital. During the 3-month observation per 65 patients were cared for on the unit. Forty-four tients were service cases, 21 of which were financ by Medicaid. Nine service patients had no insura coverage on admission but qualified for emerger Medicaid support. Fourteen service patients had B Cross or Medicare insurance. Twenty-one cases w semi-private patients, financed by Blue Cross or Me care. Therefore, the care of 46% of these patients v directly affected by the stoppage of therapeutic pas for Medicaid patients.

OBSERVATIONS

Patient and Staff Reactions to Policy

The policy of discontinuing therapeutic passes Medicaid-financed patients was announced at a regu community meeting. The patients were shock Many therapeutic meetings during the observation riod were devoted almost exclusively to the patier futility and rage at the loss of therapeutic passes. tients rejected attempts to introduce topics relatec other important therapeutic issues. They thought authorities did not care about patients with me conditions and openly wondered if mental health pl ners realized the needs of psychiatric patients. The tients believed the action showed "again" that gove ment was unresponsive to citizen needs. They w convinced that politics rather than therapeutic n governed care.

The staff was also shocked and angry at losin valuable therapeutic tool. Fostering independenc an overriding treatment goal of the unit. Short pas were used to achieve clearly defined therapeutic go Moreover, everyone felt powerless because there been no warning about the new pass procedure contingency planning was therefore not possible. M rale fell sharply and staff acted with grim resignatior

The patients thought that the loss of therape

[1]The policy was rescinded in November 1977 and Medicaid-finar patients may now have short therapeutic passes.

sses would increase their reliance on the hospital. any patients felt hopeless about leaving under these rcumstances. Chronic patients who had experienced evious separation from the hospital were panicked at e prospect of leaving "cold turkey." One patient ted, "I had so much trouble getting out of here last ne even in small steps. I don't think I'll ever get out w."

These discussions stimulated the patients to draft a tition to Medicaid officials that explained the probms created by the termination of passes and requested the decision be reversed. The petition, which scribed the patients' fears about increasing dependcy on the hospital, was circulated throughout the spital and all members of the hospital community re asked to sign it.

fects of Policy on Patients' Hospital Course and anagement

The patients' dependency concerns were well-unded. Many of them had no place to live after disarge and no job or money. Other patients had serely strained relationships with roommates and fam- members. Therapeutic passes had given patients ne to conduct job interviews, make basic living arngements, and test themselves in difficult interrsonal situations before discharge. Without these portunities, patients were severely handicapped in eparing to leave the hospital. For example, a 56-ar-old man who led a withdrawn life had abandoned s single room and earthly possessions during an epide of involutional psychosis. When the psychosis reitted, he was unable to leave the unit to make new ing arrangements and buy basic necessities. He beme a frightened, depressed, and actively contemated suicide, saying, "I'd rather kill myself than do this after I leave."

This reaction was typical of the difficulties expericed by the patients denied therapeutic passes. Most tients confronting abrupt discharge without graded paration experiences developed severe panic attacks their discharge dates approached. These patients came restless and irritable and exhibited insomnia, lpitations, and breathlessness. Several patients exrienced severe reactivation of their psychotic sympms, which delayed their discharge. Many patients, en those who had not had suicidal ideas on admison, actively contemplated suicide. These suicidal eoccupations reached epidemic proportions on two casions.

The staff attempted to conduct accompanied walks d home visits to help patients with separation fears. alks had always been offered to disorganized pants to afford some time away from the unit routine. ey were now used with organized patients to prode some predischarge independence. The walks re very stressful to the staff because this added reonsibility had not been considered in staffing plans. rsonnel cutbacks had also reduced the number of aff available for patient care. The accompanied walks and home visits further removed staff from important work on the unit.

Many patients were angry and resentful over being accompanied off the unit when they felt well. They thought that staff was suspicious about their mental condition. In some instances patients became convinced they were not progressing and doubted their own status. One patient remarked, "I feel all right now but I don't know what to think when I can't go out alone. I begin to think I'm a baby and doubt my own competence."

Effects of Policy on Interpersonal Relationships

As the patients directly experienced the impact of the decision, they became more angry and resentful toward the hospital and unit staff, especially the doctors. The patients felt the hospital administrators and doctors should have prevented government officials from making the policy. They were dismayed at seeing the doctors as helpless as they themselves were in directing therapeutic plans and repeatedly urged the staff to ignore the ruling. When pass requests were denied, patients charged that the staff was not dedicated to helping them meet the realistic problems of their lives. One woman angrily commented, "None of you care whether I can handle how I feel toward my husband. If you did you would let me out to see if I can. These family meetings aren't the same as living with someone."

The splitting of patients' anger among Medicaid authorities, hospital administrators, and unit staff created a difficult problem for social therapy on the unit. Patients frequently tried to make staff responsible for solving their problems with interpersonal relationships and daily living. Since patient independence is a unit goal, staff members were often objects of patient anger. Staff was competent in helping patients identify anger and handle it through verbalization and mobilization of personal resources. However, the staff felt therapeutically helpless when the patients expressed anger at them over the pass situation. The staff was powerless, and no solution seemed available to promote patients' personal independence. The staff could not catalyze the patients' problem-solving abilities, and pointing out the reality of the process leading to the pass policy only ignited more patient anger. The patients felt the staff had lost its credibility. The patients' resentment of staff spread to other therapeutic endeavors and anger and mistrust were pervasive. The patients and staff became divided.

The division between patients and staff was extremely frustrating because the pass policy created many therapeutic and interpersonal tensions that demanded candid communication. Patients with separation fears and suicidal preoccupations were of great concern to the staff. Moreover, patients felt cooped up and overstimulated by the close personal contact on the unit, and tempers often flared between patients. Several patients with paranoid tendencies who relied on periods of isolation overtly threatened bodily harm

when they could not leave the unit for short periods. Patients felt sexually stimulated by close conditions and expressed sexual feelings for other patients and staff during open meetings for the first time in our experience. The patients also decided they could work around pass restrictions by eloping, and several patients eloped to test this idea.

The patient community was also divided between those who could and could not have passes. Even though all of the patients denounced the situation, Medicaid patients openly expressed jealousy and anger toward patients who were receiving passes. Many patients said the situation was similar to what they had experienced all their lives. "The rich get richer." Medicaid patients frequently said they always felt like second-class citizens. Being treated differently from others on the ward reinforced their sense of inadequacy, a feeling that "I always knew others thought I was no good. Now I know it . . . I'm not."

COMMENT

This experience shows how political factors can overwhelm time-honored clinical practice and health planning activities to force irrational change on health systems. Politicians and bureaucratic agencies actively competed to uncover fiscal waste and find immediate solutions. It is logical not to charge Medicaid when a patient does not occupy a bed. However, a therapeutic pass is not an absence from in-hospital status. An interpretation permitting passes could have been made within the Medicaid regulations. Medical consultation was not sought during the process of changing the pass policy. Medical input should be a part of the decision process in order to avoid formulating policies opposed to clinical practice. Procedures to study the clinical and economic effects of the new policies were not established. Political process was the only means available to report the effects of the pass policy on patient care.

The termination of therapeutic passes had disastrous effects on patient care. Most patients who had been denied therapeutic passes developed severe panic reactions and active suicidal ideation during the discharge process. Symptom development at discharge is a known phenomenon, but the number of patients affected and the extent of their reactions increased greatly after passes were eliminated. Interpersonal tension, common on inpatient units, rose to dangerous levels. The pass restrictions reinforced the patient's low self-esteem. Patients overtly mistrusted staff over the pass issue, which made the development of positive therapeutic relationships extremely difficult.

The role of the staff was dramatically altered by the pass restrictions. Valuable staff time was diverted from intervention in the problems that brought patients to the hospital. Therapeutic meetings were largely devoted to discussing the patients' reactions to the loss of therapeutic passes, the predischarge panic and suicidal preoccupations, and increased interpersonal tensions.

Ironically, the change in pass policy, established to reduce costs, may have increased the cost of patient care. Depriving patients of graded separation experiences might lead to inadequate adjustment in the community and to increased likelihood of relapse, thereby increasing the number of hospitalizations. Moreover, so much time was drained from the unit's primary mission that patients may not have been adequately diagnosed.

Political events have a profound impact on mental health and psychiatry. The political process described herein occurred in a crisis atmosphere that suppressed open discussions. No provisions were made for planned implementation of the new policy or for assessment of outcome. In this case the political process was destructive to the patients, their care, the staff and possibly even the precipitating economic conditions. Open political processes, planned policy implementation, and well-organized assessment procedures are urgently needed to prevent destructive consequences of political action.

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ir Pollutants and the Admission Rate of Psychiatric Patients

Y MEIR STRAHILEVITZ, M.D., AHARONA STRAHILEVITZ, M.D., AND JOHN E. MILLER, M.A.

ie authors studied the correlation between mean ily levels of several air pollutants and the number of ergency room visits and inpatient admissions to a ychiatric hospital in St. Louis during one summer d fall. Nitrogen dioxide and carbon monoxide owed a positive correlation with emergency room sits by all patients, and nitrogen dioxide also had a sitive correlation with inpatient admissions of bjects with diagnoses that were unknown or could t be specified as psychotic. Nitrogen monoxide owed a negative correlation with inpatient dmissions during working days (but not during ekends and holidays) for all patients, as well as for ose with diagnoses that were unknown or could not specified as psychotic.

HE EFFECT OF air pollution on a variety of medical nesses has been studied extensively, and it is a well-tablished fact that certain air pollutants have a delerious effect on several medical illnesses (1). How-er, little is known about the effect of air pollution on sychiatric illness. In the present study, we examined e correlation between the mean daily levels of sever-air pollutants and the daily number of emergency om visits and inpatient admissions to Malcolm Bliss ental Health Center, a psychiatric hospital serving e population of St. Louis.

ETHOD

The study period consisted of 149 days during the mmer and fall of 1972. The air pollutants studied in-uded coefficient of haze, total oxidants, total hydrocarbons, carbon monoxide (CO), nitrogen oxide (NO), nitrogen dioxide (NO_2), and sulfur dioxide (SO_2). Total oxidants, CO, NO, NO_2, and SO_2 were measured in parts per million by volume. Total hydrocarbons were measured in methane equivalent parts per million. Coefficient of haze was measured by units of coefficient of haze per thousand lineal feet. The air pollutants were monitored in three stations in different areas of the city. The mean daily pollutant levels for the three stations were computed.

The Pearson correlation coefficient was computed between the mean daily level of each pollutant and the daily number of emergency room visits and inpatient admissions. Correlations with the daily number of emergency room visits were computed for the total patient population but not according to diagnosis, since no diagnostic information was available on patients who came to the emergency room but were not admitted to the inpatient service. Correlations between pollution variables and the number of inpatient admissions were computed for the total patient population as well as for selected diagnostic groups and specific diagnoses. The following diagnostic groups were used: psychotic illness (such as schizophrenia and major affective disorders), nonpsychotic illness (such as neurosis and personality disorders), and diagnosis either unknown or cannot be specified as psychotic (such as alcoholism, organic brain syndrome not specified as psychotic, and undiagnosed psychiatric illness).

Air pollution variables were correlated separately with the number of inpatient admissions for the following specific diagnoses: organic brain syndrome, schizophrenia, major affective disorder, neurosis, transitional situational disturbance, behavior disorder of childhood or adolescence, personality disorder, and alcoholism. The diagnosis used was the discharge diagnosis recorded in the medical record at the time of discharge. The diagnosis was made by at least one experienced staff psychiatrist.

Since we computed the correlation of seven air pollution variables with the number of emergency room visits and inpatient admissions, we choose an α level of .005 as statistically significant. Alpha levels of .01 and .05 were considered borderline significant. Pearson correlation coefficients were computed for the following periods: all days (N=149), days on (e.g., all days excluding weekends and holidays) (N=94), and days off (e.g., weekends and holidays) (N=55).[1]

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the time this study was initiated, Dr. Meir Strahilevitz was Direc-of Training and Research and Dr. Aharona Strahilevitz was Staff ild Psychiatrist, Malcolm Bliss Mental Health Center, St. Louis,). Dr. Meir Strahilevitz was also Assistant Professor and Dr. Aha-ıa Strahilevitz was Instructor, Department of Psychiatry, Wash-gton University, St. Louis. Dr. Meir Strahilevitz is now Associate ofessor of Psychiatry and Dr. Aharona Strahilevitz is Assistant ofessor of Child Psychiatry, University of Texas Medical Branch, alveston, Tex. 77550. Mr. Miller is Researcher-Statistician, Medi-Education Department, Southern Illinois University School of edicine, Springfield, Ill. Address reprint requests to Dr. Meir ahilevitz, P.O. Box 1502, Galveston, Tex. 77550.

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[1] For total hydrocarbons all days=73, days on=48, and days off=25.

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The reason for computing correlation coefficients separately for days on and days off was related to the following considerations.

1. The hospital was understaffed, particularly with physicians during days off. This was likely to influence the admitting physician's decision concerning admission of patients.
2. Pollution levels were likely to vary between days on and days off. Less pollution was generally expected on days off because of decreased industrial plant operations and decreased traffic.
3. Patients' exposure to air pollution could vary between days on and days off. On days off part of the patient population, such as laborers working outdoors on days on, might protect themselves to some extent from the effects of air pollution by remaining indoors.
4. The probability that patients would go to the emergency room, and therefore the probability that they would be admitted, might also vary from days on to days off.

In addition to the simple correlation coefficient we also used the first-order partial correlation coefficient, which indexes the relationship between two variables while controlling for the effect of a third variable. The partial correlation used in this study controlled for the days on/days off effect. The existence of nonzero partial correlations would indicate that there are relationships between air pollution variables and behavioral variables that are independent of the days on/days off effect.

RESULTS

The coefficient of haze, total oxidants, total hydrocarbons, and SO_2 did not show any statistically significant correlations with emergency room visits or inpatient admissions. Mean daily CO concentrations showed a significant positive correlation with daily emergency room visits for all patients during all days ($N=149$, $r=.25$, $p<.005$) and with daily inpatient admissions during all days for the group of patients with diagnoses that were unknown or could not be specified as psychotic.

NO_2 correlated positively but not significantly with admissions for all days for alcoholics ($r=.22$, $p<.01$). NO_2 also showed a simple correlation with admissions for all days as well as a partial correlation with patients with organic brain syndrome ($r=.20$, $p<.05$ and $r=.18$, $p<.05$, respectively).

Mean daily NO correlated negatively with the daily number of inpatient admissions for all patients on days on ($r=.36$, $p<.005$). NO correlated negatively with daily admission rates for patients with diagnoses that were unknown or could not be specified as psychotic on days on ($r=-.33$, $p<.005$). The partial correlation for this group of patients was also significant ($r=-.25$, $p<.005$). In specific diagnostic groups, the mean daily concentration of NO correlated negatively but not significantly with days on admissions in alcoholics ($r=-.26$, $p<.05$) and in patients with organ syndrome ($r=-.21$, $p<.05$).[2]

DISCUSSION

Our findings suggest that concentrations of pollutants may be correlated with the emerger visit and inpatient admission rates of psychi tients. Such correlations are apparently r strong ones. Even though our selection of cance level of .005 was based on reasonable erations, it was still to a certain extent arbitr important to stress that any research strategy multiple analyses may lead to spurious "sig results.

The number of patients in some of our d subgroups might have been too small to test fo cant correlation with the level of air pollutant dition, one has to keep in mind that even if th a valid significant correlation between a vari specific air pollutants, such a correlation woul course, indicate a causal relationship; it might reflect a correlation between the patient "beh variable and another set of environmental that are correlated with the pollution variable ed in our study. Keeping all of the above rese in mind, it appears to us that our data suggest tentative hypotheses.

It appears from our data that certain ps conditions may be sensitive to specific air po mean daily air concentrations of NO_2 may be ly correlated with the number of emergency r its by all patients and with the number of inpa missions of persons with diagnoses that are n or cannot be specified as psychotic.

It is possible that patients with diagnoses of ism and organic brain syndrome are particular tive to the level of NO_2 in the air, since th groups showed correlations that approached cance. Both the combined sample of patients subgroups with a positive correlation betwe levels and inpatient admissions showed a nega relation between the daily number of inpatien sions and air concentration of NO. These obse may reflect a negative correlation between a tion levels of NO_2 and NO, or they may refle tective effect of NO on the "irritating" effec on some psychiatric patients. The protective e NO on the damaging biological effects of known (2). NO is a known anesthetic and also algesic qualities (3). Thus the possibility tha have a beneficial effect on certain psychiatric deserves consideration.

The negative correlation between NO level patient admissions only on days on may be r the existence of threshold phenomena for the

[2]A table summarizing the results is available from Dr. M levitz on request.

ect of NO_2 on some psychiatric patients, with a protective effect of NO being apparent only when the level of NO_2 exceeds this threshold level. The concentration of NO_2 in the air might have been related to increased industrial plant activity during days on compared with days off.

Air concentrations of NO_2 and NO were rather low. The maximum mean daily levels measured in our study were .29 ppm for NO_2 and .97 ppm for NO. The minimum level of NO_2 known to cause biological effects in acute exposure experiments is about 13 ppm (1). Thus if our findings on the correlation of NO_2 and NO with emergency room visits and inpatient admission rates indeed reflect a valid causal association, these findings may indicate that certain psychiatric patients are unusually sensitive to the effects of NO_2 and NO.

Our findings also indicate that mean levels of CO in the air may be associated with increased emergency room visits by psychiatric patients. The maximum air concentration of CO that was recorded in our study was 23 ppm, which approaches the minimum level of 30 ppm known to cause biological effects in man in acute exposure experiments (1).

Our data suggest that it may be worthwhile to evaluate the possibility of a therapeutic effect of low levels of NO on certain psychiatric illnesses, in particular alcoholism and organic brain syndrome.

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The Role of a Cinema Seminar in Psychiatric Education

GREGORY K. FRITZ, M.D., AND RICHARD O. POE, M.D.

the goals of psychiatric education are evaluated d innovative teaching methods are sought, the rrent cinema represents an untapped resource. oughtful viewing of contemporary films with serious cussion of them in a film discussion group as part of residency training program is a useful and ioyable teaching format that helps bridge the gap tween the art and science of psychiatry.

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INDIVIDUALS BEGINNING psychiatric training usually have primarily scientific backgrounds. The difficulty of gaining acceptance to medical school encourages interested undergraduates to specialize early in sciences in hopes of furthering their chances, and medical school intensifies the scientific focus. Yet every physician needs exposure to the humanities as well, and as Nemiah (1) notes, the psychiatrist especially must be both poet and scientist. Chessick (2) summarized it well when he stated,

> The urgency for training future psychotherapists in the humanities and immersing them in the arts as well as the usual scientific subjects has a theoretical foundation. The psychotherapist, to do an optimal job, must be familiar both with the language of scientific understanding and of the humanistic imagination and he must be able to shift back and forth with facility between these maps of psychic interaction so that what he misses on one map, he finds on the other. (p. 334)

In an effort to further develop residents' humanism and capacity for empathic understanding, a number of medical schools have made use of a seminar in which major literary works are discussed. Since 1958, Bar-

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chilon has been using such a seminar in the education of psychiatrists and medical students (3). Hofling (4), Fellner (5), and Silberger (6) have reported on variations in the literature seminar format. Psychiatrists since Freud have written at length on the nature and function of art, examining the creative process and product from every conceivable vantage point. Several recent publications dealing with the psychology of esthetics are especially relevant to the use of great literature in psychiatric education (7–11). In contrast, the psychiatric literature on films is much less extensive. Munsterberg in 1916 (12) and Montani in 1938 (13) made early contributions to the esthetics of the cinema. Increasingly, psychiatrists are exploring commercial films and the work of individual filmmakers, reviewing them in depth from a number of perspectives (14–20). Schneider recently traced the history of movies' depiction of psychiatry and psychological themes (21). Didactic and documentary possibilities of film have long been recognized; the literature reflects psychiatry's and other health sciences' extensive experience with such films as audiovisual aids to education (22–27). However, reports of cinematic art being utilized in the training of psychiatrists or other mental health professionals are lacking.

Film has several advantages over literature as the artistic medium around which to build a teaching seminar. Of all the art forms, the motion picture seems to have the most direct access to the unconscious. Primary process thought utilizes visual images extensively, and part of the difficulty in adequately communicating primary process material stems from how poorly it lends itself to literary verbal language. Attempts to convey primary process in the vehicle of secondary process inevitably compromises the individuality and richness of the former even after it is retranslated into mental images in the mind of the receiver. Montani and Pietranera (13) noted that if it were possible "to transmit images from brain to brain, very often films would pass from man to man, and if the motion picture machine were so simple as the pen or as spontaneous as speaking, such would undoubtably occur." Insofar as psychotherapy entails frequent shifting between primary and secondary process thought, the viewing of films exercises that therapeutic skill.

In their nonverbal, visual imagery movies are closely related to dreams. Both utilize ambiguous images that are over-determined and multilayered in their function. To accomplish this, both movies and dreams employ the mechanisms of condensation and displacement. Film montage ignores the rules of time, size, and association to connect seemingly unrelated images; concrete similarities such as position and color express abstractions. Specific dream sequences in films, as exemplified in Bergman's *Face to Face,* as well as montage footage that is dream-like, as in Fellini's *Juliet of the Spirits,* have practical utility for the psychiatrist who seeks to help his patients through an understanding of their dreams.

Contemporary movies are technically far advanced compared with the early silent films. Today's directc have at their disposal almost unlimited technology well as actors and actresses who are artists in th own right. An increasing number of superbly talent artists are drawn to film as their medium because of tremendous potential. To think of film as art is no lo er to be restricted to the stereotype of a foreign f with subtitles shown in a small theatre where coffe served in the lobby. Pakula, Altman, and Kubrick, name a few of the growing number of creative and s cessful American filmmakers, have joined Bergm Truffaut, and Fellini in contributing serious works major consequence. Thus advances in cinematograp have led to an enlarged and varied pool of films fr which those interested in the art can draw. These sa advances are responsible for the uniquely mu sensory nature of most contemporary films. Tc screenplay that is often a significant literary achie ment on its own, auditory and visual elements are a ed. The rape scene in Kubrick's *Clockwork Oran* for example, is at once visually horrifying and m dane; the association of "Singing in the Rain" with violence emphasizes the tension arising from confl ing emotions. Contemporary cinema entails the pr essing of verbal, visual, and auditory input to tota experience the work. Intense concentration must complemented by flexible receptivity, and intellect awareness combined with emotional sensitivity, if c is to fully appreciate a film. Effective psychotherapy evaluation demands much the same process.

A final advantage of the cinema as the focus o seminar on psychodynamics in art has to do with pr ticality. The busiest resident can still find time to se film that will be discussed, when the task of readin novel would be impossible. Viewing a film, like re ing, is an individual experience, but a night at the m ies can be surrounded by social activities. The ne ness and rapidly changing nature of the cinema ma for few "experts" who can inhibit discussion or t the seminar into a passive learning experience. W films as with novels, many are made for profit or exploitation and have little artistic merit; fortunat the trend toward reruns of great films helps ensur regular supply of challenging works over time.

COURSE DESCRIPTION

Experience with a film seminar as part of the psyc atric residency curriculum in two area training p grams over the past five years has led to the exist format, which seems to best promote general c cussion and deep involvement with the films. The se inar is elective for residents in all three years of tra ing. Spouses and friends are included, which helps involve them first-hand in the training experience a also balances the traditional male perspective in p chiatry.

Several faculty members with whom the reside have sufficient contact to feel relaxed and familiar a attend. Meetings are held at the home of different p

cipants each time, and socializing over wine and nacks precedes the discussion. The seminar group sually meets monthly, the exact frequency being dermined by what films are running at the area eatres. Only one criterion is used to determine the itability of a given film for the seminar: it must sufficiently excite at least one member of the group that he she recommends it. Using this method, in combination with growing sophistication, has led to a series of vely discussions; mediocre experiences are rare. Frequently someone who was unimpressed by a film aves the seminar with an entirely different feeling, as e discussion provides new perspectives and insights. rongly negative reactions contribute greatly to the ow of the discussion and often to individual growth d self-awareness.

Early in the life of the seminar several facilitating chniques were used to stimulate or direct discussion. hese included 1) a series of questions prepared by the ader designed to relate details to the film as a whole, a short presentation by one member of his analysis the film, or 3) provocative reviews by major film itics. With time, however, group cohesiveness and embers' heightened appreciation of the film medium ade these techniques less crucial. Now penetrating estions arise within the group, and members are eager to contribute their own perceptions and analyses. terestingly, the familiarity and sophistication seem be group functions that are readily transmitted to ch year's incoming residents. It also became apparent that films are difficult to appreciate in detail because of their fast pace and multisensory input. Thus me participants see films two or three times before scussing them, perceiving new aspects with each ewing. A penlight and notepad are also useful during movie for recording impressions or questions. Reading the novel or even the screenplay is interesting for ntrast, but experience has underlined the obvious: e filmmaker's creation is unique and personal, and a eat film stands alone no matter how it is related to its urces.

In describing the literature seminar, Silberger (6) has dvocated limiting discussion to the psychology of individual characters for simplicity and thoroughness. ecognizing the difficulties involved in a broader ope, we have nonetheless found it useful to vary the cus of individual meetings. Thus the filmmaker's wn character and conflict, social or political issues at determine human behavior, philosophical positions as they affect one's life, and our own subjective dividual or group responses have all been emphazed in one or another discussion.

A combination of personal preferences and the nature of the film in question seems to determine the direction of the discussion. For example, *A Clockwork range* stimulated a discussion of the tension between ehavior control and human freedom in psychotherapy; *Deliverance* was discussed from the viewpoint of e four protagonists representing contrasting facets of e male personality; *Shampoo*, with its prominent featuring of Nixon and Agnew on TV, while the hero, George, is undone by his efforts to be everyone's friend, was discussed as a conscious/unconscious comment by Warren Beatty on the 1972 presidential campaign in which he was significantly involved; and Polanski's *The Tenant* was seen as offering psychodynamic insights through an externalized representation of the internal dynamics of paranoid schizophrenia. As a change of pace, the play *Equus* was discussed with actors from the American Conservatory Theatre of San Francisco in attendance; they added significantly to the discussion.

EVALUATION

To assess the value of such a seminar, questionnaires were mailed to the 36 residents to whom the seminar had been available during their training. Of the 31 residents who returned the questionnaire, 23 had attended more than half of the sessions and only 2 had never attended. Of those who had attended, all answered that they felt a cinema seminar was a valuable part of a psychiatric residency curriculum and would include such a seminar in a curriculum if they were designing it. The most common reasons given for the value of the seminar were that it helped to broaden the resident's perspective to include humanistic, social, and philosophical levels; it served to relate psychiatric insights to everyday life; it provided a useful opportunity for socialization with professional peers; it increased knowledge of unconscious dynamics; and it stimulated creative thinking.

COMMENT

Contemporary cinema, the vanguard of current artistic media, has great value for those learning psychiatry. Films provide an enjoyable, intense, and varied body of material without associated clinical responsibility. When psychiatrists in training or beyond want to comprehend the sources and form of humanistic imagination, integrate this with the directions of society in the 1970s, and share it all with those close to them, they are well advised to study the cinema.

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Pseudoepilepsy: A Study in Adolescent Hysteria

BY MEIR GROSS, M.D.

Of 19 adolescents with diagnosed psychogenic seizures, 13 had hysterical convulsions and 4 had amnesiac fugues. Sixteen of the patients were given a diagnosis of hysterical neurosis; 2, process schizophrenia; and 1, borderline psychosis. Thirteen of the patients were initially diagnosed incorrectly as having epilepsy and were treated for an average of 15 months with anticonvulsant medication. The therapist should always consider the possibility of psychogenic factors in children and adolescents who suffer from seizures.

THE NEUROLOGIST, especially the one who works with adolescents, may be confronted with the diagnostic dilemma of how to differentiate between real epileptic seizures and seizures that are psychogenic or functional in origin. The adolescent patient may complain convulsions associated with complete blackout a amnesia to the time of the spell, while the EEG do not show clear-cut evidence of epileptic activity shows only a nonspecific abnormality. The EEG mig show a nonspecific abnormality in a nonepileptic nc mal individual or hysterical patient. Should such a p tient be diagnosed as epileptic and treated with an convulsive medications? A decision to treat a patie for many years or for life with anticonvulsant medic tions is a very difficult one, considering the side effec of such medications, the restrictions on driving a everyday activities, the social stigma, unavailability jobs, etc. It is very important to rule out the possibili of epilepsy in psychogenic seizures, or so-call pseudoepilepsy. The correct diagnosis enables t adolescent who suffers from psychogenic seizures get the right treatment for the disorder and also i tensive psychotherapy to help him or her cope wi personal conflicts in a better and healthier way.

Psychogenic seizures are hysterical in nature. Th have components of dissociative reaction, conversi reaction, or both. Difficulties in diagnosing d

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ciative reaction or conversion reaction as hysterical urosis stem from the fact that the physician is not le to see the disease as part of the patient and does t recognize the patient as a whole person.

If the diagnostician concentrates only on the symptns, trying to classify them in an entity of a disease, or she is liable to misdiagnose the problem. The tient should be viewed and evaluated as a person. ıe symptoms should be weighed as to their value to ɜ patient and how the patient views the symptoms. ves he or she have indifferent feelings toward the seires (la belle indifference)? Is there any secondary in from the symptoms? Does the patient have an unrlying conflict that is being temporarily dealt with by veloping the symptoms? What is the symbolic eaning of the symptoms to the patient? What type of rsonality does the patient have, and what kind of fense mechanisms does he or she tend to use? Is the tient using denial, repression, and suppression as his her main repertoire of defense mechanisms? If the ınician will consider these questions when evaluating ıe patient, it is most likely that he will be able to corıctly diagnose the existence of hysterical seizures.

HE SUBJECTS

Within a period of 2 years, 19 adolescents suffering ɔm psychogenic seizures were diagnosed at the eveland Clinic. The 14 girls and 5 boys ranged in age ɔm 14–18 years (mean=15.79). In all but 2 of the paɜnts there was evidence of an underlying conflict. In ɔases one or both parents were overprotective, overɜmanding, and strict. One girl had been molested by ɜr stepfather, and another had been raped by her natal father while he was drunk. Two girls had recently ɜparated from their boyfriends. In 4 cases the parents ıd recently divorced. Two boys with low IQs were iling at school and felt hopeless about being able to ɔpe with their academic difficulties. One girl had not orked through her grief over the recent death of her other from breast cancer, and one boy had watched s mother's health deteriorate due to cancer.

Sixteen patients were given a diagnosis of hysterical ɜurosis; 2, schizophrenia; and 1, borderline psychos. Thirteen patients had histories of passing out, with ther convulsions or tremors. Four suffered from amɜsiac episodes, and 2 of these patients also had mulple personalities (1 had 2 personalities and the other ıd 3). One patient had spells of feeling "frozen," uttering, and hallucinating; 1 had fainting spells.

Thirteen of the patients were treated with antiɔnvulsive medications for 3–48 months (averge=15.54 months). Six were not given anticonvulsive edications since the proper diagnosis was made and pilepsy was ruled out. Twelve patients were treated y conventional psychotherapy, either insight oriented supportive; 5 of them also received neuroleptic or ıtidepressant medication. Seven patients were eated primarily by hypnotherapy.

DISCUSSION

From the description of these patients, it is clear that not all of them had hysterical neuroses; and if one would classify the personality patterns of these patients, not all of them would be categorized as having hysterical personalities. As Chodoff (1) noted, hysterical features appear in patients with other than hysterical personality types. Moreover, hysterical symptoms can be one of the signs of a developing borderline psychosis or schizophrenia, as seen in 3 of the patients. Rock (2) suggested that if the criteria for conversion reaction (as part of the hysterical neurosis) are not met, then the disorder should be called conversion symptoms or part of another syndrome or entity, such as schizophrenic reaction.

The female to male ratio in the present study was 14:5, almost 3:1. In his report on conversion symptoms, Engel (3) cited a ratio of 2-3:1. Since ancient times females have been more prone to hysterical neurosis.

Most of the patients in the present study were in late adolescence. Bernstein (4) has noted that this period is vulnerable to hysterical convulsions; it appears that the adolescent's turmoil is then at its peak. The struggle for self-identity and role differentiation is quite stressful for the adolescent. The adolescent is striving toward independence, while being afraid to give up the previous dependencies. The period of adolescence is one of changing behavior, lability of mood, and fragile, changing defenses (5).

The conflict area during adolescence can include separation from a boyfriend or girlfriend, grief, and divorcing parents. Passing out or change in level of consciousness during the seizure symbolizes the adolescent's need to get away from the conflictual situation. Change in level of awareness is the dissociative component of the seizure. The motor convulsion is the conversion reaction component, and it serves to release tension or anxiety. The mechanism is functional in origin and is created by the adolescent's need to use denial, repression, and suppression as the main defenses. The conflict is thus passed to the unconscious. Not one of the patients in the present study was malingering, since the process of producing the symptoms was completely unconscious.

Once the symptoms occur, there is a reduction in anxiety (the primary gain). Instead of expressing anxiety, the patient shows indifference to the symptoms. If the patient benefits from the sick role (e.g., gets more attention, is able to control the environment or manipulate family members), he or she may continue to take advantage of it (the secondary gain). Being sick helps the patient to regress and become dependent, which satisfies his or her dependency needs.

Thirteen of the patients were first diagnosed as epileptics. There was no evidence of epilepsy on any of the EEGs, but the treating physicians still considered the condition as organic and tried to treat it as such. Only after there was no response to treatment did they

refer the patients to the Cleveland Clinic's child neurologist, who requested psychiatric consultation. The first treating physicians showed denial of the possibility of functional problems in the patients. They tended to make diagnoses by exclusion and not by considering the patient as having an emotional conflict. Rock (2) also noted such attitudes by the treating physicians.

Diagnosis can be made using positive signs and symptoms, even without the elimination of organicity first. There is always the possibility of superimposed functional pathology in addition to physiological disorder (6). Hysterical convulsions should be suspected whenever there is no biting of the tongue and no fecal or urinary incontinence. Such seizures are less sudden in onset, and there is usually no bodily injury associated with them. Postconvulsive stupor or sleep is absent, and occasionally subjects may recall the event.

In 7 of the patients in the present study, hypnosis was also used in making the differential diagnosis. Schwartz and associates (7) noted that both epilepsy and hysterical convulsions can be activated by hypnosis and that the EEG of epileptic patients, but not hysterical patients, will show paroxysmal activity.

Schneck (8) suggested the use of hypnosis for the differential diagnosis of psychomotor epilepsy. He noted that convulsions can be induced during hypnotic trance; the epileptic patient cannot stop them by suggestion, but the hysterical patient can. Sumner and associates (9) noted that hysterical patients could recall what happened during their spells while in hypnotic trance but that the epileptic patients could not. They also noted that the hysterical patients tended to be more hypnotizable. The 7 patients with hysterical convulsions who were diagnosed by hypnosis were very hypnotizable, and all of them could recall the previous spells, including what had happened during them while the patients were under hypnosis (10).

Psychogenic convulsions are much more common than they are thought to be. Hysterical symptoms in children are considered rare and have been on the decrease since the rigid Victorian age (11).

Proctor (12), in a review of the literature up to 1957, noted that "there is no good bibliography available pertinent to childhood hysteria." Stevens (13) also mentioned the paucity of literature on conversion hysteria from about 1957–1967.

Is childhood hysteria as rare as one might assume from a review of the literature? It is my opinion that if more attention were paid to the possibility of childhood hysteria, it might be diagnosed more frequently. In our study 13 patients received anticonvulsant medications for an average of 15.5 months before the correct diagnosis was made, and it was made primarily because there was no response to the drugs.

Treatment of psychogenic seizures is needed to prevent fixation of symptoms. The untreated patient may hold on to his or her secondary gain, but also will be limited for life. The patient may prefer to see himself or herself as a real epileptic, will limit his or her activities, and regress in social contacts to the point of soc isolation.

It is important to reassure the child and the paren Usually the child will not accept the existence of a emotional factor in his or her condition. He or she (ten refuses to see a psychiatrist, and for this reason t first stage of therapy should focus on fostering rapp with the patient and building a good alliance that w be the cornerstone for further therapy. After the p tient's resistance is reduced, he or she will be mc ready to give up the pathological defenses. It is imp tant to reassure the parents about the very good pr nosis when the child becomes involved in therapy. the parents support the therapist it will be easier f the adolescent to give up the secondary gain, esp cially if he or she cannot manipulate them any mor

Treatment usually takes 3–6 months. The removal the seizures is not enough; follow-up is necessary order to help the patient solve the main conflict th caused the appearance of symptoms. Once the patie gives up the secondary gain he or she is more ready consider changing his or her defenses. The defenses children and adolescents are more labile than those adults and more rapid change is possible. Brief thera is usually sufficient; however, the more severe cas may require 6 months or more of therapy. In the pr ent study 6 months of therapy were enough to elir nate symptoms and enable the patients to devel healthier defenses and coping mechanisms.

CONCLUSIONS

Emphasis should be put on early diagnosis of p chogenic seizures; it is common to diagnose them c rectly only after treatment with anticonvulsant me cations has failed. With early diagnosis the progno is fairly good, since the symptoms are not yet fixa in the child's personality. If the emotional conflict dealt with in the first stages of the disorder, there less need for the patient to hold on to his or her s ondary gain. It is important that the practitioner mo from an organic point of view to an evaluation the child's whole personality and social and envir mental background. This will help the therapist making the right diagnosis early and in starting thera when it can still change ego defenses and improve t prognosis.

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hild Psychiatry Education for General Residents

JOHN F. GREDEN, M.D.

ere is continuing debate about what child chiatry experiences should be included in a general idency. The author describes the program at the iversity of Michigan in an effort to provide some ights into the interface between child psychiatry d general residency training. This program is que in several respects: a 12-month rotation in ld psychiatry is offered, and the faculty size and dget of the youth services are comparable to those he adult services. A survey of all residents and ulty pointed up numerous disagreements as to the gth of the rotation and priorities in curriculum. The hor discusses the influence of the various npetitive processes on the educational program.

HAT CONSTITUTES an ideal child psychiatry experie ce for general residents? This question has perxed generations of educators.

Although child psychiatrists began their fight for ognition as a distinct professional discipline more n 50 years ago (1, 2), and great strides have been made in some areas, in many educational programs the impact of child psychiatry in training general residents has been minimal. There are marked differences, both subjectively and objectively, in child psychiatry experiences among general residencies (3). Most programs give verbal support for learning child psychiatry, but in only a few is this actually translated into substantial didactic and clinical emphasis.

More than a decade ago Lawrence Kubie confronted this issue by advocating that *all* residents launch their training by studying infants and children (4). He also suggested that residents work with adolescents after the initial phase of learning child development, and only then would they evaluate and treat patients with neuroses and psychoses. As demonstrated by continued debate, Kubie's proposals did not resolve the problem. Had his suggestions been widely accepted, child psychiatry experiences for general residents would certainly be longer, more intense, and more influential than they currently are. Instead, it is still an exceptional residency that offers extensive child psychiatry experiences.

A program that has emphasized child psychiatry training for general residents is the one at the University of Michigan. Consequently, consideration of Michigan's program might provide some insights into the still-nebulous interface between child psychiatry and general residency training.

Several training factors seem unique to Michigan. Perhaps most important, from 1972 to 1977, almost all of the general psychiatry residents spent their entire second year on what is known as the youth service. The youth service at Michigan consists of child and adolescent programs. During the 12-month youth rotation, there is an emphasis on developmental theory, psy-

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chodynamics, inpatient and outpatient work with children and adolescents, and family therapy. The experience of the general residents is virtually identical to that of the first-year child psychiatry fellows. In essence, by the time the general resident at Michigan had completed the three-year program, he or she had also completed one-half of a child psychiatry residency. Most general residents have considered the experience educationally excellent.

A second unique feature is that the youth service is proportionately larger than that found in many other programs and is geographically distinct. With more than 50 faculty and 13 child fellows, the youth service budget is comparable to that of the adult service. The child psychiatry program at Michigan clearly does not lack visibility or strength. Perhaps this explains in part why a 12-month child rotation was initially instituted.

Since this program has been in operation for several years, it is now possible to assess it from a historical perspective, however brief. Soon after implementation of the program, many adult faculty members (perhaps predictably) began to criticize the length of the youth rotation. A major complaint was that with one-third of their residency being spent on youth services, general residents lacked the necessary time to learn basic principles of adult psychiatry. The residents themselves complained of having no time for electives. Thus a seeming paradox developed. Most of the adult faculty members acknowledged that general residents should learn child psychiatry. Most also acknowledged that the general residents *were* mastering child psychiatry exceedingly well. However, most simultaneously found themselves advocating a reduction in this apparently worthwhile experience. The beleaguered resident group frequently appeared to be caught in the middle of this argument.

To evaluate child psychiatry training for general residents, and to simultaneously collect data to assist the residency education committee in the formulation of an experiential core curriculum, a subcommittee was formed to assess this issue. A questionnaire was distributed to all faculty and residents. This paper briefly reports the subcommittee's findings, together with my subjective impressions of the issue, stemming from the perspectives of having chaired the subcommittee and later directing the general residency education program.

METHOD

In 1975 a 4-page questionnaire was distributed to various faculty and resident members at the University of Michigan Department of Psychiatry. Respondents were arbitrarily divided into 4 subgroups: adult-oriented senior faculty, general psychiatry residents, youth-oriented senior faculty, and youth fellows. The number of forms completed and the percentage of completion for each subgroup were as follows: adult senior faculty—26 (84%), youth senior faculty—16 (29%), youth fellows—10 (91%), and general psych try residents—21 (75%). A total of 73 questionnai were returned (a completion rate of 58%).

Respondents were asked to do two things: rate th attitudes about the child psychiatry rotation on a point Likert scale ranging from "strongly agree" "strongly disagree," and establish priorities for psychiatric approaches traditionally included wit general residencies. These approaches included dev opmental theory of childhood and adolescence, patient treatment of adults, outpatient evaluation children, outpatient treatment of children, outpati treatment of adults, drug therapy of children, dr therapy of adults, crisis intervention of adults, cri intervention of children and adolescents, treatment adults as parents, and youth inpatient treatment. Ea item was rated on a 5-point scale (1=highest prior and 5=lowest priority). The form emphasized that r ings should be for general psychiatry residents, r child fellows. We then used mean scores to compil sequential ranking of priorities.

RESULTS

Questionnaire Assessments

There was some agreement about priorities am child psychiatry faculty and residents and among ad oriented faculty and residents; however, there was a a great deal of disagreement. All 4 subgroups essentia agreed that the outpatient treatment of adults was very high priority (mean rating=1.3). In contrast to relative unanimity, child psychiatry faculty rated velopmental theory of childhood and adolescence h enough to place it second on their listing; child p chiatry fellows ranked it first. Adult-oriented fac and residents ranked it only fifth and sixth, respecti ly. Even more striking, adult faculty had a mean ority rating of 1.77 for drug therapy of adults, rank it second, and general residents ranked it third. A nificantly lower percentage of youth-oriented fac and fellows considered this an important item, h ever; they ranked it ninth and fifth, respectively. C patient evaluation of children was considered of m lower priority among adult-oriented psychiatrists t among child psychiatry staff and residents. Drug th apy of children and inpatient treatment of child received the lowest ratings and subsequent rankin among all 4 subgroups. In summary, 3 of the firs priority items among youth-oriented faculty pertain to child psychiatry. General residents had no you oriented items among their first 5 items, and adu oriented faculty had only 1 (teaching developmer theory).

When asked about the ideal duration for a yo services rotation for general residents at Michig 71% of the adult-oriented faculty and 60% of the gen al residents said that the current rotation of 12 mon should be shortened. None of the youth-oriented f ulty expressed this sentiment; indeed, 20% felt tha

ar was inadequate and that the rotation should be ngthened.

bjective Impressions

These questionnaire assessments provide only a rtial story of child psychiatry training for general sidents at Michigan. Since this simple study was nducted in 1975, there have been periodic deliberions about changing the rotation. As I observed and rticipated in these debates, I was struck by several ctors. First, the issue of child psychiatry training for neral residents often became confounded with anher ideological dispute, namely, analytic psychiatry rsus biological psychiatry. The child residency segent seemed to have been identified with psychonamic education, and many adult service segments ith "medical psychiatry." Whether these observaons were correct scarcely seemed to matter when ttle lines were drawn.

A second major observation was that child psychiasts who defended the length of their rotation were ncere in their belief that this was best for the general sidents. Service considerations seemed minimally portant. Most youth-oriented faculty and fellows arly believed that the best way to prepare for a caer in general psychiatry was to have extensive exsure to child and adolescent psychiatry. However, I ted that adult-oriented faculty members just as fernly believed that residents should have a shorter uth-oriented rotation. Many enthusiastically pleadfor greater exposure to such topics as substance use, geriatric psychiatry, forensic psychiatry, neuchemistry, neuroendocrinology, aftercare of chronic tients, consultation with their medical colleagues, d psychiatric research.

ISCUSSION

This assessment of the role of child psychiatry traing in the general residency program at the University Michigan perhaps demonstrates that the issue will t be resolved through simple expansion of child psyiatry rotations. The experience has value for other ograms. Historically, child psychiatrists have had to ercome territorial guarding to teach their principles general residents. In some departments their victory s been only partial. At Michigan, however, the child ychiatry segment of the department has truly atned recognition, and a different explanation must be rmulated to explain current conflicts.

I have concluded that the process is loosely comrable to a marital conflict involving issues of dirce. Unfortunately, the marital feud often includes a spute over the custody of the children. In divorce sputes the child is commonly an innocent victim (5). milarly, in this educational dispute the resident bemes an innocent victim. To carry the analogy fur-er, adult- and youth-oriented faculty seem to disree about such issues as what the resident should read, what clothing he or she should wear (such as a white coat), where he or she should "go to school," what ideology should be professed, and where the resident should spend the majority of his or her time.

In their influential publication *Beyond the Best Interests of the Child* (5), Goldstein and associates concluded that it was important to protect the child's relationships with at least one parent during a divorce. At the risk of oversimplifying a complex educational issue, I would suggest that a similar need exists among psychiatry residents in solving disputes about youth-oriented experiences in their training. Both the child of divorcing parents and the psychiatry resident in an educational dispute should be able to identify one major decision maker. For *general* residents, the role of "parent" should be filled by *general* psychiatrists. It is clear that the advice and consent of child psychiatry staff is needed to compile the best program. We cannot return to the days of resistance to child psychiatry training for general residents. Nevertheless, if we keep intact the ultimate question of what a resident is being trained for, it seems that a mini-version of a child residency is not in their best interests.

In my opinion, their best interests are served by the integrated learning of those parts of child psychiatry which enhance their treatment of adults and older adolescents, rather than those which provide skills in the treatment of children or young adolescents per se. In considering this, it appears that the program laid forth by the adult-oriented faculty in the present survey is the most acceptable. Adult outpatient treatment, crisis intervention, and psychopharmacology would head the list of priorities, but emphasis would also be given to developmental theory of childhood and adolescence. Somewhat lower on the list but still of medium to high priority would be working with adults as parents, outpatient evaluation of children, and crisis intervention with youths. Drug therapy of children and inpatient treatment of children and adolescents would be low-priority items, and in a crowded curriculum they would be excluded if time were unavailable. (As pharmacotherapy of children becomes more thoroughly investigated, however, this priority may have to be reconsidered.) Certainly no one can dispute the fact that any general resident would profit from a rotation on a child inpatient unit. Nevertheless, in time-limited residencies continued consideration must be given to the ultimate question of what knowledge, skills, and attitudes are most important at the completion of residency. I do not believe that inpatient treatment of children should be included on this list.

If the previous analogy can be continued for one final step, my ultimate wish is that the "parental disagreement" cease. For this to occur, agreement will have to be reached in some fundamental areas. Since ideological differences inevitably will continue, we need to intensify our assessment of what residents ultimately do or should be doing in the treatment of patients (6, 7). If adult- and youth-oriented psychiatrists cannot agree on what psychiatric residents should do

after training, they certainly will not agree about what has the highest priority during residency. If reasonable agreement were reached, the next question would be whether residents have the knowledge, skills, and attitudes to do what is required. Using such information, an irreducible experiential core curriculum could be devised to stipulate the educational experiences in child psychiatry that every resident should have to practice general psychiatry. One could then attempt to answer such difficult questions as How many children need to be evaluated? What ages should they be? When should these evaluations be done? Should normal families be followed? What is the role of drug therapy for children?

Considering the lack of attention given to child psychiatry generations ago, it is amazing that a custody battle could actually be developing in certain programs throughout the country. Nevertheless, this appears to be the case. In his extensive dissertation on traditionalism, Kubie (8) noted that once a program becomes operational it is difficult to modify. He attributed this resistance to "the hold on seniority rights which no one ever gives up willingly." At one point in psychiatric history, this pattern probably prevented child psychiatry from assuming its rightful place in educational programs for general residents. Data from this study suggest that the opposite can also occur. Child psychiatrists have the potential to become traditionalists, too. If this is to be prevented, we must remind ou selves what the educational end product of our effo should be, namely, a competent general psychiatr with a thorough understanding of developmental the ry and family dynamics, with fundamental skills f child evaluation and diagnosis, and with adequate tin to master the multitude of other areas now proven be relevant to the treatment of adult patients.

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LINICAL AND RESEARCH REPORTS

is section contains 1) new research findings, including preliminary data from pilot studies, either clinical or boratory; 2) worthwhile replication studies; 3) case reports that describe a truly new syndrome or cast new ht on established ones; and 4) case reports that indicate a new therapeutic procedure of potential value or call tention to adverse effects of drugs or previously unreported complications of therapeutic interventions. Pro- am descriptions and literature reviews cannot be printed in this section. Criteria for format are listed in "Infor- ation for Contributors" in each issue; papers that do not adhere to these criteria will be returned to the author.

cute Extrapyramidal Reactions with Fluphenazine Hydrochloride and Fluphenazine ecanoate

ALAN J. GELENBERG, M.D., JOANNE C. DOLLER, R.N., M.S., NINA R. SCHOOLER, PH.D., ARLENE MIESKE, R.N., M.S., JOANNE SEVERE, AND MICHAEL R. MANDEL, M.D.

Extrapyramidal symptoms (EPS) have been report- to occur in 20%-43% of patients treated with oral enothiazines (1–3) and are probably even more com- on with the piperazine phenothiazines (3, 4). For er a decade, two long-acting injectable piperazine enothiazines, fluphenazine enanthate and deca- ate, have been available for the treatment of schizo- renia. The incidence of EPS with the fluphenazine ers has been reported to be approximately 50% (5), t Ayd has stated that the long-acting fluphenazines use no more EPS than oral antipsychotics (6).

In this paper, we will present an analysis of EPS that occurred during the acute-treatment phase of a collaborative study which compared fluphenazine hydrochloride with fluphenazine decanoate. Two questions will be addressed:

1. Are there differences in occurrence of EPS between oral and depot fluphenazine?
2. Are patient characteristics such as sex, race, or age related to differential occurrence of EPS?

Method

The design of this four-center collaborative study has been described in detail elsewhere (7). In brief, 290 male and female schizophrenic inpatients, ages 18–55, entered the study when they were experiencing a psychotic episode. After they gave written informed consent, all patients were treated for 1 week with oral fluphenazine HCl in dosages that reached at least 15 mg by day 3. Patients were then randomly assigned in blind fashion to one of two groups and given continued oral fluphenazine or fluphenazine decanoate. Each patient ingested tablets twice daily and received an injection at least every 3 weeks. The fluphenazine HCl group received placebo injections and the decanoate group took placebo tablets. Dosages were adjusted according to clinical response and without a fixed ratio between oral and injectable fluphenazine. Doses ranged from 5 to 60 mg/day for oral fluphenazine and 12.5 to 100 mg every 3 weeks for injections. Benztropine mesylate, up to 6 mg/day, was used only if clinicians thought it was necessary to control EPS.

Assessments. After initial baseline screening, including psychiatric diagnosis and history, patients were rated by a research psychiatrist and a nurse at weeks 1, 4, and 7. The doctor and nurse each independently completed the Dosage and Treatment Emer-

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gent Symptoms Scale (DOTES) of the Early Clinical Drug Evaluation (ECDEU) battery (8). The present report will deal only with 1) DOTES items that reflect EPS—increased or decreased motor activity, rigidity, tremor, increased salivation, and akathisia, and 2) data on age, sex, race, history of EPS, and previous use of antiparkinsonian drugs. Signs and symptoms are rated on the DOTES by a 4-point scale ranging from 0 (not present) to 4 (severe).

Statistical analysis. Chi-square analyses (using Yates' correction for continuity where appropriate) were computed between treatments (oral or depot) for each symptom as rated by both the physician and nurse at weeks 1, 4, and 7. Eleven patients were removed from treatment because of side effects; since these cases could have affected the results, chi-squares were calculated both including and excluding them.[1] Analyses were also done for three symptom clusters from the DOTES: 1) increased motor activity and akathisia (the akathisia cluster), 2) decreased motor activity, rigidity, tremor, and increased salivation (the parkinsonian cluster), and 3) a central nervous system cluster (8). Finally, correlations between each of the EPS and age, race, sex, history of EPS, and history of antiparkinsonian drug use were examined.

Results

At the end of the first week of the study, when the double-blind phase was initiated, the mean initial dose for patients in the oral fluphenazine group (N=147) was 24.4 mg/day (SD=10.2). The mean initial injection in the decanoate group (N=143) was 33.6 mg (SD=13.7). At the end of the fourth study week, the mean oral dose was 24.2 mg/day (SD=13.4, N=139), and the mean injection was 35.5 mg per three weeks (SD=19.21, N=134). By the end of the seventh week, the mean oral dose was 24.2 mg/day (SD=14.8, N=122), and the mean decanoate dose, 35.1 mg per three weeks (SD=19.5, N=118).

Analyses of symptom differences between the two groups revealed a few scattered treatment effects ($p \leq .05$), but none that was significant for both doctors' and nurses' ratings at the same rating period and none that remained over any of the periods. This was true whether or not the 11 patients who dropped out because of side effects were included in the data analysis.

Based on analyses of physicians' ratings in the cluster groups, 21.2% of patients on oral medication suffered from akathisia at week 1, 19.5% at week 4, and 25.2% at week 7. By contrast, 22.7% of decanoate patients showed akathisia at week 1, 22.7% at week 4, and 27.5% at week 7. These differences are not statistically significant. A comparable analysis of the parkinsonian cluster revealed that 27.4% of the patients on oral therapy showed parkinsonian signs at week 1, 41.5% at week 4, and 31.9% at week 7. Parkinsonian signs were present in 23.4% of decanoate patients week 1, 25.7% at week 4, and 35.5% at week 7. Agai differences were not statistically significant. Similar analysis of the CNS cluster revealed no statistica significant differences between treatments. Corre tions between doctors' and nurses' ratings were co sistently high and statistically significant.

The following EPS were correlated, in both phy cians' and nurses' ratings, with benztropine usage: A ter the first treatment week of nonblind oral fluphen zine, 57% of the patients were receiving benztropin and akathisia and rigidity were significantly associat with benztropine prescription. At week 4, in the o fluphenazine group, use of benztropine (70% of the p tients) was associated with decreased motor activi rigidity, and tremor, while among the decano group, use of benztropine (64% of the patients) w correlated with akathisia and rigidity. By week 7, al thisia and rigidity were associated with benztrop use (74% of oral and 69% of decanoate patients) both forms of fluphenazine. Abnormal involunta movements were associated with *less* benztropine u

Analyses of demographic data demonstrated no s nificant relationship between EPS in this study and p tients' age, race, sex, history of EPS, or previous an parkinsonian drug use.

Discussion

We found no significant differences in the occ rence of akathisia or Parkinson's syndrome within t first 7 weeks of drug therapy between patients trea with fluphenazine decanoate and those treated w fluphenazine HCl. In addition, we found no correlat between the occurrence of any of the signs of th two disorders and age, sex, race, history of EPS, previous antiparkinsonian drug use. Benztropine u was significantly related to the presence of EPS, as have reported earlier (7). However, since this tre ment was based on clinical indication, no conclusi can be drawn about the effectiveness of benztropir

Our data are essentially in agreement with Ay finding that the incidence of EPS did not differ sign cantly between patients on fluphenazine decano and those on oral preparations. (It is conceivable t the enanthate preparation may be associated witl higher incidence of EPS, as has been reported [5], we did not assess this.) We also did not find a high cidence of akinesia, as measured by decreased mo activity, although we were not looking for some of more subtle behavioral manifestations that Rifkin a associates subsumed under the term "akinesia" 10).

In summary, data from this study suggest that, spite their relatively high incidence, akathisia a Parkinson's syndrome are not serious problems dur the first 7 weeks of treatment with either fluphenaz HCl or fluphenazine decanoate. Furthermore, long-acting ester did not produce more EPS than oral form.

[1]Other patients who did not complete the study were removed because of failure to improve or for administrative reasons.

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Hypnopompic Hallucinations and Treatment with Imipramine

BY ROBERT SCHLAUCH, M.D.

Hypnopompic hallucinations occur during the owsy state just before awakening and are thought to e related to hypnogogic hallucinations, which occur st before one falls asleep. The hallucinations are usuly visual, although they may also be tactile or audiry, are often well-formed, and appear to be coming ward the individual. They occur in normal individuls, but their incidence is difficult to determine beuse of the general reluctance to report hallucinaons. However, evidence suggests that most people ill experience hypnogogic phenomena at some time their lives (1).

Although hallucinations as a result of therapy with icyclics were described during the first decade after e introduction of these drugs, little note of this phenomenon has been taken recently. Recent reports ave dealt largely with psychoses resulting from atronic side effects of tricyclic antidepressants. I wish to ll attention to tricyclic-related hallucinations that do t result from toxicity. This phenomenon may have levance to the therapeutic effects of these drugs as ell as factors in compliance.

ase Report

Ms. A, a 61-year-old woman, was referred to me because depression of 2 years' duration. She had been taking amiptyline, 50–100 mg h.s., for 6 months without significant nefit or side effects.

Ms. A had an obsessive-compulsive personality with a pical endogenous depression. She complained of insomnia, crying spells, worry, anxiety, preoccupation, and agitation. She had no psychiatric history before her depression and had never hallucinated. Her father was an alcoholic, but there was no other mental illness in the family. Both parents and her brother had died 25 to 30 years before. She had a healthy 68-year-old sister. Neither of her two daughters had significant mental illness.

I prescribed imipramine, 150 mg h.s., and after 6 weeks Ms. A described herself as "1,000% improved"; she had returned to her normal affect and functioning. However, at this time she began to awaken about once a night, between 2 and 4 a.m. Initially, she saw her bedroom bathed in a red glow, although she knew it should be dark. She saw one to three people around the room, dressed in white. Some were strangers and others were known to her.

She became quite frightened when these figures came toward her, and she would close her eyes, "turn away or go to sleep." If very frightened, she would "wake up." Once she spoke to her husband in Polish "so that they wouldn't understand me."

Ms. A suspected the imipramine had something to do with these frightening experiences. She considered stopping the medicine but confided in her daughter, who convinced her to tell me about it. Her dose was then decreased to 100 mg h.s. without change in her affect, but the hallucinations persisted and even increased from about two to four a week. Therefore, after 6½ months, the dosage was changed to 50 mg q.a.m. The hallucinations stopped within 3 days, but after 3 weeks her depression had returned. I again placed her on 150 mg of imipramine h.s. and the hallucinations began after 3 days, followed by gradual disappearance of her depression.

At the time her hypnopompic hallucinations began, the patient was given a complete medical evaluation, including physical examination, chest X ray, ECG, urinalysis, blood chemistry, electrolytes, CBC, T_3, T_4, and FTI. All were normal except an ECG that showed incomplete right bundle branch block.

After she had been taking imipramine for 10 months, the frequency of her hallucinatory experiences had decreased to about once a week. Six months later her hallucinatory experiences had decreased to about one a month, and the imipra-

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mine was again reduced to 100 mg h.s. However, within 2 months, her depression had returned (she had had one hallucinatory experience) and she was again given 150 mg of imipramine, h.s. Within 1 month she was again well and at the time of this writing, 2 years after imipramine therapy was initiated, she remains well and experiences hypnopompic hallucinations about once a month. At no time during her treatment has she experienced disorientation or excessive anticholinergic symptoms.

Discussion

This case demonstrates a clear relationship between the onset of hypnopompic hallucinations and imipramine therapy in a patient without a history of hallucinatory or hysterical symptoms.

In an extensive review of visual hallucinations with imipramine, Klein (2) summarized seven reports in which most patients who hallucinated were on high-dose imipramine therapy (250–400 mg/day). In his own study, 12 of Klein's 209 patients developed hallucinations. Nine of these were schizophrenic and 11 of the 12 were taking 300 mg/day of imipramine. The 12th patient developed hallucinations after 6 days on a dose of 150 mg/day. Klein noted no evidence of "confusion approximating an organic mental syndrome" in his patients, who did not differ significantly in age, sex, or diagnosis from the rest of his sample. In 9 of the 12 patients, the hallucinations were dose-dependent; decreasing the drug to 150 to 200 mg/day terminated the phenomenon.

I believe that several factors hamper our understanding of the occurrence of hallucinations in patients taking imipramine. First, there has been a failure to differentiate the various types of hallucinations, which include 1) hallucinations due to anticholinergic side effects, 2) hallucinations in psychotic individuals or those with a history of hallucinating, and 3) hallucinations in individuals with no evidence of psychosis, hysteria, or organicity.

In addition, the fact that these phenomena are hypnopompic or hypnogogic has not been attended to sufficiently. It is known that imipramine increases stage 4 sleep (3) and is associated with an increased incidence of stage 4 phenomena (4, 5). Because hypnopompic hallucinations are a stage 4 phenomenon it is reasonable to expect them to occur with imipramine therapy.

The antidepressant effects of REM deprivation and the REM-depriving effect of imipramine have be well noted (6). Vogel (7) has postulated that this RE deprivation is the primary mode of action of the t cyclic antidepressants.

However, since depression is associated with an i creased frequency and duration of wakefulness and marked decrease in stage 4 sleep (8), this sleep sta may play an important role as well. This case stu shows a clear correlation between imipramine dosag recovery from depression, and stage 4 sleep phenom na. Is it not possible that enhancement of deep sleep therapeutic for many depressives but is observable only scattered cases?

In addition to suggesting a mode of action of imipr mine, hypnopompic hallucinations may also play role in patient noncompliance. Most people are frigh ened by hallucinations, and should a patient correct associate his medicine with his hallucinations he mig assume he was getting worse and therefore disconti ue the drug. If he does not discontinue therapy alt gether, he might still be reluctant to report the hallu nations to his doctor. Psychiatrists should be aware hypnopompic hallucinations and question patien about them if this appears indicated. Patients shou be reassured to learn that the hallucinations are b nign, occur less often with prolonged therapy, a mark improving rather than deteriorating men health.

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ncidence and Morbidity of Self-Induced Water Intoxication in State Mental Hospital atients

"Y C.J. JOSE, M.D., AND J. PEREZ-CRUET, M.D.

The syndrome of self-induced water intoxication is a stinct clinical entity that occurs almost exclusively in sychiatric patients. The syndrome consists of comulsive water drinking associated with symptoms of ater intoxication. Rountree's classic paper on water toxication (1) enumerated the symptoms as follows: stlessness, asthenia, polyuria, frequent urination, dirrhea, excess salivation, nausea, retching, vomiting, uscle tremor and muscle twitching, ataxia, tonic and onic convulsions, frothing at the mouth, helpssness, stupor, and coma. Since the first case report ppeared in 1938 (2), there have been relatively few ports of psychiatric patients with this syndrome. owever, the general impression is that the overall indence of self-induced water intoxication in psychiatc patients is much higher than the number of pubshed reports would suggest. This underreporting may flect the transient nature of the syndrome or lack of vareness or interest in this clinical condition. We ve investigated the incidence of water intoxication the long-term-care psychiatric population of a large idwestern state mental hospital in a metropolitan ea; we are not aware of any previous studies on the cidence and morbidity of water intoxication.

ethod

Nursing and medical staff of the nine long-term psyiatric wards of a state hospital were asked to provide e names of patients they thought were drinking wa r in excessive amounts. These patients were interewed and their charts were reviewed in detail for obctive evidence of compulsive water drinking and wa r intoxication. A patient was diagnosed as a mpulsive water drinker if 1) the staff of the ward and e patient both felt the patient drank water in excesve amounts or 2) the staff felt the patient was drinkg excessive amounts of water and there was docuented evidence of this in the patient's chart. Water toxication was diagnosed if the patient or the chart dicated a history suggestive of cerebral impairment sociated with excessive water consumption.

esults

Of the 239 patients surveyed, 16 (6.6%) had a consisnt history of compulsive water drinking; of these, 8 (3.3%) had symptoms of water intoxication. In addition to these 8 patients with self-induced water intoxication, data were obtained from 4 other patients from the same institution who were not included in the survey because they were not living in the long-term psychiatric wards at the time the survey was carried out.

The mean age of the 12 patients was 42 years (range, 22–54 years); 8 (67%) were female. Ten of the 12 (83%) carried a diagnosis of schizophrenia; the other 2 were mentally retarded. The most common mode of presentation of water intoxication was generalized seizures. Nine of the patients (75%) had a total of 16 incidences of water intoxication with seizures. Of the remaining 3 patients, 1 was found semiconscious following excessive water drinking and the other 2 had only the minor symptoms of nausea, vomiting, dizziness, and lightheadedness.

The clinical diagnosis of water intoxication was confirmed by measures of serum electrolytes in 8 patients (serum electrolyte determinations had not been done for the remaining 4 patients). A low serum sodium level was a consistent finding in these patients (mean=121 mEq/liter, range=110–131 mEq/liter, normal range =136–145 mEq/liter). Only 1 patient was investigated for the syndrome of inappropriate secretion of antidiuretic hormone, and the findings supported this diagnosis. The patient was a 43-year-old man with a diagnosis of paranoid schizophrenia who showed natriuresis (urine sodium=48 mEq/liter) and high urine osmolality (506 mOsm/kg H_2O) despite his low serum sodium (110 mEq/liter) and low serum osmolality (230 mOsm/kg H_2O). Renal and adrenal functions were normal and he did not show evidence of dehydration. Thus, all of the criteria for the diagnosis of inappropriate secretion of antidiuretic hormone (3) were fulfilled.

All 12 patients recovered without any apparent sequelae, despite multiple episodes in some cases. The disability lasted from a few hours to a few days.

Discussion

The present study supports the general impression that the incidence of water intoxication in psychiatric patients is much higher than the number of reports in the literature would suggest. Why only certain psychiatric patients with compulsive water drinking develop water intoxication is not fully understood. We have reviewed the syndrome of inappropriate secretion of antidiuretic hormone in relationship to psychiatry (4) and feel that at least in some cases of water intoxication, the inappropriate secretion of antidiuretic hormone induces the symptoms.

The present study also showed that the treating phy-

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sician is not always aware of the syndrome. In some cases, the diagnosis had been delayed for years and the patients had undergone expensive and intrusive investigations and received prolonged, unnecessary anticonvulsive medications for seizures of undetermined origin that were almost certainly due to water intoxication. Electrolyte estimates at the time of the seizure in those patients with a history of compulsive water drinking are essential in establishing the proper diagnosis of water intoxication.

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Insanity Plea: Legislators' View

BY RICHARD A. PASEWARK, PH.D., AND MARK L. PANTLE, M.A.

Although philosophic discourses on the plea "not guilty by reason of insanity" (NGRI) abound in the psychiatric and legal literature, there have been few empirical studies, most of which have examined the characteristics and disposition of persons who enter the plea. There have been, to our knowledge, no studies directed toward the beliefs held by individuals or groups about the plea.

The present investigation surveyed state legislators in Wyoming to ascertain their estimates of the incidence and success rate of the insanity plea in Wyoming and their beliefs about certain procedures that govern the operation of the plea.

Method

A questionnaire was mailed to all members (N=92) of the Wyoming state legislature in December 1977. The return rate was 35% (N=32). Although this is a minority return, we think that the responses provide some information on legislators' knowledge of the plea. Of the respondents, 27 were male, 3 female, and 2 did not specify their sex. Five were attorneys and 24 were not (3 respondents did not indicate occupations). Thirteen reported that they or members of their immediate families had been the victim of a crime at some time. The types of crimes they reported ranged from murder (one case) to petit larceny (five cases).

Results

In estimating incidence of the NGRI plea and its success rate, respondents were told that during the period considered (1970–1972) there were 21,012 felony indictments in Wyoming (1). The legislators' mean estimate of the number of persons who pled NGRI was 4,457.84, with a median of 3,025.50 and a range of 18,567. The mean estimate of the number of perso adjudicated NGRI was 1,794.28 (median=501.0 range=0–13,600). In fact, 102 defendants made th plea and only 1 was so adjudicated.

Consistent with respondents' overestimation of t frequency and success rate of NGRI plea was their sponse to the statement, "The insanity plea is used t much. Too many people escape responsibility f crimes by pleading insanity": 15 respondents agre strongly, 11 agreed somewhat, 2 disagreed somewh and 2 disagreed strongly.[1]

Asked whether "there is ever a situation in which person accused of a crime should be excused from sponsibility for it because of mental illness," 21 leg lators responded affirmatively and 10 negativel Thus, a large majority of legislators expressed agre ment with the basic philosophy underlying the NG plea.

As a group, the legislators were even more empha in rejecting the notion, as proposed in Illinois and N York, that acquitted NGRI defendants should treated in the same fashion as convicted individua Only 4 respondents agreed that "if a person has be found not guilty by reason of insanity to a crimi charge . . . the consequences and/or punishm should be the same as for a person found guilty of same crime who is not mentally ill"; 26 disagreed.

Respondents indicated a fairly good awareness procedures involved in the NGRI plea. Twenty-six the legislators indicated that either the jury or judge was responsible for the ultimate decision o defendant's sanity; two believed psychiatrists m the decision, and one thought it was the county torney. Most legislators were also aware of the d position of acquitted NGRI defendants. Nineteen

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[1]Not all respondents answered each question, so response freq cies varied.

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cated that the person is mandatorily committed to e state hospital until he/she is considered sane, nich is the requirement of a Wyoming statute. Ten ated that the NGRI patient was hospitalized for a pulated time; and 1 expressed the belief that the in-vidual was placed under the jurisdiction of the pro-tion department.

mment

Our results indicate that Wyoming legislators ossly overestimate the frequency and success rate of e NGRI plea in the state and seem to believe the fense is overused. However, a large majority still cribes to the basic philosophy of the NGRI plea. We spect the widespread publicity given by the media to e more notorious NGRI cases contributes to legisla-s' gross overestimates. Although it would be easy decry the lack of knowledge of legislators in this sphere, we believe that the blame rests with members of the mental health and legal professions. In this belief, we are influenced by the written statements of a number of legislators who returned uncompleted questionnaires with remarks indicating that their task was to promulgate law, while ours was to provide them with the appropriate data. Considering the extreme paucity of objective information concerning the NGRI plea in the literature, we find ourselves forced to agree with this position, but we hope that this deficiency will be rectified through systematized studies of the NGRI defense.

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eudonursing: A Case of Galactorrhea

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Galactorrhea, or inappropriate lactation, has been cognized for centuries. Within the past decade, med-l investigators have reexamined this condition and lineated syndromes according to the presence or ab-nce of menstrual dysfunction and alterations of rum prolactin. Although prolactin secretion is regu-ed by the brain (1), and this hormone is known to luence behavior in lower animals (1), little is known out the psychological correlates and/or etiologic role emotional factors in galactorrhea. The following se report is prompted by the highlighting during ne-limited psychotherapy of the unique psycho-ysiologic aspects of galactorrhea. In contrast to the ore traditional psychosomatic diseases, this case of eudonursing, like those of pseudocyesis, proved to unusually responsive to a relatively superficial ex-oration of dynamics and underlying depression.

se Report

A 26-year-old woman was seen for seven sessions as part a comprehensive psychoendocrinologic evaluation of per-tent postpartum lactation. A planned and largely uneventful pregnancy had been followed by the delivery in October 1976 of a healthy first-born male child. Breast feeding was experienced as mutually pleasurable and was used to comfort and pacify dysphoric feelings in both mother and child. The patient continued to nurse despite her return, at 3 months postpartum, to her position as a pediatric radiology technician and an exhausting trip, 7 months postpartum, to her sister's university graduation. According to the patient's journal this trip marked the onset of a sexual dysfunction characterized by diminished libido, anorgasmia, and decreased coital frequency. At 10 months postpartum she developed uterine cramps, her intrauterine device was removed, she resumed oral contraception, and discontinued nursing. Her son had become at this point "a very independent child . . . so active that he broke his playpen . . . and seemed on the verge of weaning himself anyway." At 20 months postpartum, her galactorrhea persisted and she sought an endocrinologic evaluation. Her other complaints were the sexual disturbance, neurasthenia, and heightened irritability with her child and husband. Physical examination showed expressible milk in one breast, which did not decrease with a trial off of the oral contraceptives. The serum prolactin was normal (5.1 ng/ml). Formal visual field testing, sellaturcica polytomography, computerized tomography, and other pituitary and thyroid hormone levels were also normal. Psychiatric referral was made to investigate links between her physical condition and emotional life.

The patient had a brother 4 years older and a sister 6 years younger. She was the acknowledged favorite of her father, an alcoholic laborer, and the confidante of her mother. The mother, a chronically unhappy woman, was locked in a sexually impaired and vituperative relationship with her husband. Interestingly, she too had developed galactorrhea, which lasted for 20 years after the birth of the patient's sister.

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The patient spent a lonely childhood; she was afraid to invite friends over because of her parents' marital discord and her father's drinking. Despite intermittent periods of depression that began in high school, she excelled in her studies. She met her fiancé during a bout of depression in her sophomore year of college and married him following graduation. Until recently, their 5-year marriage had been a happy one, with a satisfactory sexual relationship and significant progress toward attaining financial security.

A short-term therapeutic contract was established. After the initial session she reported a dream in which a child, tethered to an I.V. bottle, accidentally jarred the pole. As it tottered and began to fall the patient desperately strove to catch it.

A poem she wrote 15 months postpartum contained the following thoughts:

I lie here in the dark
wondering if I can let him go

I gave up breast feeding him long
before I was ready to
That was the beginning
I miss the closeness, his
dependency on me

Every day now he's a little
less mine and a little more of
himself

It's sad to be a mother

In the second session she reported a lessening of her irritability. By the third session the flow of milk had diminished noticeably, and it disappeared totally by session 5. By session 6, she noted a return to her usual sexual functioning. Her generalized improvement continued through termination of this therapy and referral for long-term, insight-oriented therapy.

Discussion

This case recalls Helen Deutsch's observation that breast feeding cushions the impact of the physical separation that takes place at birth (2). The key unconscious conflict for this mother was the longing for reunion, the desire for separation and liberation, and the fear of losing everything. The exploration of these dynamic issues, manifested in the dream, poem, and associational material, led to a rapid resolution of her depression and symptom amelioration.

In the psychiatric literature abnormal lactation has been discussed chiefly in connection with pseudocyesis. In one review galactorrhea was present in 76 of 202 cases (3). Analytic case reports of patients with combined pregnancy delusions and galactorrhea also emphasize the paramount importance of separation/dependency issues, citing even premature termination of treatment as a potential precipitant (4, 5).

Galactorrhea occurs in 12%–46% of women of productive age, although fewer than 1% will be av of it and/or seek relief (6). Causes include pitui adenomas and neuroleptic medication, but the si largest category, typified by this patient, is idiopa galactorrhea with normal menses (7). Two-third these cases follow delivery and in 86% the basal lactin level is normal (7). The condition is endocr logically indistinguishable from that found with longed nursing. It has been postulated that the bre in these normoprolactinemic women are more se tive to prolactin, presumably due to an alteratio receptors (8). The present case suggests that this be corrected by psychotherapy.

Increasing information regarding the bioamine and peptidergic control of hypothalamic and pituit hormone secretion and the role of these substance brain function provides an opportunity to reexam the psychiatric and neuroendocrine aspects of same event (9). Since its isolation in 1970, prolac the phylogenetically oldest pituitary polypeptide h mone, has been demonstrated to play a significant in the premenstrual, pseudocyesis, and galactorr syndromes (1). Bromergocryptine, hypophysector and psychotherapy have all been associated with r malization of elevated serum prolactin levels (10), psychiatric evaluation and treatment should prove be of major importance in the future study of th complex psychopathophysiologic phenomena.

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ychological Correlates of Preferences for Clitoral or Vaginal Stimulation

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A popular misconception is that Sigmund Freud was ponsible for positing two separate and distinct es of orgasms. For example, Offer and Simon (1) erred to "Freud's statement concerning the dif- ent kinds of orgasm," Sherfey (2) cited "the vaginal gasm as defined by Freud . . .," and Brecher and echer (3) stated, "Sigmund Freud presented, in ree Essays on the Theory of Sexuality, a theory that men can experience two kinds of orgasm—one cli- al, the other vaginal." However, Freud did not dis- ss either clitoral or vaginal *orgasms;* rather, he pro- sed that 1) in early childhood the female's chief togenic zone is the clitoris, 2) during puberty the male must adopt the vagina as the leading or domi- nt erotogenic zone, 3) fixation on clitoral preference presents childish masculinity (4–6), and 4) fixation clitoral preference may result in orgasmic dysfunc- ns (sexual anesthesia) during coitus (5, 7).

It is obvious, then, that Freud's thesis, which sug- sted a psychological basis for preferential stimula- n of the clitoris or vagina preceding orgasm, was torted by the assumption that there existed distinct ysiological correlates of such stimulation. When ch physiological distinctions were later disproven , Freud and his thesis received the misdirected ver- assaults. This is unfortunate, since there do exist finite psychological preferences for either clitoral or ginal stimulation as a precursor to orgasm (9, 10). rther, it is possible that these preferences are re- ed to important psychological differences.

This study is intended to provide some much- eded data in an area that is top-heavy with theory. accord with Freudian theory, I hypothesized that males who prefer clitoral stimulation are more mas- line or less feminine and that they experience fewer gasms during coitus than do those who prefer vagi- penetration. I also tested the following popular hy- theses: females who prefer clitoral stimulation 1) are re neurotic, 2) are less assertive, 3) have engaged re often in coitus, and 4) have had fewer and/or less joyable emotional and physical relationships.

The subjects were 306 nonvirgin unmarried women o volunteered to complete an anonymous, con- ential four-part questionnaire. Students were ap- ached in cafeterias and libraries. Nonstudents were solicited in medical hospitals (staff, patients, and visitors), a ski lodge, and three businesses located in different parts of the city. The sample included 187 college and university students (mean age=19.3 years) and 119 nonstudents (mean age=27.0 years). The nonstudent group included such professions as barmaid, freelance writer, hairdresser, librarian, model, private investigator, professional golfer, telephone operator, teacher, and tour coordinator. The most common occupations were secretary/typist/stenographer (16.9% of nonstudent sample), clerks (accounting, bank, or mail—16.8%), technicians (laboratory, medical, or research—9.2%), and nurses (5.9%).

Each volunteer completed a 5-item questionnaire regarding her sexual history, the Rathus Assertiveness Scale, the Eysenck Neuroticism Scale, and the McGill Gender Role Inventory.

The five sexual history items were as follows: 1) emotional relationship with opposite sex (none, unsatisfying, satisfying), 2) physical relationship with opposite sex (none, unsatisfying, satisfying), 3) sexual intercourse (never, occasionally with one person, frequently with one person, with more than one person), 4) coital orgasm (never, sometimes, frequently), and 5) most pleasurable part of lovemaking (clitoris stimulated by hand or mouth, penis within the vagina).

The Rathus Assertiveness Scale is a 30-item self-report inventory. The respondent indicates how characteristic each item is of her on a 6-point scale ranging from "very characteristic of me" (+3 points) to "very uncharacteristic of me" (−3 points). Nonassertive items are then subtracted from assertive items, so that positive scores reflect assertiveness and negative scores reflect nonassertiveness.

The Eysenck Neuroticism Scale is a 24-item self-descriptive checklist with yes-no answers. High scores reflect neuroticism.

The McGill Gender Role Inventory consists of 20 items. Each is rated on a 7-point scale ranging from "never or almost never true" (1 point) to "always or almost always true" (7 points). There are 10 masculine items and 10 feminine items, which are summed separately.

The sample was divided into those who preferred clitoral stimulation during lovemaking (99 students, 47 nonstudents) and those who preferred penile intromission (88 students, 72 nonstudents). Two-factor analyses of variance with unweighted means were used to analyze ordinal data. The two factors were sexual preference (clitoral, vaginal) and student status (student, nonstudent). The nonstudent group was significantly older than the student group, $F(1, 302)=193.7$, $p<.0001$, but the clitoral and vaginal pref-

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erence groups did not differ in age and there was no significant Age×Student status interaction.

There were no significant effects or interactions for neuroticism or assertiveness. The students were more feminine (mean=45.1) than the nonstudents (mean =43.1), $F(1, 302)=6.98$, $p<.01$. Those who preferred clitoral stimulation tended to be more masculine (mean =43.4) than those who preferred vaginal stimulation (mean=41.7), $F(1, 302)=3.61$, $p<.06$.

Questions regarding sexual history were subjected to chi-square analyses with Yates' correction as necessary. More of the women in the clitoral preference group had never had an orgasm during coitus (18.5%), and fewer of them reported having frequent orgasms than those in the vaginal preference group (6.9% versus 46.5%), $\chi^2=9.58$, $p<.01$. When students and nonstudents were analyzed separately, more clitoral preference students reported never having orgasm during coitus (21.2% versus 8.0%), $\chi^2=6.45$, $p<.05$.

In addition, the ratio of vaginal to clitoral preference was larger in the nonstudent group, $\chi^2=5.27$, $p<.05$. It is difficult to interpret this finding precisely, but it could imply that vaginal preference develops with maturity, age, or coital experience. Since there was no relationship between amount of sexual experience and clitoral vs. vaginal preference, a maturational factor is more likely to be responsible, which would lend some support to Freud's transfer theory.

The results lend modest support to Freud's hypotheses that a woman who prefers clitoral stimulation to vaginal penetration tends to be more masculine and experiences fewer orgasms during coitus. However, one may explain the results in less pejorative terms than did Freud. For example, the reason that females who prefer clitoral stimulation attain fewer orgasms during coitus is not necessarily that they are obstinately clinging to childish masculine sexuality. It may very well be that women have less opportunity for direct clitoral stimulation in coitus, and those who pre such stimulation will be less satisfied and find orga more difficult to attain. Likewise, the masculinity a sociated with clitoral preference need not be regr sive. It may well represent a mature ability to ma one's preferences known despite contrary social pr sure. Although this assertiveness is more typica "masculine," it is now regarded by many as a mc mature response for the woman than it was in the pa The mechanism whereby such differences emerge mains to be more fully examined; however, the pr ent results do indicate that, despite physiological sir larities of orgasms produced by clitoral or vagi stimulation, there are psychological differences t tween those who prefer clitoral and those who pre vaginal stimulation.

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Depression and Psychotic Regression Following Prolonged Methylphenidate Use and Withdrawal: Case Report

BY ALVIN A ROSENFELD, M.D.

Methylphenidate is used widely in treating minimal brain dysfunction. Remarkable symptomatic improvement is seen in some cases, while little effect is noted in others. Few adverse reactions have been reported (1, 2). There is debate over whether the drug caus growth retardation. There have been rare reports tics (3), psychotic episodes from initiation or abuse the medication (4), and choreoathetosis (5). The *Phy cians' Desk Reference* states that "careful supervisi is required during withdrawal, since severe depressi as well as the effects of chronic overactivity can unmasked." There are few reports of these sympto in the literature. Although depression following lo term amphetamine use is widely known, such a sy

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rome has not been described with methylphenidate se in children.

ase Report

Allan A, a 9-year-old boy, was first evaluated because of epression, despondency, and suicidal ruminations. Allan ad been taking methylphenidate for over 3 years because of minimal brain dysfunction." His parents stated that with- it the medication Allan was "wild" and "uncontrollable," nning around the house, climbing trees, swinging from ings recklessly, and doing nonstop cartwheels in the family om.

Allan was the product of Mrs. A's first marriage. From rth, Allan seemed to be overactive, cried incessantly, and as "inconsolable." He was said to have celiac syndrome d several other allergies, but dietary change did not affect e situation. Mrs. A was anxious, upset, and depressed er her seeming inability to alleviate Allan's continual rest- ssness and crying.

When Allan was 2½, Mrs. A separated from his father, aving Allan with a relative for a few weeks. She later met d married Mr. A, a corporate executive.

When Allan was 5, Mr. and Mrs. A took him to a child ychiatrist for evaluation. The diagnosis was "hyperactive ndrome of childhood with psychoneurotic symptoms and rsonality traits." The hyperactivity diagnosis appears to ve been made on behavioral grounds, such as impulsivity d poor peer relationships at school. The psychoneurotic mptoms were said to be environmental.

After he was started on methylphenidate treatment, Al- n's behavior improved markedly. His mother felt that for e first time he was not uncontrollable. His school work mained excellent for the next several years, and his dis- ractability, impulsivity, and poor peer relationships im- oved. His IQ was in the very superior range, and he was an cellent athlete. His parents tried to stop the methyl- enidate on several occasions but noted each time that Al- n became uncontrollable.

When Allan was 8, Mr. and Mrs. A again requested a psy- iatric evaluation because Allan "hates himself and wants die." In addition, he was irritable, had frequent temper trums, and became inconsolable after even minor errors. lan was a very bright but depressed boy. He was sad, in- ibited, controlled, and somewhat suspicious in his play. Al- ough he wished to stop taking the methylphenidate be- use he felt it indicated there was something wrong with n, he also said he liked it because when he misbehaved, he uld always say he had forgotten to take his medication. ychological and neurological assessments were essentially rmal. Twice weekly psychotherapy was recommended to lp Allan with his depression, his poor self-esteem, and his fficulty getting close to classmates. Although the neurolo- st thought the methylphenidate was indicated by Allan's story, the psychiatrist decided that once an alliance had en formed with the family the methylphenidate should be ered and, if possible, stopped.

In the first months of therapy, Allan's play was inhibited d constricted, and he expended much effort to do things rfectly. He became overwhelmingly upset at the slightest ror. He insisted on competitive games but became ex- edingly frustrated and sulky if he lost. When anxious, he ached his genitals repetitively or rushed to the bathroom.

After 2 months of therapy, his parents stopped the methyl- enidate abruptly without consulting the physician. Allan remained in control for 7 days, but the next day became aggressive at school and punched another child. When seen in therapy that day, Allan was extremely overactive, ran from item to item, jumped on cabinets, and was unable to pay attention to anything for more than a few seconds. His speech was pressured and marked by frank paranoia. He felt his mother had numerous spies watching him at school and thought the therapist might also be a spy. Much of his play was exceedingly primitive. At one point, he formed an elongated piece of clay, placed it around his rectal area, and let it drop into a toilet as he laughed wildly. He then picked the clay up, placed it over his genital area, called it a penis, and had it break into little pieces, laughing in an uncontrollably anxious way. He was oriented, and no hallucinations were noted. It was thought that he was severely regressed and functioning at a psychotic level.

Allan was seen daily or every other day for the next week, and the therapist was in regular contact with Allan's parents. Over the next week, the psychosis slowly cleared. Although he was transiently more difficult to control, there was an added vivacity to his personality (his therapist noted that Allan had had a "zombie-like" quality previously).

With support, Allan's parents have been able to manage the home situation quite well and he has shown only mild behavior problems there. His school performance has continued to be excellent for the 10 months since methylphenidate was discontinued and he has made more friends. However, it is clear that he suffers from serious anxiety over issues of separation and loss. This anxiety is currently manifested at times of threatened loss by attention-seeking overactivity, "running away" games in therapy, sadism and guilt, and mild risk-taking. There has been no further depression or suicidal ideation.

Discussion

In this case, a severe depression was seen in an 8-year-old boy who had been given prolonged methylphenidate treatment. When the drug was withdrawn, a transient disorder of psychotic proportions appeared after 8 days, lasted about a week, and resolved spontaneously. This may be an isolated phenomenon or something others have seen but not yet reported. This case may suggest that the increased activity noted in some cases of methylphenidate discontinuation may result not from underlying minimal brain dysfunction or the unmasking of "chronic overactivity" but from a specific withdrawal phenomenon.

This case highlights the difficulty in making a diagnosis of hyperactivity. Further, it raises the possibility that the combination of an overwhelmed and depressed mother and a child with severe separation anxiety might lead to a mistaken diagnosis of "hyperactive child syndrome." Methylphenidate responsiveness may not be a good indicator of the difference between the correct and the mistaken diagnosis.

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Tardive Dyskinesia and Antiparkinsonian Medication

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Tardive dyskinesia, a neuroleptic-induced extrapyramidal hyperkinetic syndrome, is characterized by involuntary, repetitive, and purposeless movements that vary in localization and form. Common sites of involvement include the mouth, lips, tongue, and jaw, and, at times there are choreoathetoid movements of the neck, trunk or limbs (1). In a recent study of tardive dyskinesia in 261 schizophrenic outpatients undergoing long-term maintenance treatment with neuroleptics, we reported that 31% had tardive dyskinesia and that patients with tardive dyskinesia had fewer parkinsonian symptoms, particularly of the hypokinetic type, than those without tardive dyskinesia (2). These results suggest a balance between parkinsonian and dyskinetic symptoms. The present study was undertaken to investigate further the relationship between tardive dyskinesia and parkinsonism in schizophrenic patients receiving long-term maintenance treatment with neuroleptics. We examined the effects of the administration and subsequent withdrawal of the central anticholinergic (antiparkinsonian) agent procyclidine HCl on the extrapyramidal symptoms of 20 patients with significant tardive dyskinesia.

Method

Subjects. Twenty chronic schizophrenic patients (10 men and 10 women) were selected from the long-term wards of Hôpital Louis-H. Lafontaine, Montreal. Selection criteria required that patients 1) had received long-term maintenance neuroleptic treatment for at least 5 years, 2) had not taken antiparkinsonian drugs for at least 3 weeks before the study; and 3) had significant tardive dyskinesia (a score of at least 5 on a 9-point clinical global impression scale of tardive dyskinesia. All patients with physical illness, neurologic disease, alcoholism, epilepsy, or drug addiction were excluded from the study. All subjects completed the week trial (1 week with procyclidine and 1 week without procyclidine). The patients' ages ranged from 34 73 years (median=59), and their total length of hospitalization was 5.6–39.8 years (median=28.3).

Drug administration and assessment procedure. Patients were given procyclidine HCl (Kemadrin) 30 m day (10 mg t.i.d. in 5-mg tablets) for 1 week and the the drug was stopped for 1 week. All patients except were receiving neuroleptics at the time of the trial. T neuroleptic dose, which in chlorpromazine equivalen (3) ranged from 20 to 1850 mg/day (median =400 m mean=560 mg), was kept constant during the study p riod. One patient required a reduction (240 chlo promazine units) and 2 patients required increases (1 and 200 chlorpromazine units) in their maintenan neuroleptic medication during the trial, but for the p tients in question these were relatively minor change No other psychotropic drugs, hypnotics, or min tranquilizers were given during the trial. Patients we rated on Chouinard and Ross-Chouinard's extrapy midal symptom rating scale (ESRS) (4) before and a ter procyclidine treatment (days 0 and 8), and again week after withdrawal of procyclidine. The ESRS w completed independently by two psychiatrists (G. and C. de M.) during the same interview and a fir rating was made by consensus.

Results

Table 1 shows the mean scores for the ESRS befo and after 8 days of procyclidine treatment. Me scores at day 8 were compared with those at day using the paired t test. Procyclidine treatment led to significant decrease in the mean total score for parki sonism (excluding nonacute dystonia). Howeve there were significant increases in the mean scores f dyskinesia of the jaw and other involuntary dyskine movements and a tendency toward higher total scor for tardive dyskinesia. There was a negative correl tion that approached significance ($r=-.45$, $p=.06$) b tween the changes in total scores for parkinsoni symptoms and dyskinetic movements, indicating th patients with increased scores for dyskinetic mov

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BLE 1
ean Scores for Extrapyramidal Symptom Rating Scale Before and ter 8 Days of Procyclidine Treatment (N=20)

xtrapyramidal Symptom	Day 0	Day 8	p*
rkinsonism objective examination			
Total score	6.3	3.4	<.05
yskinetic movements			
Lingual	3.8	4.1	
Jaw	3.1	4.0	<.05
Bucco-labial	2.6	2.6	
Truncal	1.6	1.9	
Extremities	3.6	3.6	
Other involuntary movements	0.9	2.0	<.05
Total score	15.5	18.1	<.10

aired t test (2-tailed).

ents tended to have decreased scores for parkinson-m. One week following withdrawal of procyclidine a rther evaluation of these patients revealed that the ean total scores for parkinsonism and dyskinetic ovements had returned to baseline levels (scores of 6 and 14.5, respectively). A random sample of 10 of ese patients assigned to placebo in a study com-ring deanol and placebo was reevaluated 1 month ter withdrawal of the antiparkinsonian, and the ex-apyramidal symptoms remained stable at baseline vels.

scussion

The present study shows that the central anti-olinergic agent procyclidine had an effect on tardive skinesia opposite to that on parkinsonism, improv-g the latter but exacerbating the dyskinetic move-nts. These findings, which are in agreement with re-rts by Fann and Lake (5) and De Fraitas and associ-es (6), are consistent with the dopamine super-nsitivity hypothesis of tardive dyskinesia (7). Indeed, ocyclidine may have excerbated dyskinetic symp-ns by its dopamine reuptake blocking property and/or its central anticholinergic activity, thus ex-gerating a dopaminergic-cholinergic imbalance (9). s there is evidence that early signs of tardive dyski-sia may be reversible (10), uncovering the syndrome use of a central anticholinergic (antiparkinsonian) ug may aid in its early detection and prevention. The esent study demonstrates that the exacerbation of dive dyskinesia by the short-term administration of ticholinergic drugs is readily reversible, so the pro-dure does not appear to be harmful to the patient.

We have observed that the hypokinetic parkinsonian symptoms of rigidity and akinesia often act to reduce or cover the expression of dyskinesia (2). By alleviating hypokinetic symptoms, central anticholinergic drugs could help to prevent the masking of tardive dyskinesia by neuroleptic-induced hypokinesia. Early discovery of tardive dyskinesia in this way might lead to its reversal by means of a timely reduction in antipsychotic dosage. On the other hand, it is possible that the hyperkinetic parkinsonian symptoms of tremor (especially pill-rolling) and akathisia are precursors of hyperkinetic symptoms of dyskinesia (2). Hyperkinetic parkinsonism may coexist with dyskinetic movements in patients who are at the stage when their symptoms are progressing from one syndrome to the other. One way to avert the development of severe tardive dyskinesia may be to prevent patients from developing severe hyperkinetic parkinsonian symptoms by reducing their neuroleptic dosage at the first signs of hyperkinesia. We therefore propose that uncovering tardive dyskinesia by short-term administration of an antiparkinsonian may aid in early detection and help to prevent the subsequent development of incapacitating forms of the disorder.

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Alleviation of Manic Symptoms with Catecholamine Agonists

BY WALTER ARMIN BROWN, M.D., AND BARBARA MUELLER

In its simplest form the catecholamine hypothesis of affective disorder postulates that depression is associated with a functional depletion of catecholamines at central synapses and mania with a functional excess. Accordingly, the following pharmacologic agents might be expected to increase the severity of mania: L-dopa, which increases brain levels of catecholamines; apomorphine, which stimulates postsynaptic dopamine receptors; and methylphenidate and *d*-amphetamine, which release and block the reuptake of catecholamines. However, in the course of a study involving these drugs the two patients who received them during a manic episode experienced transient alleviation of manic symptoms.

Observations

Two physically healthy men, aged 54 and 46, with well-documented histories of mania and depression participated in a neuroendocrine research study for which they had given informed consent. They had been taking no medications other than lithium. Within 1 week after a double-blind switch from lithium to placebo they became manic, with marked hyperactivity, pressured speech, and elated, irritable, labile mood (episodes similar to those they had experienced in the past). They were the only subjects in the study who became manic after the switch to placebo.

After they had been given placebo for 10 days they received L-dopa (500 mg p.o.), apomorphine (0.5 mg s.c.), methylphenidate (20 mg p.o.), and *d*-amphetamine (15 mg p.o.) in a random sequence on a double-blind basis. Each drug was given at 10:00 a.m. on a different day following an overnight fast. Patients were observed continuously during the hour before and 2 hours after drug administration and intermittently during the rest of the day.

Neither patient showed an increase in mania after the administration of any of these drugs. In fact, about 45 minutes after the administration of each drug, the patients' rates of speech and gross body movement approached normal levels, and their comments indicated that they felt more relaxed. These effects lasted for about 3 hours, after which the full-blown manic syndrome recurred. About 45 minutes after administratic of each drug one patient dozed intermittently for 3 minutes. *d*-Amphetamine seemed to be more effecti than the other drugs in alleviating manic symptoms.

Discussion

These observations are entirely consistent with th only other study we know of in which *d*-amphetamii was administered to manic patients. Beckmann ar Heinemann (1) gave *d*-amphetamine (30–50 mg I.V.) six manic patients. They assessed the patients by usi the Beigel-Murphy scale before *d*-amphetamine i fusion and at 30, 90, 150, and 270 minutes after i fusion. All six patients experienced suppression their manic symptoms, and three of them showed tendency to fall asleep. These changes were reflect in their mania scores, which showed a significant d crease 30 minutes after infusion. Their scores i creased thereafter, reaching initial values appro mately 3 hours after infusion. These authors point out that *d*-amphetamine significantly reduced euphoi symptoms but not aggressive symptoms. A placebo fect has not been definitively ruled out in either o study or the one of Beckmann and Heinemann, but t temporal pattern of alleviation and recurrence symptoms is consistent with the pharmacodynamics these drugs. These data in the aggregate indicate th mania is certainly not worsened and is likely to transiently alleviated by *d*-amphetamine.

Janowsky and associates (2) observed mixed clinic changes following intravenous infusion of meth phenidate (0.5 mg/kg) to 10 acutely manic patients. Dopa has been shown to induce hypomania in so bipolar manic-depressive patients, and *d*-amphetami and piribedil (a dopamine receptor stimulating age have been shown to induce mania rarely in such p tients (3, 4). These findings and those we have repo ed are difficult to reconcile with current thinking (i. the catecholamine hypothesis) on the pathophysiolo of mania. Although reduction of mania following l doses of dopamine receptor agonists might be accou ed for on the basis of presynaptic inhibition, such mechanism cannot account for the antimanic effe we have noted with L-dopa and methylphenidate a we and others have noted with *d*-amphetamine. Phy stigmine, a central cholinesterase inhibitor, has a been found to alleviate manic symptoms (5). Do mine agonists have been shown to have anticholiner effects, so it is not likely that the results we have no are mediated through increased cholinergic activ (6). However, in view of data suggesting that cholin gic stimulation can activate catecholamine systems

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ıd of the findings suggesting that catecholamine gonists reduce mania, it is possible that the effects of ıysostigmine on mania are mediated by central cate-ıolamine pathways.

The fact that L-dopa and tricyclic antidepressants ın precipitate mania in some bipolar manic-depres-ve patients is certainly consistent with the hypothe-s that an "excess" of catecholamines at central syn-ses can have some role in inducing mania. These ıta, however, along with ours and those of Beck-ann and Heinemann showing rapid transient alleviа-ın of manic symptoms with *d*-amphetamine, would em to indicate that the induction, maintenance, and eviation of mania involve different and complex uronal processes that are not limited to shifts in the ailability of catecholamines at central synapses. hile other investigators have pointed to clinical, armacologic, and physiologic observations in af-ctive disorder that cannot be accounted for by the techolamine hypothesis (7), this hypothesis contin-s to dominate and possibly restrict our thinking. Al-ough it has had great heuristic value and demon-rated usefulness, slavish acceptance of the cate-olamine hypothesis in the absence of confirmatory ta can limit further progress in our understanding of affective disorders and impede the development of novel effective treatments.

The rapidity of the response to catecholamine agonists noted in this and the Beckmann and Heinemann study suggests the possible usefulness of these drugs in the treatment of mania.

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edical Histories of Psychiatrically Referred Delinquent Children: An Epidemiologic udy

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The purpose of this paper is to report differences tween the medical histories of a group of delinquent ildren recognized by court personnel as requiring ychiatric evaluation and the medical histories of eir nonreferred court-involved peers. In a previous per (1), we reported that a random sample of delin-ent children known to the juvenile court had signifi-ntly more numerous and serious medical problems an a matched sample of their nondelinquent peers. that study, we chose to compare non-psychiatrical-referred delinquents with nondelinquents to avoid ewing our findings toward pathology. We wondered, wever, whether objectively ascertainable differ-ces existed between the medical histories of two oups of delinquent children, one referred for psychiatric evaluation and one never referred. If there are significant differences that indicate specific vulnerabilities in psychiatrically referred delinquents, the greater professional attention given the referred group would be justified. A finding of no significant differences between the groups would suggest that psychiatric referral was a chance happening and that most court-related children, psychiatrically referred or not, require professional attention.

We have previously reviewed the current literature on the medical status of delinquent children (1). To summarize, while some investigators have reported an association between perinatal and/or neurologic disorders and subsequent maladaptation (2–7), others have found little if any relationship between ill health and delinquency (8, 9).

Method

A consecutive sample of 65 delinquent children referred to a court clinic for psychiatric evaluation in the early 1970s was matched for age, sex, race, geographic area, and, as far as could be ascertained, socioeconomic status with a sample of 65 non-psychiatrically

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TABLE 1
Medical Problems of Psychiatrically Referred Delinquent Children and a Matched Sample of Nonreferred Court-Involved Children*

Medical Problem	Psychiatrically Referred Children		Non-Psychiatrically Referred Children		Analysis	
	N	%	N	%	χ^2	p
Perinatal difficulties	15	37.5	4	10.0	8.352	.00
Child abuse	9	13.8	2	3.1	4.866	.02
Accidents and injuries through 16 years	50	76.9	37	56.9	5.873	.01
Head and face injuries through 16 years	32	50.0	21	32.8	3.896	.04
Head and face injuries before age 2	7	10.8	2	3.1	2.984	.08
EEG through age 16	9	13.8	2	3.1	4.752	.03
Skull X rays through age 16	13	20.0	6	9.2	3.020	.08
Seizures through age 16	4	6.2	0	0	4.127	.04
Parental psychiatric treatment	22	33.8	10	15.4	5.969	.01

*Percentages are based on the actual numbers of subjects for whom information was available.

referred children known to the juvenile court during the same period of time. One-third of the subjects in both groups were girls; 44% were black and 56% white. The average number of offenses was 10.5 for clinic-referred children and 2.92 for non-referred children ($t=2.34$, $p=.02$). Thus the clinic-referred children were significantly more delinquent than the nonreferred children. They tended to have committed more serious offenses (i.e., offenses against persons) and to be younger at the time of their first offense.

Medical histories were evaluated by reviewing hospital records from the main general hospital serving the area. The choice of this data source and its limitations and advantages have been discussed in detail previously (1, 2). As in our previous studies, records were assessed in terms of numbers of hospital contacts, reasons for contacts (e.g., accidents, illnesses), and timing of medical events.

In a previous study, the prevalence of past or present psychiatric treatment in state facilities of the parents of delinquents was obtained (10). These objective data on parental mental health were also used in the present study.

Results

Psychiatrically referred delinquents did not differ significantly from their nonreferred peers in terms of numbers of hospital contacts. Both groups made frequent use of all services.

As can be seen in table 1, a significantly greater proportion of psychiatrically referred delinquent children had experienced perinatal difficulties, child abuse, and injuries, specifically head and face injuries, through age 16. The severity of injuries is indicated by the greater proportion of referred children who had received EEGs and skull X rays. Furthermore, a greater proportion was recognized as having seizure disorders. Differences between the two groups were evident by age 2 years.

It is noteworthy that a significantly greater proportion of psychiatrically referred children had a parent known to have received psychiatric treatment at one of the major state facilities serving the area (33.8% versus 15.4%).

Discussion

Our previous epidemiologic studies indicated th delinquents as a group suffered from more adve medical histories than their nondelinquent peers. T present study shows that those delinquents referred a psychiatric clinic affiliated with a juvenile court h even more serious medical histories than did their n referred peers.

The nature of the problems found in the clinic- ferred group is of special importance. These child were significantly more likely than the nonrefer group to have experienced perinatal difficulties, ph ical abuse, and head and face injuries. Furtherm many of the clinic-referred group of delinquents h experienced documented head and face injuries dur early life, with significant differences noted by ag years. The parents of clinic-referred delinquents w a more psychiatrically disturbed population than parents of nonreferred delinquents.

These findings have special implications for all cl cians evaluating children for court purposes. It wo seem that referral by probation staff for psychia evaluation is not a random happening. Those de quent children for whom evaluations are sought t to come from the most psychiatrically disturbed fa lies and to have experienced the greatest amount early CNS trauma, including perinatal difficult head and face injuries, and child abuse. The delinqu child referred for psychiatric evaluation is, therefo especially likely to be both psychologically and ph ically vulnerable to maladaptation if appropriate di nosis and treatment are not initiated.

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ardiovascular Safety of Rapid Treatment with Intramuscular Haloperidol

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Haloperidol and other neuroleptics administered intramuscularly at close intervals can provide rapid control of the severe symptoms of acute psychotic decompensation (1–5). How safe is the procedure? In this study we investigated the effects of rapid treatment with intramuscular haloperidol on heart rate and blood pressure in acutely psychotic patients.

Method

The subjects of this study were 25 recently hospitalized schizophrenic adults (16 men, 9 women) in good physical health. The patients' mean age was 26 years. Approximately three-fourths of them (N=19) had had symptoms of schizophrenia for less than 3 months, the remainder for somewhat longer. Written permission for treatment in the study was obtained from the patients after they were informed of the risks and possible benefits.

Each patient was assessed clinically and psychometrically. Patients were then given an intramuscular injection of haloperidol, 2.5 to 10 mg, depending on the severity of symptoms. Injections were repeated every 0 minutes if necessary after the therapeutic response nd vital signs were evaluated. Vital signs were recorded before treatment and every 30 minutes for 6 ours following the first injection. Cardiac function as monitored closely in 6 of the patients by means of he Holter monitor. Laboratory tests conducted before nd after treatment included complete blood counts, MA-12 analysis, serum electrolytes, urinalysis, and etermination of values for serum creatinine phoshokinase (CPK).

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Results

During the 6-hour study period, the mean total dose of haloperidol administered to the patients was 22.5 mg (range=10–40 mg). Of the total of 145 injections given, 65 were 2.5 mg, 72 were 5 mg, 6 were 7.5 mg, and 2 were 10 mg. All patients completed the study.

Analysis of variance indicated that there were no significant reductions in mean values for blood pressure and pulse rate; both decreased gradually within the normal range (table 1). These decreases paralleled the calming effects of haloperidol and its control of core symptoms of psychosis, including thought disturbance. (We plan to write a separate report on therapeutic results in this study.)

Only one patient, a 36-year-old woman who weighed 54.5 kg, had a clinically significant blood pressure decrease. Her blood pressure decreased from a pretreatment value of 100/60 mm Hg to 90/50 mm Hg after she received the first dose of haloperidol, 5 mg I.M. It then decreased to 80/50 mm Hg after a second injection of 5 mg was given 30 minutes later to control severe symptoms. Pulse rates in this patient were 80 beats per minute (bpm) before treatment and 72 bpm after both injections. Although she did not have symptoms of hypotension, parenteral treatment was stopped to avoid further decrease in blood pressure. Her blood pressure returned to pretreatment levels approximately 6 hours after the initial injection.

Holter monitor recordings indicated abnormal heart rates with sinus arrythmia in 2 of the 6 patients. In both cases, the cardiac abnormalities were present at baseline recordings and did not worsen during treatment. One of these patients had sinus arrythmia and an episode of syncope and was subsequently diagnosed as having mitral valve prolapse. Because such phenomena are part of the symptomatology of mitral valve prolapse, the role of haloperidol in this case, if any, is not clear. The patient, a 24-year-old man weighing 70.4 kg, had a grade III/VI systolic murmur before treat-

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TABLE 1
Mean Blood Pressure (mm Hg) and Pulse Rate (bpm) of 25 Patients Treated with Injectable Haloperidol (10–40 mg)

Time Lapse (minutes)	Systolic Pressure		Diastolic Pressure		Pulse Rate	
	Mean	SD	Mean	SD	Mean	SD
0	122	17	79	12	88	15
30	121	18	77	12	84	15
60	116	24	77	12	84	17
90	120	18	79	12	86	18
120	123	17	79	11	87	20
150	120	14	78	9	85	18
180	118	17	77	9	82	18
210	119	18	77	11	80	17
240	119	17	75	10	80	17
270	118	16	77	11	81	15
300	117	15	76	11	80	14
330	115	16	75	10	79	16
360	114	12	75	11	81	16

ment but had been allowed to participate in the study after his case was reviewed by a cardiologist. The patient had a brief episode of syncope about 3½ hours into the treatment period after he had received a total of 37.5 mg of haloperidol. Blood pressure and pulse rate, taken immediately, were 164/74 mm Hg and 60 bpm. The patient did not lose consciousness completely, and his subsequent clinical course was uneventful. The Holter monitor recording for this patient indicated sinus arrythmia with abrupt changes in heart rate; syncope was preceded by tachycardia (130 bpm) that shifted rapidly to bradycardia (50–55 bpm). These changes in heart rate, however, were similar to other changes that occurred before as well as during his haloperidol treatment. The diagnosis of mitral valve prolapse was established by echocardiogram the day after the episode of syncope.

The second patient with abnormal Holter monitor recordings was also a 24-year-old who had abrupt changes in heart rate, e.g., 83 to 45 bpm in 2 beats. He had also had sinus arrythmia before treatment with haloperidol, and it did not worsen.

Holter monitor recordings in the other four patients were unremarkable. Results of laboratory tests were also unremarkable. Values for CPK were characteristically elevated before treatment because the patients were psychotic (6). These values were elevated further (300–2541 IU) by the trauma of intramuscular injection.

Comment

The results of this study indicate a high degree of cardiovascular safety for healthy subjects who require treatment with moderate doses of haloperidol at 30-minute intervals for the control of the symptoms of acute psychosis. Nevertheless, it is prudent to assume that the risk of cardiovascular effects during rapid treatment with neuroleptics may be greater in elderly psychotic patients and all patients who are medically ill, particularly with cardiovascular disease. Patients in these categories should be treated with small doses of haloperidol and monitored closely throughout rapid treatment with neuroleptics. Idiosyncratic reaction to haloperidol and other neuroleptics is also a possibility for any patient.

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ETTERS TO THE EDITOR

is section contains reactions to Journal *articles, statements of opinion, comments on Association activities, quests for information, etc. Letters should be submitted in duplicate to the Editor, who makes all decisions garding publication. Letters must be typed double spaced throughout and should not contain more than 500 rds and 5 pertinent references. Criticisms of published articles will automatically be sent to the author(s) for sponse. Letters will be edited for clarity and conformance with* Journal *style. We regret that we cannot inform iters of the disposition of letters or return those which are not printed.*

P: Neurochemistry, Treatment, and More

SIR: We would like to comment on "Phencyclidine-Induced chosis" by R. Michael Allen, M.D., and Steven J. Young D. (September 1978 issue). First, our clinical experience pports their conclusion that phencyclidine (PCP) produces linical picture that is closer to that of schizophrenia than es amphetamine. If this is correct, then understanding the rochemical basis of the effects of PCP is of considerable portance. We would like to elaborate on the personal comnication the authors cited from us, which indicates that effects of PCP may be due in part to its actions on dopae metabolism, and to suggest that reserpine, which cks dopamine storage, and/or α-methyl-*p*-tyrosine MPT), an inhibitor of dopamine synthesis, should be tried he treatment of prolonged psychotic reactions from PCP.

The effects of PCP on dopamine-dependent behaviors in s are mediated by presynaptic effects on dopamine neus rather than effects on postsynaptic dopamine receptors. us, PCP causes ipsilateral turning in rats with 6-hydroxypamine-induced lesions of the substantia nigra (unpubhed data from Fessler, Sturgeon, and Meltzer). Its effects dopamine metabolites are blocked by γ-hydroxyyrolactone, an anesthetic that inhibits the firing of dopae neurons (1). We have found that the effects of PCP on omotor activity and stereotypy are almost completely inited by reserpine treatment; in contrast, amphetamine's ects on locomotor activity are potentiated by reserpine treatment. This indicates the effects of PCP are mediated part by storage pool dopamine that is depleted by reserne. We have also found that the effects of PCP on turning havior in the rat are inhibited by pretreatment with MPT, which indicates that some actions of PCP may also pend upon release of newly synthesized dopamine. Finalwe have evidence that PCP can block dopamine uptake in ro.

This multiplicity of effects of PCP on dopamine may conbute to the diverse effects of PCP on behavior compared h amphetamine, whose actions on dopamine appear to be ited to release of newly synthesized dopamine and blocke of dopamine uptake. It is tempting to conclude that store pool dopamine may have more relevance to behavior an has previously been appreciated. Since both drugs afct other neurotransmitter systems, however, we are not pposing a simple relationship between dopamine and psypathology.

We would nevertheless urge clinical treatment with reserne, which is clinically available, or AMPT, which is available for clinical investigations, or with both, for individuals with prolonged schizophreniform psychoses following PCP use. This might prove more effective than using dopamine receptor blockers such as haloperidol, since such drugs increase the activity of dopaminergic neurons by negative feedback from postsynaptic dopamine receptors as well as presynaptic mechanisms. Increased presynaptic activity could overcome the effect of the neuroleptics.

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SIR: We were very interested in the article by Drs. Allen and Young on phencyclidine-induced psychosis. We have recently completed a study of 15 patients with PCP psychosis and we obtained similar data, which indicated that it is difficult, if not impossible, to separate PCP psychosis from schizophrenia on clinical grounds (1). However, we disagree with the authors on a number of other points.

First, the fact that it is difficult to separate two diseases on clinical grounds does not make one a model for the other. There are basic differences in the phenomenology of the two processes: 1) PCP psychosis has an inherent component of sensory deprivation due to the anesthetic properties of the drug, which are lacking in schizophrenia, and 2) in our experience, PCP psychosis has a strong component of cognitive impairment, the clearing of which is our criterion for discharge, while the cognitive impairment in schizophrenia is classically less important. Nevertheless, we have found one intriguing parallel component in the two processes: temporal disorganization. Based on work by Melges, we studied temporal disorganization (impairment of time sense) in PCP psychosis and found preliminary evidence that on at least one test of temporal disorganization (estimating a period of 30 seconds) PCP users consistently underestimate, as do schizophrenics. The implications of such findings have been discussed elsewhere (2).

Second, we disagree with the authors on psychopharmacologic management. A clear distinction should be made between acute and chronic patients. We have always used diazepam to sedate our acute patients because of several reports of cardiac arrhythmias and hypotension in acute PCP patients treated with antipsychotics (1, 3, 4). Not all clinicians will agree with this treatment, and the treating psychiatrist will be under considerable pressure from the nursing staff to "control" these potentially violent patients, but we have seen one case of the interaction (with haloperidol) and would caution against it. Seclusion and restraint should also be used with caution; we have had one patient who developed acute renal failure secondary to acute rhabdomyolysis (tearing of muscles and release of myogloben) due to prolonged restraint. On the other hand, we have no reservations about the use of antipsychotics in PCP patients who are suffering the chronic effects of abusing the drug or in subacute cases once there is no free drug in the serum. Thus, we would advise caution in the acute treatment of these patients and encourage the initial use of diazepam in doses of up to 60 mg/day rather than antipsychotics.

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Drs. Allen and Young Reply

SIR: Athough we are sensitive to the points made by Drs. Yesavage and Freeman, we feel we can answer them satisfactorily.

As to their first criticism of our proposing prolonged phencyclidine-induced psychosis as a good model for schizophrenia, we can only reiterate our position that in our study PCP produced a clearer, longer lasting schizophreniform illness than other previously reported drug models. As to the drug model concept of schizophrenia itself, the amphetamine model of schizophrenia proposed by Snyder and the psychedelic model summarized by Wyatt (1) have been generally accepted as conceptually useful in the study of schizophrenia. Recent work with PCP indicates that it fits both the dopamine hypothesis of schizophrenia (2) and the dopamine-acetylcholine balance hypothesis (3) of schizophrenia quite well. Garey and Heath (4) demonstrated that PCP inhibits the reuptake of dopamine in rat striatal synaptosome preparations, and Finnegan and associates (5) demonstrated that PCP has both potent dopaminergic and anticholinergic activity. Thus our clinical data fit quite well with the recent neurochemical data.

As to their criticism of our psychopharmacological management of our patient sample, we can only say that there are no significant differences between our views. We stated in our report that none of our patients was acutely intoxicated with PCP. Our patients were all subacute and chronic in their presentation. None had acute neurologic signs and none had ingested PCP less than 8 hours before hospitalization. Unfortunately, we did not have the facilities to monitor blood PCP levels in our patients. We agree that for acute PCP intoxication diazepam is the treatment of choice and physical restraint should be minimized and good nursing care maximized. We were discussing our treatment of behaviorally disordered patients following their acute toxic phase, and we found that although neuroleptic treatment was not extremely effective in reducing psychotic thought processes, it was helpful in preventing dangerous agitation or destructive behavior.

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SIR: The emergence of phencyclidine (PCP) as a ubiquitous drug of abuse has become a matter of concern. The intensity of behavioral disruption PCP produces in humans seems to surpass that of all other drugs of abuse. No antagonist is now available to combat the behavioral effects of acute PCP intoxication. Therefore, the recent finding in the laboratory that baclofen is effective in antagonizing the psychomotor effects of PCP in experimental animals is of interest.

Phencyclidine produced intense locomotor stimulation in Swiss mice that was characterized by compulsive running activity. This effect was completely blocked by baclofen, administered before PCP or at a time when the effect of PCP was maximal. In albino rats, the characteristic head-sways produced by phencyclidine was effectively antagonized by baclofen. In both of these experiments, the impressive blocking effect of baclofen started within 5 minutes after intraperitoneal administration and lasted for 60–90 minutes with no return of the PCP-induced symptoms. However, baclofen failed to influence *d*-amphetamine-induced locomotor stimulation. Diazepam, currently used in the management of PCP intoxication, was ineffective in these tests. Details of the pharmacologic and biochemical studies will be published elsewhere.

Baclofen, a putative γ-aminobutyric acid (GABA) agonist (1, 2), has been found useful in the treatment of spastic disorders (3). It also has been reported to produce beneficial effects in Huntington's chorea (4) and tardive dyskinesia

l has been found to be a safe drug. Other properties of this ıg, such as its muscle relaxant and anticonvulsant effects l sedation also could prove to be beneficial in the manage- nt of acute phencyclidine intoxication. Whether baclofen ɔ blocks the psychopathologic effects of PCP in man has be investigated, and efforts to perform such studies in this spital have been initiated.

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ıtelet MAO Research: Complications

SIR: Arnold J. Friedhoff, M.D., and associates are to be nmended for their excellent discussion of the comxities involved in interpreting human blood platelet bio-ɜmical results (August 1978 issue, pp. 952–955). The au-ɾrs are correct to point out that platelets represent a heter-ɜneous population; in fact, new platelets produced under ıditions of increased demands are larger than average and ◂ain large throughout their life span (1). Ordinarily, plate-ɜ become smaller with age, and physiologic differences ɹe been found among platelet populations of varying sizes . To complicate matters further, platelets stay in the circu-ɾon 7–14 days and may be removed either randomly or ectively with aging (3). Thus, any biochemical platelet ɜasure is an average of a heterogeneous population sam-ɔd at a particular time in an ever-changing production-de-■uction equilibrium.

The blood platelet is an organized, but dying, fragment of ɜgakaryocyte cytoplasm and, as such, is totally dependent ɔn the megakaryocyte for its integrity. By the time one nples a platelet population, a series of critical steps have ◂eady occurred in the megakaryocyte population. Interingly, megakaryocytes produce platelets after replicating ɜir chromosome complement a variable number of times, l the amount of platelet-producing cytoplasm apparently ɾelates with the chromosome complement (4). For exple, platelets are probably produced from megakaryoɜes varying in ploidy values from 4N to 64N (where ɜone haploid chromosome complement) (5). This raises ne interesting questions about the quality and quantity of nsfer of genetic material to the platelet population, per-■ps adding to the heterogeneity. Thus, although platelet AO levels are genetically determined, the amount found at ɣ one time may be dependent on the chromosome comɜment of the megakaryocyte population from which the ■telets were derived. Furthermore, since psychoactive ıgs can affect leukocyte and erythrocyte production, one has to wonder whether certain megakaryocyte parameters may also be affected in certain instances, since these three types of blood elements originate from the same stem cell line (the pluripotential stem cell) (1, 3).

The purpose of this letter is not to discourage the use of platelets in schizophrenic research but to agree with the authors and to point out further complexities in the use of the platelet model in psychiatric research. Research in the areas discussed above may lead to a better understanding of seemingly contradictory laboratory results.

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Considering Lethal Catatonia in Cases of Hyperpyrexia

SIR: Two recent articles in the *Journal* (1, 2) have presented cogent facts and arguments implicating phenothiazines as a contributory cause of hyperpyrexia in the mentally ill, an association first suggested by Ayd (3). It is important to keep in mind that hyperpyrexia in the mentally ill may be due not to the medications the patient is taking but to a mysterious illness known as lethal catatonia. The syndrome was first reported by Luther Bell in 1849, more than a century before there were any phenothiazines. Hyperpyrexia has been noted in about a third of the patients with lethal catatonia (4). We need to be careful to avoid automatically attributing a patient's hyperpyrexia to medication.

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ROGER PEELE, M.D.
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Freud and Religion

SIR: I wish to point out an error in "Report of the Task Force on Religion and Psychiatry: Phase III" (June 1978 issue, Official Actions). The remark attributed to Freud, "And now you must not be surprised if I plead on behalf of retaining the religious doctrinal system as the basis of education

and of man's communal life . . . ," is actually that of Freud's fictional antagonist in "The Future of an Illusion" (1). Throughout this work Freud erected this pro-religion straw man, only to demolish him.

However, this is less of an error than it appears—and in fact becomes a sort of truth—if we choose to read the fictive opponent's comments as the unconscious opinion of Freud himself. There is ample documentation of Freud's mixed feelings on the subject. In the very work in which he declared religion a "universal obsessional neurosis" (2) Freud opined that "a progressive renunciation of constitutional instincts [which he considered one of the foundations of social cohesion] is effected by religions, in that they require the individual to sacrifice his instinctual pleasure to the Diety." This was repeated in 1908. Only a few months before he suggested in "Leonardo da Vinci" that religion may be a determinant of neurosis, he had written Pfister regarding "the happy state of earlier times when religious faith stifled the neuroses" (3, p. 16). In a 1919 addendum to "Leonardo" and in *Group Psychology and the Analysis of the Ego* he reiterated this. Although on balance "The Future of an Illusion" and "Civilization and Its Discontents" are unfriendly toward religion, "Moses and Monotheism" contains a much softer view.

Many pages could be written about the determinants—some of which are noncognitive and unconscious—of Freud's ambivalence. I think these include 1) conflicts over aggression and competitiveness vis-à-vis the father, 2) dependency-independence conflicts vis-à-vis the mother and, to a lesser extent, the father, 3) unresolved feelings on the issue of mortality-immortality, including Freud's search for meaning in life, 4) contact with the repressive and hypocritical religion and mores of fin de siècle Vienna and their repercussions on his patients, 5) Freud's ambivalence toward his Jewishness, and 6) the dichotomy between rationalistic-positivistic and romantic elements in Freud's education manifested partly by his lifelong battle with his speculative or "mystical" tendencies (4).

Although an ocean of ink has been spilled on the topic of religion and psychoanalysis, there is still great controversy about the compatibility of the two. Part of the problem is that the philosophical and ethical implications of psychoanalysis have not been fully worked out. The questioning, reductionistic attitude of dynamic psychology seems quite foreign to the faith-enjoining modus of religion. In considering such questions, it may be wise to keep in mind what Freud said in 1927:

> In point of fact psycho-analysis is a method of research, an impartial instrument, like the infinitesimal calculus, as it were. . . . If the application of the psychoanalytic method makes it possible to find a new argument against the truths of religion, *tant pis* for religion; but defenders of religion will by the same right make use of psychoanalysis in order to give full value to the affective significance of religious doctrines. (1, p. 127)

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EDWIN R. WALLACE IV, M.D.
Baltimore, Md.

A Comment on Runaway Houses

SIR: I am writing in regard to "The Runaway Center a Community Mental Health Center" by James S. Gord M.D. (August 1978 issue). It is quite true that runav houses provide a most needed service. Many disenchar youth see the traditional physician's office as "est lishment" and therefore to be shunned. Going to a runav house instead enables them to avoid being seen as psych ric patients and also serves as a diversion from the path leads to juvenile court. At times, a few days away from family plus supportive counseling will help the young per reenter school and family. The runaway center also ke the teenager from living on the streets and turning to dru prostitution, and other antisocial behaviors.

It would appear that runaway houses are the answers today's youth. Unfortunately, their appeal is also a g source of weakness. The staffs of such centers are o poorly trained or untrained. Psychopathology often goes noticed until it erupts into a full-blown psychotic episc With relatively poor diagnosis and treatment regimens, sociopathic teenager is able to find a place where he or can take full advantage of the other adolescents.

Techniques of family therapy are sophisticated eno today for effective intervention if there is continuing tr ment. However, the expertise for such treatment is o found in the traditional agencies such as the commu mental health center.

Everyone who works with young people realizes tha number of different agencies have contact with the sa type of problems. Physicians, psychiatrists, mental he centers, hospitals, juvenile courts, and runaway houses see troubled young people. Each staff has a different philo phy and level of training. If one agency could coordir with another, it is probable that each agency would be be able to serve the needs of this group and the service wo be more cost effective. There is a need for both levels service but an even greater need for communication and operation between services.

PAUL KING, M.D.
Memphis, Tenn.

Dr. Gordon Replies

SIR: Dr. King's introductory paragraph emphasizes importance of runaway centers in diverting young pec from incarceration in mental or penal institutions and helping young people who cannot or do not want to surv on the streets. His final paragraph correctly highlights importance of cooperation among the staff of runa houses and other mental health, social service, and juve justice agencies. However, in between, Dr. King make number of damning and unsubstantiated statements at the staff of runaway houses, the services offered to yo people who come to them, and the success of family ther in working with young people who stay at home.

Although family therapy might indeed help young pec

live happily at home, it has generally been unavailable or, delivered by many mental health professionals, unacceptle to young people who have left or are about to leave their mes. Therapists may speak of "family problems," but ung people often feel the therapists share the parents' int of view and biases. In any case, when things get rough, e young people are the ones who have to take the psychogical tests or enter the mental hospitals.

The runaway house provides a context in which family erapy takes on a different meaning. When offered by counlors who have proved themselves empathic and trustorthy, by a staff that eschews labeling, diagnosis, institunalization, and medication, family therapy is regarded by ung people as a catalyst to productive change, not an inrument of parental domination. I noted in my article that most half of all the young people who come to stay at runay centers are involved in family therapy and that onearter of the clients of runaway houses continue to live in eir own homes while they receive individual and family unseling.

Dr. King's indictment of the training of runaway house unselors is unfair. I noted in my article that over the last ur to five years runaway centers have "made use of ineasing numbers of mental health professionals [and have] ined their workers in techniques of individual, group, and mily therapy." These informal efforts have been comemented and enlarged by formal recruitment of mental alth professionals. Nationally, 25%–35% of the counselors runaway centers have M.S.W.s, and almost all of the rest ve undergraduate majors in psychology, social work, unseling, education, or child development. The credenils and training of this direct service staff are comemented by the consultants available to runaway centers: most every program has a psychiatrist or Ph.D. psycholost involved in providing services or consultation. Finally, der the terms of the Youth Development Bureau's technil assistance contract with the Educational Systems Corpotion each of the 150 federally funded runaway programs is so involved in 9–10 days a year of training workshops.

In settings where much of the staff has received advanced ofessional training, where all staff members are keenly vare of the techniques used by mental health professionals, e "diagnosis and treatment regimens" are anything but elatively poor." Although the diagnostic labels that are stomarily used in psychiatric practice are rarely applied to naways, the level of sensitivity to the psychosocial situaon of each young person seems as high in most runaway ograms as it is in most psychiatric centers for troubled olescents.

Finally, though there is indeed an exploitation of runays by older people who live on the street or in the neighorhoods of some runaway centers, I have seen very little idence that "sociopathic teenagers . . . take full advantage the other adolescents" in the runaway center itself. The sy communication between staff and clients in a runaway nter, the constant presence of counselors, and the daily ouse meetings militate against the secrecy and authorarianism that foster bullying and exploitation. My impreson is that fighting and exploitation occur no more and perps less often than they do in mental hospitals, schools, or the street.

I would suggest that Dr. King visit Runaway House, Inc., Memphis, Tenn., for a firsthand look at the kind of training that the counselors receive (*all* of the primary counselors ve master's degrees in one or another of the human servces fields) and that other psychiatrists who are particularly concerned with adolescents and their families contact the Youth Development Bureau (the Humphrey Building, Washington, D.C. 20201) for the names of runaway centers near them. The runaway center is an extremely valuable mental health resource.

JAMES S. GORDON, M.D.
Rockville, Md.

Difficulties with Retrospective Studies

SIR: In "Discriminating Features of Borderline Patients" (July 1978 issue), John G. Gunderson, M.D., and Jonathan E. Kolb, M.D., presented an excellent study but misrepresented the results. Their statistical methods have been criticized in the past (1, 2). These methods make theirs a retrospective study because key statements (DIBs statements) and sections of dysfunction are identified and weighed appropriately (by means of standardized coefficients) *after* the patients are assigned to either the borderline or control groups. This stepwise discriminant function analysis gives the "best fit" for the patients who are already placed in certain diagnostic categories, but the fit would not necessarily be as good with prospectively chosen patients. Certainly, the diagnostic accuracy would be much less than the 81%–100% found in this study. This is a retrospective method, and the next logical research step would be a formula that weighs each DIBs statement and section score in accordance with the standardized coefficients the authors found in this study and application of this formula prospectively to a mixed group of patients.

Because this is a retrospective study statistically, the conclusions are overdrawn. They have not "shown that borderline patients can be discriminated with high accuracy from matched comparison groups." Instead, this study has shown that once a patient has been given a presumptive diagnosis of borderline disorder, then certain statements are more likely to be true (or false) about this patient than about patients with other diagnoses. This study neither strengthens nor weakens the argument that there exists either a "recognizable personality disorder," or a "definite syndrome"—nor does it provide a "replicable means for identifying borderline patients." That must still be done by using these data in a prospective fashion.

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FREDERICK W. ENGSTROM, M.D.
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Drs. Gunderson and Kolb Reply

SIR: Dr. Engstrom is thoughtfully conservative in his interpretation of our study. Although the diagnostic accuracy of 81%–100% we achieved is suspiciously high, there is nothing inherent in the methodology to suggest that "the diagnostic accuracy would be much less." In fact, we applied the discriminant function prospectively to a different sample of 29 independently diagnosed borderline patients and found

that it accurately identified 22 (76%) of them. Despite these encouraging results we can agree with Dr. Engstrom that our study is exploratory, and a more definitive replication using randomly selected patients is warranted. We have provided a methodology and isolated clinical variables to make this feasible. In the meantime, it seems reasonable for anyone wishing to define borderline patients to use the set of discriminating criteria we have reported.

JOHN G. GUNDERSON, M.D.
JONATHAN E. KOLB, M.D.
Belmont, Mass.

A Cannabis Caveat Reinforced

SIR: The article "Marijuana Use in Schizophrenia: A Clear Hazard" (October 1978 issue) by Darold A. Treffert M.D., is a timely caveat. Those of us whose practices include large numbers of adolescents anxiously search the literature for documentation of our repeatedly confirmed observation that marijuana is a psychiatrically deleterious drug, especially for vulnerable or unstable youths.

I am certain that the literature would contain many more such observations if clinicians actively sought to make the temporal connection between marijuana use and acute psychotic episodes. A good case in point is another article in the same issue of the *Journal*, titled "Hyperthermia Associated with Chlorpromazine and Full-Sheet Restraint." Drs. Greenland and Southwick called our attention to an important association but failed to underscore another important association, which they state explicitly but without comment. I refer to the fact that their 18-year-old patient was well until 2 weeks before admission when his acute symptoms of psychosis began. "He also expressed considerable guilt about *recent frequent marijuana* use. He did not use any other licit or illicit drugs, including alcohol" (emphasis added).

I am indebted to Dr. Treffert for recording his observation and to the *Journal* for publishing it. I would hope that this type of observation will influence those who still entertain doubts about the untoward and adverse effects of cannabis.

DORIS H. MILMAN, M.D.
Brooklyn, N.Y.

Diagnostic Issues in Bromism

SIR: "Bromism: Alive in Well" (July 1978 issue) by Ira Brenner, M.D., was an interesting case report, but I would raise several questions about the author's assumptions.

Contrary to Dr. Brenner's claim, there has been a reported case of bromism caused by chronic drinking of well water (1). Furthermore, the patient in Dr. Brenner's article could have been experiencing alcohol withdrawal rather than bromism; her alcohol consumption increased during the week before admission, and on entering the hospital she displayed restlessness, poor memory, and a tremor, all of which resolved quickly during hospitalization. We are not told the patient's blood alcohol or admission or her vital signs during the period of symptom improvement. Moreover, only one high serum bromide level (38.3 mg/100 ml) was reported, and the level apparently returned to normal within one week. If only one serum bromide level was abnormal, the possibility of a laboratory error cannot be ruled out, especially in light of this patient's rapid improvement. Although Dr. Brenner makes a good case in incriminating the well water as a cau of bromism, I think substantial evidence seems to be lackin

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CHARLES B. SCHAFFER, M.D
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Dr. Brenner Replies

SIR: I appreciate Dr. Schaffer's interest in my article a would like to respond to his hypothesis that alcohol wi drawal and/or laboratory error could account for the tient's clinical picture. On admission, she was not toxicated and did not smell of alcohol, so alcohol levels w not obtained. Her consumption of alcohol was about beers a day, but she did not experience significant alco withdrawal. She did not have autonomic instability, el trolyte imbalance, or subjective feelings of shakiness, a she received only 10 mg of diazepam during her entire hos talization. Her agitated depression did resolve quickly, w tremor and hopelessness diminishing by the end of the f week without specific treatment. However, the effect of hospital milieu itself on her improvement is a factor tha more difficult to assess. It should also be noted that when returned from a weekend at home, her bromide level creased to 15.9 mg/100 ml, after it had decreased to and l eled off at 5 mg/100 ml. This was another instance of consumption of well water correlating with an increase serum bromide level, and we felt that the laboratory resu were reliable. The well water was also assayed and found have a 2.1 mg/100 ml concentration of bromide.

Nevertheless, the substantial evidence that Dr. Schaffe seeking implies a need for many more clinical data, and can begin with the reference that he cited.

IRA BRENNER, M.D.
Charlottesville, Va

Sex Chromosome Anomalies and Manic-Depressive Illness

SIR: I should like to comment on the letter from Jacq Lesage, M.D., and Guy Chouinard, M.D., about "Mar Depressive Illness Associated with Klinefelter's Syndro and Essential Tremors" (June 1978 issue). Although the thors are right to point out that this observation is in line w the X-linked dominant hypothesis of inheritance in ma depressive illness (1, 2) and may possibly explain some parent cases of father-to-son transmission of the disease their suggestion that the case was the first to be repor with both illnesses is incorrect. In another section of issue, Stanley N. Caroff, M.D., reported on a patient w both syndromes and noted that eight such cases have b described previously.

Nevertheless, this type of observation has theoretical plications for the inheritance of manic-depressive illn since according to the sex-linked dominant hypothesis, tients with the XXY constitution may be expected to hav greater incidence of manic-depression than XY males cause they behave genetically like females. Conversely,

its with the XO genotype (Turner's syndrome) may be s prone to affective illness than normal XX females. To knowledge, there have been no reports to date of an as-iation between Turner's syndrome and manic-depressive ess. The question of potential associations between sex omosome anomalies and manic-depressive illness de-ves further attention and can only be answered through ll-designed epidemiologic studies.

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JULIEN MENDLEWICZ, M.D., PH.D.
Brussels, Belgium

s. Lesage and Chouinard Reply

SIR: As Dr. Mendlewicz points out, our case report is not first one describing the association of Klinefelter's syn-ome with bipolar affective disorder. However, it is, to our owledge, the first report of these two illnesses associated h hereditary essential tremor. The point we wanted to phasize was that similar cases could explain the father-to-n transmission of bipolar affective disorder and should be minated by chromosomal analysis before suggesting that it incompatible with the X-linked dominant inheritance of olar affective disorder. It should also be noted that even a gative result on chromosomal analysis does not entirely minate Klinefelter's syndrome, since some patients may ve pure gonadal dysgenesis, i.e., the phenotype of Kline-ter's syndrome with a normal chromosomal analysis.

Another phenomenon that may also explain the observed her-to-son transmission of bipolar affective disorder is saicism, which (although rare) could result in father-to-n transmission of a bipolar affective X-linked dominant ne despite normal chromosomal analysis. Finally, we uld suggest that in cases of father-to-son transmission of olar illness, a complete genital physical examination ould be performed in order to eliminate Klinefelter's syn-ome or sexual mosaicism associated with affective illness.

JACQUES LESAGE, M.D.
GUY CHOUINARD, M.D.
Montreal, Que., Canada

Defense of the Hypothetical-Deductive Method

SIR: There has been considerable criticism (1, 2) of the thodology described by Silvano Arieti, M.D., in "Psy-otherapy of Severe Depression" (August 1977 issue). ere seems to be a tendency to reject any methodology that es beyond the purely descriptive and phenomenologic radigm. Although this approach is a sine qua non for a ience of human behavior, there is certainly a place for the pothetical deductive method, which uses a theoretical mework as a heuristic approach for intervening in a therapeutic manner with patients who are seriously disturbed.

This heuristic approach can also serve as an interim means of representing the data observed until there are data sufficient to further modify the theoretical framework. Freud recognized the ultimate psychobiologic basis of severe mental illness, which he designated the narcissistic neuroses. Dr. Arieti has done no less in his interpretation of schizophrenia (3), where he presents a tentative model of schizophrenia as a psychophysiologic disturbance of the central nervous system. Needless to say, his model is based on considerable observational data, which include the probable evolution of cognition from paleologic to secondary process thinking. I might add that much of Dr. Arieti's work will probably be validated empirically in the future as we learn more about the functions of the right and left cerebral hemispheres. Stevens' work (4) on a possible gating mechanism in the mesolimbic system will probably also play a role in elucidating the underlying pathophysiology of true process schizophrenia.

It is my opinion that with our new knowledge of the central nervous system and its function, coupled with the clear understanding that all knowledge is influenced and modified by the participant observer, both the nomothetic and idiographic methods of investigation as described by Dr. Arieti must be employed if any workable conceptual framework of human behavior is to be formulated. The fact that the human central nervous system processes data at a neocortical level as well as at lower levels provides substantial evidence to indicate that Dr. Arieti's psychodynamic form of intervention can have definitive effects on how the individual processes his perception of reality. Psychodynamic intervention can be more than anecdotal, and a modified science of human behavior based on observation and psychological intervention can evolve. I refer those who find this idea difficult to accept to Dr. Arieti's *Interpretation of Schizophrenia* (3) and to Hans Reichenbach's *The Rise of Scientific Philosophy* (5) wherein he points out that the hypothetical deductive method is a special form of induction.

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CLIFFORD J. SIMSKE, M.D.
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Jealousy and Nontraditional Marital Units

SIR: In his article "Pathological Tolerance" (June 1978 issue) Emil R. Pinta, M.D., followed traditional psychiatric thinking by reporting three triads within a framework of marital and personal pathology. Although it is true that there were problems in all 3 cases after the introduction of the third person into the relationship, this does not necessarily mean that the relationships or the psychodynamics are evidence of pathology. Dr. Pinta reports no data on the quality of the dyadic relationship before the addition of the third

partner, so it is impossible to gauge the effect of the third partner on these relationships. Certainly, societal disapproval and lack of responses in individual behavioral repertoires (due to a lack of socialization for triadic marital experiences) may be a factor in the development of the problems noted by Dr. Pinta.

Dr. Pinta states that "the emotional involvement among members of the triadic unit, the stability of the unit, and the absence of three-person sex are characteristic features of this symptom complex and distinguish it from . . . other nonmonogamous sexual lifestyles." From this statement, it seems that he is unfamiliar with the work of Constantine and Constantine (1), who found that group marriages were often characterized by intense emotional involvement among the members of the group and that the group marital situation was used by members as a vehicle for nonneurotic need satisfaction and personal growth. Other workers who have investigated alternative sexual lifestyles have pointed out the potential for intimacy and maturation within such units and have noted the relative absence of neurotic pathology (2, 3).

Psychiatrists need to devise new techniques for working with nontraditional marital units. One such methodology would be marital therapy with all three members of the triad. Constantine (4) and Blood and Blood (5) have worked extensively with nonpathological jealousy within such units and have also devised new therapeutic techniques for working with jealousy. Psychiatrists should be careful to avoid automatically labeling nontraditional lifestyles and their participants as "deviant" or "pathological" and should seek to identify, from the participants' point of view, the benefits of such lifestyles. Therapy should be aimed at the resolution of problems within the context of the participants' goals for treatment, not according to the bias of the psychiatrist.

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KENNETH SOLOMON, M.D.
MONA SOLOMON, M.S.
Richmond, Va.

Dr. Pinta Replies

SIR: The comments made by the Solomons raise important questions regarding the nature of jealousy and the opposite attitude, tolerance. Traditionally, jealousy has been regarded as a sign of healthy commitment to a valued partner. Freud expressed this viewpoint when he wrote that jealousy is a normal feeling, much as grief is. If anyone appears to lack jealousy the inference is that it has undergone repression and therefore will play an even larger role in the unconscious (1). There is, however, another attitude toward jealousy that emerges from experience with nonmonogamous lifestyles (such as open marriages, swinging, group marriages, etc.). This viewpoint regards jealousy as an unhealthy sign of possessiveness and dependency that indicates an inability to care and allow for the development of one's partner. Studies by Constantine and Constantine and others quoted by the Solomons appear to substantiate this opinion. Furthermore, the flexible nature of jealousy is borne out by sociological comparisons and by experimental communities such as the one at Twin Oaks (2).

I did not mean to state in my article that all tolerant extramarital relationships are pathological. Each situation must be regarded separately. In the cases I presented the tolerant attitude of the spouse seems to be a pivotal dysfunctional element in the marital relationship. I also feel that the extramarital nature and stability of the described relationships promoted the acting out of unconscious psychological mechanisms more than many other lifestyles would. I agree with the Solomons that those of us in the mental health field must try to avoid labeling as pathological lifestyles that differ from our own or from our view of normalcy. As in the case of jealousy, tolerance should be regarded as dysfunctional or pathologic only when it appears to interfere with personal growth and interpersonal relationships in a chosen lifestyle.

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EMIL R. PINTA, M.D.
Columbus, Ohio

Corrections

In the November 1978 Official Actions section (p. 14[illegible]) the name of Om Prakash, M.D., was misspelled.

There was an error in table 2 of "Prediction of Individual Dosage of Nortriptyline" by Thomas B. Cooper, M.A., and George M. Simpson, M.B., Ch.B., in the March 1978 issue. Blood levels are given in ng/ml.

The staff regrets these errors.

OOK REVIEWS

ress Disorders Among Vietnam Veterans: Theory, Research id Treatment, *edited by Charles R. Figley, Ph.D. New ork, N.Y., Brunner/Mazel, 1978, 311 pp., $15.00.*

The United States military involvement n Viet Nam be- n in earnest during the bold and exuberant presidency of hn Kennedy and ended with the disorderly evacuation of e American Embassy in Saigon in April 1975. The slide om the exhilaration of Camelot to the cynicism of Water- te was unexpected and unsettling. Ever now it remains fficult to write with moderation about this epoch and its ajor casualties—the Viet Nam veterans.

The editor introduces this book by setting out six goals: 1) define the construct of Viet Nam-related combat stress sorders, 2) to alert mental health professionals to the theotical and clinical importance of these disorders, 3) to perade the reader that the residue of combat stress transcends ne and situation, 4) to remind the reader that little is known out long-term results of catastrophic combat experience, to provide treatment guidelines, and 6) to expand the owledge base of combat-related stress disorders. These e ambitious tasks to accomplish in one volume, they are ade even more elusive by the editor's attempt to be evenhded. Twenty-four authors contribute to this book. The ader will recognize the names and views of well-known tiwar activists as well as those of some career military ental health professionals.

Two representative points of view seem in conflict. The st hypothesis is that the military experience in Viet Nam as unique and traumatic because of the inherent nature and vagery of the conflict, the elusive character of the enemy, e impact of individually tailored one-year duty rotations, d the ambivalent homecoming accorded combatants. The ediction made in this view is that initial opinions about the ccessful adjustment of veterans will not stand the test of ne and that many veterans will seek help months and years er for delayed catastrophic stress disorders (post-Viet am syndromes). The second hypothesis is that attempts to ribute the problems of veterans to their unique experience Viet Nam does not withstand scrutiny and does not lead to ique symptomatology. It is suggested in this view that ose who have had the most difficulty during military duty d as veterans are those who already had the greatest numr and kinds of preexisting handicaps in personal skills.

The text is divided into three major sections involving thetical formulations, some representative research reports, d suggestions for treatment of this special veteran popuion. The first and third divisions include papers based on erature reviews and clinical experiences with an emphasis psychosocial perspectives. The section on research reorts presents the results of descriptive follow-up studies th appropriate statistical analyses. Sixty-one pages in this tion are used to reproduce the various research inument questionnaires; this will not likely prove useful to ost readers.

Sober reflection suggests that, for many, the war in Viet am is still not over and the need for mental health services targeted for Viet Nam veterans continues and may increase. This book provides a service to those who work with veterans by examining pertinent treatment issues and offering relevant suggestions for therapeutic interventions that may be useful in making the mental health professional better informed and more empathic.

WILLIAM D. WEITZEL, M.D.
Lexington, Ky.

A Child's Journey: Forces That Shape the Lives of Our Young, *by Julius Segal, Ph.D., and Herbert Yahraes. New York, N.Y., McGraw-Hill Book Co., 1978, 336 pp., $10.95.*

Dr. Segal, Director of Public and Scientific Information Programs of the National Institute of Mental Health, explains in his introduction his aim in coauthoring *A Child's Journey:*

> My aim was to produce neither a traditional textbook nor a How To Manual of Child Rearing but a resource grounded on research findings for all of us who care about children and who want, therefore, to know more about why they develop as they do and what they can and cannot do to shape the outcome of their journey.

Dr. Segal's editors add that the book was written particularly for parents but that it is also addressed to all adults "who deal in any way with children—teachers, doctors, psychiatrists, psychologists, social workers, lawyers and judges." It is perhaps unfair to ask a child psychiatrist to review this book for an audience that will consist primarily of psychiatrists because this is not the expressed target audience of this volume.

It will come as no surprise to the reader who scans between the lines that this book presents nothing new, exciting, startling, or stimulating to the adequately trained child psychiatrist who keeps up with recent developments in either the professional press or the Sunday magazines. However, it presents a well-thought-out, nonjudgmental integration of many theories of child development and associated research findings. The authors look at genetic and environmental factors in child development. They describe factors that other lay authors have often neglected—fathers, peers, schoolteachers, poverty, and racism. The research findings are presented in an objective way and are not confused with graphs, statistics, and other technical decorations, which would serve the lay reader. However, the authors' biases are apparent in the research findings they choose to highlight: one chapter is devoted to genetics and at least seven chapters to environmental factors. Even the casual reader has some hint as to the authors' predisposition about important factors in child development.

The final chapter, titled "Protecting Your Child's Mental Health," is probably the most important one for concerned, politically active psychiatrists. The authors summarize the

points they feel are the most important in the preceding 12 chapters, such as the importance of planning for, wanting, and anticipating children; recognizing and accepting children's individuality; the critical nature of the early mother-infant attachment; and the importance of a stimulating environment. The relevance of the relationships between fathers and their children, the importance of peers, the effect of malnutrition on child development, and the poisons of racism are also highlighted. This chapter is more than a summary. The authors exhort their readers to become involved in lobbying "for children" and "for the sake of children." They stress the need for an informed public advocate to enunciate their concerns about tax reform, reorganization of health care, racism, sexism, and energy as these political and critical issues impact on child health and development. They also exhort their readers to care for children and to commit themselves to child welfare. No respectable child psychiatrist can argue with the final line of this passionate sermon.

ELISSA P. BENEDEK, M.D.
Ann Arbor, Mich.

Methods of Group Psychotherapy and Encounter: A Tradition of Innovation, *by Jerrold Lee Shapiro. Itasca, Ill., F.E. Peacock, 1978, 354 pp., $12.50.*

Perhaps the best measure of this contribution to the literature is signaled by the 48 pages of reference and the 5 pages of index. The work encompasses the history of group therapies over a broad expanse of time. It covers a broad range of styles of leadership and applications. Most helpful is the liberal citation of authors in connection with each aspect of group psychotherapy under consideration. This makes it an ideal companion piece to Yalom's text (1). At times the careful documentation obstructs the easy flow of the book, but at the same time it generates an extremely valuable source book for anyone embarking on a program of work and learning in this field.

Inclusion of excerpts from group therapy sessions is a very helpful way to convey the essence of the various styles the author uses in making the multiform communication of the group more intelligible. By doing so, however, he had to be selective and could not cover all variations of group treatment procedures. This does not materially detract from the book, but it does contrast with its global reach.

For an overview and an introduction to the literature this is a substantial book. It is also a useful tool for the teacher of group psychotherapy who plans to present the entire field rather than one specific approach.

REFERENCE

1. Yalom ID: The Theory and Practice of Group Psychotherapy. New York, Basic Books, 1975

JAY W. FIDLER, M.D.
Piscataway, N.J.

Psychiatric Examination in Clinical Practice, *by J.P. Leff, B.Sc., M.D., and A.D. Isaacs. Oxford, England, Blackwell Scientific Publications (Philadelphia, Pa., J.B. Lippincott Co., distributor), 1978, 143 pp., $11.75 (paper).*

Those of us in psychiatry who have learned to think predominantly in terms of intrapsychic conflict as having great etiological and diagnostic significance pay almost exclusiv attention to the form and/or content of patients' mental pr ductions, which are elicited by a verbal interchange. The a thors of this book are to be commended for bringing to th reader's attention the fact that observation, study, ar classification of a patient's behavior can be most helpful understanding psychopathology. Furthermore, others ca view the same behavior and arrive at a basis for agreeme and standardization.

This wonderfully brief book contains a wealth of inform tion and clinical wisdom. It begins by drawing attention t the need for careful, systematic observations, beginning wi the initial contact with the patient. The reader soon becom aware of the authors' capacity to make terms and concep explicit and to use clinical vignettes for further elucidatio The few drawings amplify some of their major points. Or gets a very vivid impression of what is being described. Le and Isaacs have attained their goal of setting down phenom na in such a way that clinicians who follow their guidelin can attain diagnostic concurrence.

Although the two chapters on taking a history describe method and style that I have generally set aside, they cov all the essential points in an orderly way. The reason th this technique has been found inadequate is that it is possib for the clinician to be misled. Repression and denial mig cause the patient to leave out essential material when th structured question-and-answer type of interview is used. is not enough for the interviewer to know all the right que tions to ask. Patients reveal more of themselves when the feel that the interviewer is "tuned in" to them. Perhaps th is more important when dealing with neurotic symptoms.

It is hard to understand why neurotic symptoms a grouped in the same chapter with affective ones. This rais the question of what audience the authors had in mind f their book. The inclusion of a chapter on childre makes appear geared to a general readership. However, the are covered most intensively (present mental state, delusion hallucinations, organic conditions, and affective symptom pertain to a hospitalized or equivalently ill group.

In summary, this book has some excellent feature: lucid ty, specificity, graphic description, brevity, and emphasis c observation. Its weak point is that it does not amply cov neurotic symptoms and personality disorders. Instead, it i cludes a section on the examination of children, which mig be beyond the skill and experience of a general psychiatris Nevertheless, it is well worth reading for practitioners as refresher and for students as a primer. They will find subt points of clinical wisdom expressed in a readable and mem rable fashion.

ROBERT W. ATKINS, M.D.
Rochester, N.Y.

Freud in Germany: Revolution and Reaction in Science, 189 1907. Psychological Issues Monograph 41, *by Hannah . Decker. New York, N.Y., International Universities Pres 1977, 348 pp., $15.00.*

A first impression that the period from 1893 to 1907 is t short for an exploration of the reception of psychoanalysis Germany is dispelled by reading Hannah Decker's boo Freud himself considered the first stage of reception to ha ended around 1907. That year Karl Abraham returned fro clinical work in Zurich and a visit to Vienna and establish a psychoanalytic psychiatric practice in Berlin.

Decker begins with a description of German culture and ociety. Then she divides her book into three parts: one on erman medicine and psychoanalysis, a second on the new" psychologists and psychoanalysis, and a third on the ducated German public and psychoanalysis. In each part he considers particular elements, such as the unconscious, reams, sexuality, and psychopathology. These sections ake it possible to focus on specific elements that varied in ow they were received by the three groups, but it also akes for repetitiveness.

German psychiatry was primarily interested in the psyhoses. Psychiatry and neurology were allied, and the laboatories for brain anatomy and experimental psychoathology in university settings were prestigious. Kraepen's clinical classification of diseases appeared in 1896 in his extbook, which exercised a decisive influence. The "new" sychologists stressed experimental methods and did not onsider psychopathology part of their field. Germany at the ime was not a fertile field for psychoanalysis.

Freud himself did not facilitate as much as he could have he favorable reception of his pioneering work. He did not ttend general medical meetings, did not publish in widely ead journals, and was slow to reveal the nature of his psyhoanalytic technique and its separation from hypnotism. le was identified with hysteria, considered to be an un-Germanic condition, and with childhood sexuality, a sensitive ubject in Wilhelmine Germany.

Decker attacks Ernest Jones as the official codifier of reud's view that his work was ignored and that what attenion it got was unrelentingly hostile. She refers to two myths: One is that for about ten years after *Studies on Hysteria* ppeared, Freud was uniformly ignored and rejected. The econd is that all real opposition to psychoanalysis centered n sex" (p. 172). I found her presentation of a varied and omplex reaction to Freud's work convincing.

Eugen Bleuler and C.G. Jung, although based in Zurich, ere very influential in Germany because they published in he German literature. Their propsychoanalytic positions ere invaluable. Many Americans at that time learned about sychoanalysis through Bleuler and Jung, who, in contrast o Freud, were hospital-based. Since psychiatrists in both ermany and America were primarily working in hospitals ith psychotic patients, Zurich was crucial to the spread of sychoanalysis. Karl Abraham worked at Bleuler's hospital efore returning to Berlin to practice psychoanalytic psychitry.

This book is illustrated with pictures of Abraham, Aschafenberg, Bleuler, Hellpach, Kraepelin, Stern, Wundt, Zieen, and several others whose positions and statements bout psychoanalysis are selectively quoted by the author. n addition to the references the author lists 33 interviews he conducted between 1963 and 1968, primarily with German psychoanalysts who came to the United States. Decker as written a valuable work that provides an understanding f what transpired in Germany and also illuminates the comarative reception of psychoanalysis elsewhere.

ARCANGELO R.T. D'AMORE, M.D.
Washington, D.C.

The Experience of Depression, *by Dorothy Rowe. New York, N.Y., John Wiley & Sons, 1978, 273 pp., $19.50.*

One of the most critical challenges facing both the clinician and the researcher is the need to reconsider the nature of depression. Only by the discovery of new hypotheses can the wide variety of information relevant to this issue become more deeply understood and more properly integrated. In our understanding of the phenomenon of depression, the field of psychiatry has moved steadily from a more descriptive position delving from a psychoanalytic point of view into some of the intrapsychic forces that generate depression to broader concepts that consider the significance of biological influences as well.

In this book the author explores another dimension, the environment of the depressed patient or, more particularly, the nature of the more intimate human interactions between the patient and key people in his environment. The author's approach is more than simply transactional. She explores the pertinent premises that affect the individual's perception of himself and others and that are expressed and colored by the very language used to form contact.

Dr. Rowe's work is essentially a report of a clinical investigation using special interviewing strategies carried out over a period of several years with a limited number of patients subject to depression. In the context of psychotherapy she explores at length the relationship between depression, the way the patient experiences himself and reality, and the approaches used to help patients recover from depression by modifying some of their critical assumptions. These assumptions have set the stage either for depression or for the recurrence or tenacity of a disabling emotional state.

This book will certainly be of interest to anyone who is seriously involved in depression research. Its value for the practicing clinician and the teacher, however, is decidedly limited. Much of the presentation consists of detailed case histories and experiences in the course of therapy that are rewarding only to substantiate Dr. Rowe's thesis. In a more fundamental way, although her observations stimulate the reader's thinking, they do not—for a variety of reasons (including the limitations in the style and manner of presentation and in the way her conclusions are drawn and discussed)—offer a significant new insight into the problem under study.

FREDERIC F. FLACH, M.D.
New York, N.Y.

Cultural Conceptions and Mental Illness: A Comparison of Germany and America, *by John Marshall Townsend. Chicago, Ill., University of Chicago Press, 1978, 148 pp., $11.00.*

Townsend, an anthropologist, contrasts what he calls the social role approach to mental disease with the clinical universalist approach. The social role approach implies that there should be a broad cross-cultural variety in symptoms because symptoms are social roles and should be specific to particular cultures. Traditional clinicians claim that the signs and symptoms of mental disorders (particularly schizophrenia) are universal and that societal reactions to these symptoms are similar throughout the world.

A variety of tests given to approximately 100 mental patients, 100 mental hospital staff, and 500 high school students from both the United States and Germany revealed massive differences between German and American concepts of mental disorder. Germans tended to believe that mental disorder is biologically caused and is not affected by the environment. Americans tended to believe that mental disorders are, at least in part, a question of personal willpower and are strong-

ly influenced by factors in the social situation of the individual. American patients were more likely to consider mental disorder "curable" than German patients. American patients also tended to cite disturbed or bizarre physical characteristics as "diagnostic" criteria of mental illness, whereas German patients tended to cite disturbances of mental functions, cognition, and judgment. These differences affected the ways in which patients coped with the hospital setting. For instance, 79% of the German patients felt that only a doctor could decide when a patient should be released, but only 49% of the American patients gave this opinion. American patients emphasized compliance with hospital regulations to obtain release (47%), but only a few German patients gave this response (5%).

Townsend reviews the literature on social labeling and clinical universalism and comes to the conclusion that neither side is right. He argues that some of the confusion is caused by the different patient populations considered by the opposing theorists. He suggests that less extreme or middle-range cases may be more susceptible to social labeling and so are cited by theorists who favor this viewpoint, while more extreme cases provide better examples for universalists. He proposes a model that he feels reconciles the two camps. However, this leads him to the psychiatrically dubious position that, at least at one level, psychiatrically disturbed patients in the beginning consciously "put on" their disturbed behavior.

I was disappointed with this book. The author approaches the problem of mental illness from the viewpoint of the social role theorist, an approach that is somewhat confusing to psychiatrists because it uses some psychiatric terms (e.g., "conversion") in a different sense. Acceptance of the sick role by mental patients does not necessarily signify conversion to the disease concept but may be a progressive step, as in conversion hysteria and hypochondriasis. Development of additional symptoms during hospitalization may be due not to the prescribed and imposed sick role but to ego regression.

Because of its focus on social role, this study does not explore other aspects of transcultural differences in psychopathology. I would suggest that length of hospitalization of mental patients depends more on their clinical condition than on other considerations. Townsend conveys the impression that the opposing camps of clinical universalists and social theorists are large, but in fact most contemporary psychiatrists occupy the middle ground. Although he tries to reconcile the social role approach with the biological approach in one chapter, a much more comprehensive view of the problems involved seems to be needed. In particular, the complete disregard of intrapsychic problems in this book is regrettable.

E.D. WITTKOWER, M.D.
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Language and Cognition in Schizophrenia, *edited by Steven Schwartz. Hillsdale, N.J., Lawrence Erlbaum Associates (New York, N.Y., Halsted Press, John Wiley & Sons, distributor), 1978, 276 pp., $18.00.*

This volume presents the research efforts of major investigative teams who have examined the language of schizophrenic patients as a sample of their abnormal behavior. The hypotheses tested range from the idea that language and cognitive style are aberrant social phenomena in the interpersonal process of the schizophrenic patient to the idea that they are related to asymmetrical brain function. The variou measures described in the studies reported are exact, e haustive, and sometimes excessive. A critique leveled b one of the authors is that each chapter seems to justify th validity of a hypothesis by "good" research design. Sever chapters are difficult to read because they contain exhau tively detailed data but only a limited discussion of co clusions from these data.

In a chapter on the lack of progress in the understanding c cognition in schizophrenia, Blaney points out that a failure study proper subsamples of schizophrenic patients is a maj error. I have also often wondered whether poor communic tion efforts by schizophrenic patients compared with contr subjects might be explained by a major difference in motiv tion.

The chapter by Oltmanns and Neale provides a ver thoughtful critique of some of the best research efforts in th scope of this volume. These authors direct their attention t distractibility in schizophrenia as a major psychological de cit that seems to be correlated with genetic predispositio Building on the reader's preparation by the preceding cha ters for the investigation of a biological basis for the hypot esized psychological deficits in cognition and perception schizophrenic patients, Shimkunas develops a very readab analysis of the data regarding the hypothesis that cerebr asymmetry accounts for some major theories—Chapma and McGhie's "filtering deficit" hypothesis, Salzinger "immediacy" hypothesis, and Bleuler's "associational deficit.

The editor of the book does not attempt the impossible his chapter; he does not summarize and integrate the maj conclusions of the rest of the chapters. Rather, he presents review of important literature and published critiques of th substantive theories of cognition and language as behavi variables in the schizophrenic syndrome. On the basis of th review he produces an inclusive schema for a more "defin tive" study of schizophrenic language.

This volume provides a description of the "state of th art" as well as an outline for more definitive work needed t establish a fuller biological and psychological understandin of cognitive, perceptual, and communicative behavior in th schizophrenias.

JAMES R. STABENAU, M.D.
Farmington, Conn.

Power and Authority in Adolescence: The Origins and Resolutions of Intergenerational Conflict. Publication 101, *by th Group for the Advancement of Psychiatry Committee c Adolescence. New York, N.Y., GAP, 1978, 213 pp., $6.5 (paper).*

This unusually well-written and balanced monograph is con cerned with power struggles and accommodations betwee the generations. It addresses broader issues as well. The au thors acknowledge that the adolescent/adult interface ha changed dramatically since work was first started on th monograph. At that time adolescents and adults in Weste culture seemed much more sharply at odds with one anothe —divided over such issues as Viet Nam and drugs. Howeve the present work is not irrelevant. Power and authority issue between the generations will always be germane because th progressive assumption of authority is necessary to a healt adolescence and the relinquishing of some authority is re quired of adults.

The authors consider power and authority issues from in-apsychic, intrafamily, intracultural, and intercultural per-ectives, maintaining a primarily psychoanalytic orienta-on. Considerable weight is given to the shift away from a ell-defined patriarchal society. It would have been of inter-t if the authors had searched for other, smaller changes in ılture that might account for the increased polarization be-/een the generations 5–10 years ago and for its subsequent minution. The committee did not assume that the shift om a strictly patriarchal model applies equally across all cioeconomic groups. In fact, I found the authors' sensitivi-to "minority" perspectives, whether in terms of race, ec-ıomics, or sex, particularly praiseworthy.

Approximately one-third of the book is concerned with a ychoanalytic review of child development. This might ap-ar to be an unnecessary expenditure of space, since so ny excellent descriptions of childhood development are ailable elsewhere. Careful reading, however, shows that e authors weave the tracing of power and authority issues roughout their consideration of child development. The imary determinants of such issues take place in the forma-e years of child development within the context of the nu-ar family. The vicissitudes of these struggles between the nerations in childhood can be traced specifically to each ase of psychosexual development. In addition to under-nding this from the viewpoint of the individual child, the thors illustrate how parents who have had difficulties in solving certain of their own conflicts at various levels of velopment are prone to interfere with their children's res-ıtion of similar or dissimilar conflicts.

Having retraced childhood development from the point of ew of the authority/power issue, the authors examine cer-n dynamics and phenomena of the authority gap (e.g., nflict within the family, student-school conflict, sexuality, d drug use). They also include an interesting discussion of w adolescent musical tastes portray many adolescent au-ority and power issues.

The role of psychiatrists in helping families deal with ado-scent/adult authority issues is briefly presented. "Negotia-n" as a therapeutic technique is offered as a model for ervention in family therapy. Although the authors are reful to indicate that there are many other ways to ap-oach family therapy, the fact that this is the only model esented in the book seems somewhat a departure from its herwise carefully balanced and even-handed presentation. owever, it may be that negotiation is a particularly suitable hnique for families trying to deal with authority and pow-issues. More clinical examples scattered throughout uld have been helpful but would have compromised the evity of the monograph. All in all, the book accomplishes aims in a sensitive, optimistic, and constructive manner.

HOWARD S. SUDAK, M.D.
Cleveland, Ohio

e Hite Report: A Nationwide Study on Female Sexuality, *by ere Hite. New York, N.Y., Macmillan Publishing Co., 76, 438 pp., $12.50.*

This report is a compilation of 3,000 responses to 4 dif-ent questionnaires designed by the author. The partici-nts were a self-selected group of women recruited through ilings to various national women's organizations, through urch newsletters, and through notices placed in several gazines. Although the validity of this research design is questionable, the book does replicate some findings of earlier, more carefully controlled research projects. The report finds that clitoral stimulation resulted in the most intense physiological orgasms for these women, that only 30% of the participants climaxed regularly from vaginal stimulation alone, and that there was a high correlation between a history of no masturbation and inability to achieve orgasm.

The book is organized to emphasize the content of the respondents' answers rather than to present a statistical breakdown of responses. This descriptive material of sexual feelings and practices is of some interest and value to the psychiatrist. Unfortunately, much of it is presented to create complaints about men. The author's thesis is that current heterosexual physical interaction reflects the inferior status of women in our culture in general. This thesis creates a bias in her presentation of material. For example, in dealing with responses regarding stimulation of women by men, the author emphasizes men's selfishness, ineptness, or, at best, ignorance. This bias becomes particularly evident in the chapter on lesbianism, in which, by contrast, lovers are portrayed as invariably sensitive, considerate, and skilled.

The author pays little attention to the woman's role in letting her partner know what she needs; she represents the man as unwilling to listen. The author's bias also causes her to characterize intercourse as an overvalued form of sexual interaction, developed in the interest of procreation and male satisfaction. The majority of her female respondents, however, claimed that intercourse was important and gratifying, whether or not it was their primary means of achieving orgasm.

The more positive aspects of the book—the chapters on the importance of touching and on varieties of sex play that do not necessarily end with intercourse—are echoed by most sex therapists today. The book also raises several important social issues, including the effects of the sexual revolution on woman's status in society, the sexual options for today's older woman, the effect of a woman's economic position on her sexuality, and sexual pleasure as a panacea for societal ills. From the psychiatrist's point of view, little emphasis is placed on intrapsychic dynamics or interpersonal needs as factors in sexual behavior.

VIRGINIA A. SADOCK, M.D.
New York, N.Y.

Early Child Care in Poland, *by Maria Ziemska. New York, N.Y., Gordon and Breach, 1978, 148 pp., $16.75.*

This book is the eighth in a series of monographs on early child care in various countries, including Hungary, Sweden, the United States, Switzerland, Britain, France, and Israel. As the author points out in the introduction, it is essential that the reader have some knowledge of the history of Poland in order to understand the vast changes that have occurred there—the destruction of institutions and its effects, particularly on the family unit. The country was partitioned by Russia, Prussia, and Austria in the late eighteenth century, and these countries continued to influence Poland for many years. Some strides were made in unifying the country between World Wars I and II, but the latter war resulted in the catastrophic loss of some eight million Polish people, devastating families and leaving millions of children without parents. This book tells of the efforts to rebuild Poland in the context of a socialistic political system of government.

Some of the efforts have been more successful than oth-

ers. The state regulates most areas of life, including child rearing. Prenursery schools have been established for the care of infants up to three years of age so that both parents can work. Mothers can be given maternity leave with pay, and this is encouraged. There are nursery schools for children from ages three to six, and first grade begins when the child is seven. Attempts are made to give all infants and children adequate medical and psychological attention, but the author readily admits that there are many things yet to be accomplished. There is a shortage of personnel in some of the schools, but efforts are being made to train more professionals.

Throughout the book the heavy flavor of socialism is obvious. Nevertheless, the Polish people have a strong sense of the family unit and are attempting to provide for the physical and emotional needs of their growing children. There is a recognition that children who spend at least part of the time in their own homes do better than those who are full-time in almost orphanage-like care. Foster home placement and adoption are encouraged.

This book is not primarily aimed for the psychiatric reader. The author pays much more attention to maintenance of physical health and education than to emotional development. Although the book is not particularly well written, it should be of interest to those who want to know more about Poland and how the Poles are dealing with child rearing in the present social and political system.

STUART M. FINCH, M.D.
Tucson, Ariz.

Currents in Alcoholism, Vol. III: Biological, Biochemical, and Clinical Studies, *edited by Frank A. Seixas, M.D. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1978, 589 pp., $31.50.*

Currents in Alcoholism, Vol. IV: Psychiatric, Psychological, Social, and Epidemiological Studies, *edited by Frank A. Seixas, M.D. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1978, 487 pp., $26.50.*

This is the second set of volumes resulting from the annual National Alcoholism Forum jointly conducted in May 1977 by the National Council on Alcoholism and the American Medical Society on Alcoholism. These two volumes contain the work of 183 investigators reporting 76 studies.

As was true of the first two volumes, these are not books to be read cover to cover to find out all about alcoholism. Rather, they represent a unique opportunity for both new and veteran alcoholism investigators to share ideas and capture those ideas in print. As such, these volumes are important as markers of where we are trying to comprehend this very complex disorder.

There are four summarizing chapters for the project reports that follow. These are "Progress in the Study of Ethanol's Actions in the Brain" by Goldstein, "Progress and Perspective: Clinical Research in Alcoholism, 1967-1977" by Sellers, "Psychology/Psychiatry and the Alcoholic" by Schuckit, and "Social and Epidemiological Research" by Irgens and Jensen. Some bits and pieces that attraced my interest include the following:

Alcohol withdrawal is a central autonomic outflow, not a peripheral response. Exercise reduces alcohol consumption in rats. Would it do the same in humans? We are learning more about the effects of acetaldehyde on the mitochondria of the liver, heart, and brain, but we do not know why body manufactures aldehyde dehydrogenase in excess of need to metabolize internally produced alcohol. The amou of time it takes for the "zonk" effect from intoxication subside depends on the complexity and novelty of the ta A simple sweat test has been devised to determine if a p son has been drinking and how much. This could be an fective monitoring device. When we look for new agents reduce the severity of alcohol withdrawal, alcohol its should not be forgotten; it is safer than some drugs in curre use.

Alcohol increases feelings of self-worth in adolescents. 192 heroin addicts, 37% were also alcoholic. Nonverbal se sitivity in counselors correlates with therapeutic success. coholics presented with the fixed-interval drinking-decisi who chose not to drink did best on follow-up. A standa depression scale showed 66% or more of an alcoholic sam to be depressed, but clinical examination of the same sam revealed only 9% to be depressed. Drinking in the morn may be more related to severe depression than to addictic Alcoholics with high extraversion and neuroticism sco have lower tolerance to alcohol and show greater cogniti impairment. The Shipley Hartford is the best test for orga brain damage and demonstrates that recovery in the first days is not complete. There continues to be a belief that ducing total consumption of alcohol will reduce the numb of heavy drinkers. A first-time arrest of an adolescent possession of alcohol does not indicate serious problen but multiple arrests do. Alcohol use among high school s dents continues to rise slowly, but marijuana use is incre ing more rapidly. A study of a statewide treatment system Oklahoma showed that more costly programs do not tr the more impaired, as had been expected, and that there no evidence that greater cost leads to better results. Ear stage alcoholics are less likely to be abstinent than late-st alcoholics after treatment.

ROBERT A. MOORE, M.D.
San Diego, Calif.

The Child in His Family, Vol. 4: Vulnerable Children, *edit by E. James Anthony, M.D., Cyrille Koupernik, M.D., a Colette Chiland, M.D., Ph.D. New York, N.Y., Wiley-Int science (John Wiley & Sons), 1978, 660 pp., $25.95.*

The appearance of this volume is one more step in the pu lication of papers originally prepared for the 7th Congress the International Association for Child Psychiatry and All Professions held in 1970. The format and organization these volumes have been described in previous reviews the *Journal*. The opening paper, "A New Scientific Regi to Explore" by Anthony, was the Presidential Address the 1974 Congress.

Anna Freud sets a comfortable note in the prologue pointing out that the concept of vulnerability is the proce of development itself. A number of papers return in one w or another to this theme, stressing the critical relationsh among origin, life process, family relations, and commun activities in the life of the child who has difficulty in copi Anthony's paper actually sets forth a covert ambivalence our society: "In the child rearing methods, we are rais children who no longer care to rear children." This proph ic statement is related to the interactions in the family str ture that will beset the child of the future.

The groupings made by the editors include the physi

spects of vulnerability, the psychodynamic approach to ulnerability, the vulnerable infant, vulnerability and parent, nd a fascinating series of papers titled "Vulnerable Chil-ren of Madness." Continuing the theme, another section oncerns vulnerability of the disadvantageous environment nd, finally, the life history of the vulnerable child.

Preventive measures in vulnerability serve to conclude the olume with a return to the theme in Solnit's "Retrospect on ie Vulnerable Child." His paper points out the need for in-reasing the maximum potential of the family in the commu-ity. He notes, however, that at times a regression through lness may provide a moratorium so that the continuity of fect in the environment can provide a critical renaissance f the child's worth as opposed to his vulnerability. His com-ient, "Bringing up children is not the same as preparing iem for life," is a note included in a variety of the papers.

I found certain papers more exciting than others. A pithy aper by Erlenmeyer-Kimling concerning research focuses matched by an excellent paper on the long-term rehabilita-on of the retarded by Clark and Clark. Yvon Gauthier's udy of the fantasy life of vulnerable disadvantaged children early describes a background of the need for care with iese children. Two articles, one by Park on the positive val-e of parents and another by Brown on positive outcomes of are, set a tone rather different from the many studies of the ifficulties children face. Moreover, some of the papers in ie life history section include optimistic follow-ups.

For those of us who worry about small children, the de-cription of an excellent preschool program by Vaughn and ssociates is clear, realistic, and well-formulated as a teach-ig program. The entire section on vulnerability and mad-ess offers not only clinical pictures but some serious guide-nes for the clinician dealing with the problems of children ving in families in which mental illness is an environmental iven. Bruno Anthony, for example, notes that children of ianic patients have considerably fewer psychological in-olvements with their parents but that the children of schizo-hrenic patients get locked into the illness process; the out-ome of the process is thus altered by the child's interaction.

Studies of this sort point the way to experiments in care nd are supported by Bohman and Sigvardsson's 18-year udy of adopted children. Their feeling is that adopted chil-ren fared better in many instances than other children laced in foster homes or left in difficult home settings.

The epilogue by Mahler should not be neglected. As one ight expect, she uses the theme of separation and ties it to ulnerability. Her short paper is a biography pointing out the mely value of transitional phenomena as well as of separa-on in providing a moratorium (like the "fallow year" theo-y) that enables one to reorganize one's talents, take advan-ge of one's anxieties, and go forward in a positive fashion.

HENRY H. WORK, M.D.
Washington, D.C.

epot Fluphenazines: Twelve Years of Experience, *edited by rank J. Ayd, Jr., M.D. Baltimore, Md., Ayd Medical Com-unications, 1978, 160 pp., $15.00.*

Frank Ayd organized an international symposium on de-ot fluphenazines at the Sixth World Congress of Psychiatry i Hawaii. The proceedings of this conference are published i this monograph. The papers focus on a common theme in n interesting and convenient package of scientific informa-ion that provides an update on the progress of maintenance treatment with depot neuroleptics. Indeed, I cannot help but note that these interesting and clinically relevant papers satisfy the criteria for a good continuing medical education program. Because maintenance treatment is so widespread and important this book is of value to any psychiatrist, but it is particularly important for the clinician who is involved with the long-term treatment of patients suffering from chronic illness.

Several authors discuss their clinical experience (e.g., Donlon, Imlah, Marriott, and Heinrich). Docherty provides analysis of the studies on the degree of prophylaxis of depot medication compared with placebo or oral drug. He calculates the percentage of relapses on depot drug and divides this by the percentage of relapse on placebo or on short-acting oral drugs. He calls this index the prophylactic effect or PR index. I have a theoretical disagreement about his use of the percentage of patients relapsing. There is conceptual merit in asking whether the rate of relapse of patients on maintenance drugs or placebo is constant over time (1). As patients relapse, they are dropped from the study; obviously, then, as there are fewer and fewer unrelapsed patients, the number of unrelapsed patients is continually shrinking. This introduces an artifact in using the number of patients relapsed or the percentage of patients relapsing.

The clearest comparison of depot intramuscular versus oral drug is provided by three controlled studies. In a careful study Rifkin found essentially no difference between oral and depot fluphenazine. This finding is supported by a recently completed NIMH double-blind study (2) that found the depot drug to be equal to the oral. The other two studies reported in *Depot Fluphenazines* found that the depot drug was superior to oral fluphenazine, a finding supported by the natural history studies. One possible explanation for this discrepancy would be that some studies focus on patients who have a history of not taking their medication. In these cases depot fluphenazine would have a real advantage. Other studies may have a population of patients who take oral medication with good compliance. Docherty finds that depot fluphenazine is more beneficial in comparison with placebo than oral drug. However, the studies with depot drug generally lasted for 6 to 9 months, but the studies of short-acting drugs often took 24 to 48 months. Use of percentage relapse may create an artifact here. Even so, the general trend of the results suggests that depot fluphenazine might be better than oral maintenance medication.

Leff reviews the combined results of studies of the families of patients receiving maintenance drug or placebo. Relapse occurs in patients who come from families in which there is a high degree of expressed emotion (e.g., critical comments, hostility, and emotional overinvolvement). This obviously opens up an important lead for future investigations. It appears that some families are more successful at keeping their family members out of the hospital than others. If the interpersonal psychosocial supports of these families can be elucidated, these principles could be applied to families who do not do it naturally.

There is much information of real substance in this volume. These carefully collected hard data are needed as grist for an interesting methodological critique. If only clinical anecdotes were included, there would be little to say. This volume has the special merit of providing a good continuing educational program. It has a coherent educational purpose in that it deals with the practical and important clinical question of depot medication and has a number of high quality papers presenting data and opinions about this clinically important issue. It is a book that must be read by physicians dealing

with chronic schizophrenic patients. I would give it an excellent recommendation.

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Law and the Mental Health Professions: Friction at the Interface, *edited by Walter E. Barton, M.D., and Charlotte J. Sanborn, A.R.T. New York, N.Y., International Universities Press, 1978, 319 pp., $15.00.*

This book is the product of a conference held at Dartmouth College in August 1976. The contributors are a distinguished group of lawyers and mental health professionals who have been active at the interfaces of law and mental health systems. The staggering escalation of legal activism in the mental health field is borne out by the estimates of attorney Bruce Ennis of the American Civil Liberties Union in his contribution. Ennis states that in 1971 there were about 10 lawyers with "substantial training and experience in representing mental patients in test case litigation," whereas in August 1976 there were "more than a thousand" such lawyers. He estimated that in 1976 there were 300 test cases being litigated on the rights of patients. This represents an enormous involvement of both lawyers and mental health professionals in legal proceedings, particularly in public mental health areas. Despite all this activity, Zigmond Lebensohn observes that "there is little evidence that this legal attention has done mental patients much good."

There are 13 chapters in this book devoted to 6 substantive legal issues: Recent Court Decisions and Practice, Informed Consent, Malpractice, Confidentiality, Competence and Responsibility, and Patient Rights and Involuntary Admissions Limited to Dangerousness. Two or three authors present differing points of view in each of the 6 areas. This creates a mildly adversarial structure, but these are all men and women of goodwill who can be characterized as trying to reason together over major controversies that are currently unsettled and unsettling. There is anguish but no acrimony. The lawyer contributions (6 of the 15 authors and co-authors) are all experienced in communicating with the mental health professions and in general are lucid and not overly legalistic in their presentations.

The chapters are superior for the most part, but there is a range from excellent to average. These proceedings were ambitious in scope and complexity of the issues confronted. The book is certainly worthwhile, at the very least as an updating of the evolution of important areas that affect mental health practice. To a degree the authors presage what is coming as well. In particular, informed consent to treatment appears to loom as the newest escalating conflict between the two professions. Private practitioners will not be excluded from legal actions involving this vexing issue.

There appear to be glimmerings of at least empathy and some understanding between the disciplines in this work. For example, attorney Bruce Ennis acknowledges that test case litigation is at best a "blunt instrument." Perhaps such efforts as this book represents, in which reasonable men women gather and communicate in good faith and with tual education across disciplinary lines, will lead to gr equity for all, not the least our patients.

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Ethical Issues in Death and Dying, *edited by Tom L. B champ and Seymour Perlin. Englewood Cliffs, N.J., P tice-Hall, 1978, 368 pp., $8.95 (paper).*

Although this collection of readings is organized for un graduate courses, much of it is potentially useful for med students, for their teachers, and for other physicians. editors line up authoritative statements on two or more s. of such controversial issues as the determination of de the morality of suicide, the rights of the dying patient, thanasia and natural death, and the significance of life death.

Death seems to be a subject that is here to stay in hig education; this book makes it possible to review a well lected representative sample of the rapidly multiplying lit ture. It is concerned not only with ethical issues but w biological, philosophical, legal, and psychological aspect well.

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Ethological Psychiatry: Psychopathology in the Context Evolutionary Biology, *edited by Michael T. McGuire, M. and Lynn A. Fairbanks, Ph.D. New York, N.Y., Grune Stratton (Harcourt Brace Jovanovich), 1977, 219 p, $15.00.*

Ethological studies of animal behavior begin with obser tions of animals in their natural environment. From the observations hypotheses are derived that are experimenta tested. During testing, variables are systematically manip lated to determine the immediate causes and consequenc of behaviors, how behaviors are organized, and how th appear, change, and disappear over time. Hypotheses abc a behavior's adaptive functions, either in the present or the species' evolutionary past, are usually part of a final e planatory scheme. Abnormal behaviors are viewed agai the background of corresponding normal behaviors, a adaptive functions of even pathological behaviors are cc sidered.

Ethology (originally a branch of zoology) and comparati psychology have become increasingly indistinguishable, a the arch-Skinnerian view of animal behavior as infinite plastic is now rarely encountered. Members of any speci learn some behaviors easily and others only with extrer difficulty. Study of the lives of animals in their natural en ronments (as opposed to study of behavior in a Skinner bo often explains why.

Whether ethological theory can validly be applied to th study of human behavior is currently controversial. Are h man lives so variable across and within societies and shaped by learned and nongenetically transmitted langua and culture that evolutionary considerations are m leadingly irrelevant? Sociobiology, a logically and historic ly related subfield of ethology, relates behaviors to the cc

)t of inclusive fitness, the idea that evolutionary pressures not on individual organisms competing against all others on individuals who bear genes that they share in increasly diluted fashion with increasingly distant relatives. To ciobiologists, altruistic acts function to increase the sural of one's own genes. Again, to what degree language l culture emancipate humans from selection pressures opting in this way is hotly and acerbically debated.

f ethology and sociobiology are applicable to human be-vior, they are potentially highly relevant to psychiatry. e evolutionary history of our species ultimately provides source of Freudian drives, Jungian archetypes, and Adan strivings for power. If an evolutionary perspective can shown to be relevant to the understanding of human be-vior, it can provide a framework for reconciling these and er apparently conflicting conceptualizations of the ultite well-springs of human activity. Further, even if human iavior is so culture-determined that evolutionary considtions cannot add to our understanding of it, ethology's phasis on observing behavior in natural contexts (in con-st to psychiatry's traditional predilection for self-reports behavior) is still welcome. The methodology for such obvational work on problems of human behavior is becomincreasingly refined.

For these reasons, *Ethological Psychiatry* is a valuable ntribution. Its 10 chapters focus on the study of human iavior, especially theoretical issues. Three chapters are eral and theoretical, 3 deal primarily with models of psyatric dysfunction, 2 discuss the behaviors of psychiatric ients in psychiatric settings, and 2 discuss the implicais of ethology for psychotherapy. Without exception, the lity of the papers is high. They reflect each author's derstanding of the current state of the field. Most contribrs acknowledge that knowledge of the ethological paramrs of human behavior is still rudimentary, but a few sugt more certainty about the true nature of psychopathologstates than the data presented and cited warrant. The ok also offers critics of the position it exemplifies a numof easily assailed targets, e.g., the implications for psytherapeutic practice suggested by a few contributors will ke even sympathetic psychiatrists as unsupported.

thological Psychiatry delineates an evolutionary perctive of behavior and discusses the issues involved in lying behavior by direct observation. As such it will be ful to psychiatrists who have been looking for an orgaed exposition of the salient issues in ethology and their ential relevance to psychiatric theory and research.

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dc Principles and Techniques in Short-Term Dynamic Psytherapy, *edited by Habib Davanloo, M.D. New York, V., SP Medical & Scientific Books (Spectrum Publtions), 1978, 549 pp., $30.00.*

This book is a summary of the proceedings of the First and ond International Symposia on Brief Psychotherapy held Montreal in 1975 and 1976. It features the work of Peter neos, D.H. Malan, and Habib Davanloo, but there are alcontributions by Judd Marmor, Hans Strupp, and others.

The principal authors are psychoanalysts, and their chief pose is to demonstrate the fruitful application of psychoalytic principles to brief psychotherapy. They wish not only to show the effectiveness of this form of treatment, to which they are strongly committed, but to explore its limits. In their view (and most outspokenly in the writings of D.H. Malan), brief psychotherapy is not simply supportive help for relatively healthy persons who find themselves in a situational crisis. The authors feel that, through the application of analytic principles, brief psychotherapy can produce significant character changes and deal with issues at the core of the personality rather than just with the more superficial aspects of adjustment. Malan also feels that analytic brief psychotherapy can be applied with great benefit to patients with serious neurotic difficulties as well as to those with milder problems.

Each author approaches the patient in his own characteristic fashion. Sifneos, for example, often uses confrontation and anxiety-provoking questions, while Malan emphasizes interpretation of the transference and the technical importance of linking it with childhood. They have in common a psychoanalytic orientation and a very active therapeutic style designed to help patients focus on what seems clinically most relevant at the moment.

Does brief psychotherapy regularly accomplish what the principal authors believe it accomplishes, or are major changes and personality restructuring during short-term treatment more a matter of serendipity, as Marmor suggests at one point in the discussion (p. 189)? The reader will be able to draw his or her own conclusions because one of the very positive features of this book is the generous use of clinical material and the openness with which it is presented. More than a dozen interviews are reported verbatim, and the reader has the opportunity to observe how the patient is approached and the material developed. The skill of the therapists is very evident, but I also got a sense that, to make time count, they try almost too hard to acquaint the patient with his fundamental dynamics. Thus, for all the emphasis on interpretation, their interventions often seem more educational than truly interpretive. They are inclined to explain and point out to the patient what his unconscious contains rather than facilitate the progressive clarification and unfolding of emerging preconscious material. Therefore, insight is seen more as something imparted to the patient than developing within the patient as constricting defenses are gradually loosened. In keeping with this stance, not much is said about the role of working through and the limitations that time constraints inevitably place on brief psychotherapy.

Perhaps the book's weakest link is the review of the literature, which does not quite do justice to the topic. There is also a tendency throughout to treat the contributions as "new" even though they deal with issues that have been in and out of the psychoanalytic literature consistently, particularly since the work of Stekel, Rank, and Ferenczi. These caveats aside, this is a useful book that combines solid material and a thoughtful presentation with a dash of controversy. It will be read with interest.

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Psychopathic Behaviour: Approaches to Research, *edited by R.D. Hare and D. Schalling. New York, N.Y., John Wiley & Sons, 1978, 378 pp., $32.00.*

This book contains 20 chapters that are revised versions of papers presented at an international conference on psychopathic behavior held in Les Arcs, France, in 1975 and sever-

al additional chapters written later. The conference was supported by the Scientific Affairs Division of NATO and attended by people from 13 countries whose areas of interest were in psychology, sociology, psychiatry, psychophysiology, genetics, criminology, and corrections.

The authors write in a condensed, tight style and add a long bibliography to each chapter. Because of the complexity of the presentation, with reviews of controversial points and tentative hypotheses, I shall present only a small portion of the material with the hope of conveying its value. The book is a must for serious students of psychopathy.

The first 3 chapters discuss the concepts of psychopathy, sociopathy, and antisocial personality. Lack of clarity in these concepts makes for major problems in the selection of both psychopathic and control subjects for research. For example, a commonly used source of psychopathic subjects is the prison, but is the prisoner a special subtype of psychopathy? What psychological dimensions are appropriate for control subjects?

The excellent fourth chapter presents a historical overview of the concept of psychopathy. At the present time there is a basic difference between the concept that describes a type of personality defined by its social maladjustments and the concept that includes abnormal personalities defined by deviation from normality and by making either the subject or society suffer.

Chapters 5–11 are chiefly concerned with methods of studying psychopathic subjects or the application of particular techniques to investigate specific dimensions of psychopathy. For example, in one study psychopathic subjects were recruited for research by the following newspaper advertisement: "Wanted charming, aggressive, carefree people who are impulsively irresponsible but are good at handling people and looking out for number one." The subjects who answered were found to be similar to prison subjects in most ways, including a high frequency of arrests, but they differed in having a low frequency of convictions.

Chapters 12–15 discuss genetics and psychopathy. The concordance rate for criminality in 9 twin studies was 55% for monozygotic twins and 13% for dizygotic. Adoption studies show that psychopathological adoptees have a higher percent of psychopathological relatives than nonpsychopathological adoptees. Studies of children who later become psychopathic reveal an excess of stealing, incorrigibility, truancy, running away from home, discipline problems in school, and school retardation. Parents with a diagnosis of antisocial personality produce more children with behavior problems, but broken homes, association with bad companions, and social class alone do not seem to have much influence on creating psychopathy.

Three chapters deal with psychopathy, criminality, and delinquency. Internal controls are regarded as factors that inhibit criminal behavior. There is evidence that parents who emphasize motives or intent are likely to build in their children a greater resistance to temptation to break societal rules. The unprincipled opportunism of the psychopathic person is his tendency to exploit people and the system. This contrasts with the socialized person who says, "I would never have thought of doing a thing like that." Some workers believe that teaching parents how to reinforce desirable behavior and how to be better parental models themselves may reduce juvenile delinquency.

A final chapter concerns treatment. A widespread view is that no demonstrably effective treatment for psychopathology exists, but many believe that if the psychopath lives in a situation where firm but not oppressive rules are enforced and his manipulative attempts are unsuccess some benefit may follow. Milieu therapy may also help. alternative is the institutionalization of the psychopath u he has reached the age when most "settle down," usu between 30 and 40. A quotation from Aubrey Lewis repea by a writer of one of the chapters seems apropos here:

> The conclusion of the whole matter is somewhat gloor The diagnostic groupings of psychiatry seldom have sh and definite limits. Some are worse than others in t respect. Worst of all is psychopathic personality w its wavering confines. Its outline will not be firm u much more is known about genetics, psychopatholo and neuropathology.

PAUL E. HUSTON, M.D
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Clinical Uses of Dreams: Jungian Interpretations and Enr ments, *by James A. Hall, M.D. New York, N.Y., Grune Stratton (Harcourt Brace Jovanovich), 1977, 350 p \$22.50.*

Clinicians who are disenchanted with classical Freud interpretation of dreams and yet are convinced of the imp tance of dream interpretation will find this book of con erable value. It deals with the largely neglected but extrer ly important clinical contributions of C.G. Jung to dre theory and interpretation. Although Jung more or less a avowed "theory" and spoke of empirical facts, his phen enological observations and ideas are appropriately pla by Dr. Hall in the form of theory in order to compare it w others.

The book begins with a history of dream interpretat from antiquity to Freud and explains the various other th ries and the laboratory study of sleep and dreams, examin the latter from both Freudian and Jungian perspectives major thesis of the book is that Jungian dream interpreta rests not only on the personal unconscious but also on potential of the collective or objective unconscious for h ing by its creative and synthetic functions.

Classical Freudian theory emphasizes the reductive defensive analysis of dreams as disguised repressed unc scious wishes. Jungian theory postulates that dreams are unconscious compensation for distortions of consciousn the dream having a guiding prospective function. Class Freudian theory interprets the hidden, latent dream be the facade of the manifest dream. Hall points out that Ju ian theory postulates that the so-called manifest dream is actual dream and there is no latent dream. Free associa is a technique that takes one away from the "disguis dream. The Jungian technique of amplification of dream ages, with meticulous attention to all dream images, ke the emphasis on the dream itself. Hall notes that the s regulatory or compensatory function of the dream does imply teleology but a movement toward wholeness and i viduation.

The author's theory of the dream ego and its relation to the waking ego is a particularly valuable clinical const allowing distance and perspective. Despite all theories, I correctly recognizes the basic art of dream interpreta and its essential subjective nature. This is an inescapable ment in the science of clinical dream interpretation.

The brief chapter on Polyani's tacit knowing theory se to be an attempt to bridge a gap with a controversial, brill

ory. It adds nothing to the book. Perhaps it might have n replaced by a longer chapter on enactments, the dis-sion of which is relegated to the last 17 pages of the book. it is hardly enough space to do justice to painting, sand-work, imaginal techniques, guided imagery, active gination, dance, art, and poetry. Still, in all, Dr. Hall naages to give considerable balance, scope, and depth to g's ideas in this book, from typology to the central arche-e of the self from which dreams are said to come. As an oduction to Jungian dream interpretation this book is hly recommended.

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nce and Treatment: Clinical Uses of Hypnosis, *by Herbert egel, M.D., and David Spiegel, M.D. New York, N.Y., ic Books, 1978, 370 pp., $18.50.*

ne of the stated goals of this interesting volume by Her-t Spiegel and his son David is "to make clinical experi-e with hypnosis more systematic by using measurements have evolved during the past decade." Among the basic positions presented are that trance capacity and depth stable characteristics that are bracketed to distinctive sonality styles and that assessing hypnotizability is diag-tically important because it facilitates the choice of treat-nt method. The authors emphasize that one of the diffi-ies in psychiatric practice is coordinating the methods of tment with the problems and personalities of patients king help. Clinically assessing hypnotizability works to-d resolving this problem by "helping the clinician to e a better match between patient and therapist."

o achieve this objective a rapid clinical assessment pro-ure, the hypnotic induction profile (HIP) is recommended crucial in our evaluation of a patient for treatment." The is an easily learned, rapidly administered induction pro-ure that permits an estimate of trance capacity. In con-t to the Stanford Hypnotic Susceptibility Scales and the vard Group Scales, which require a long, arduous period duction, the HIP takes no more than 5 to 10 minutes. An nious score sheet facilitates recording of responses. e the HIP has been obtained and scored, self-hypnosis be readily taught the subject so that he can apply strate-consonant with his trance capacity. The HIP technique xcellently presented and illustrated, and the reader can n it without great difficulty. Obviously, expertise in the hod requires a good deal of practice.

he authors attempt to substantiate the relevance of the to psychopathological syndromes and personality quali-by presenting clinical and research data. Drawing on ek mythology, they depict three personality styles: Dio-ians, who display high hypnotizability scores and mani-extensive psychological dysfunction, role confusion, a fixed sense of inferiority; Apollonians, who show low notizability, are organized and critical, and whose con-and reason dominate passion; and Odysseans, who are nid-range hypnotizability, subject to alternative moods, function between reason and feeling. The authors con-l that their typologies may be of help in adapting tech-ues to patients' personality styles.

here is a good deal of controversy surrounding the issue he relationship of hypnotizability to psychopathological dromes. The authors present some challenging opinions emerged from their work with the HIP. Schizophrenic patients and those with obsessive-compulsive disorders are not the best of subjects, but hysterical and highly suggestible people are traditionally presumed to be the most hypnotizable. These facts come out in the scores obtained by the authors in the HIP. However, is the reason for this inherent in the syndromes themselves, as the authors imply, or in such adventitious factors as inability to fix attention, distrust, fear, and distortion of reality present in many schizophrenic and obsessive patients, contrasted with the needs for fusion and other motivational forces that make many hysterical patients peculiarly susceptible to suggestion? Clinical experience validates the ability of schizophrenic patients to enter trance states once they have established a trusting relationship with their therapist, and some of the most resistive subjects are hysterical patients who regard a hypnotic relationship as a threat. By and large we are far from possessing definitive answers regarding the connection of hypnotizability to diagnostic categories because good and bad subjects spread themselves over the entire spectrum of psychosis, borderline personality, neurosis, and character disorders.

Another debatable issue in hypnosis about which the authors take a definite stand is whether or not hypnosis is a stable and reliably measurable trait. They suggest that hypnotizability remains the same under all or most situations. There is no true clarification of this issue in the book. The HIP would seem to lend itself admirably to testing the authors view by using it with patients and voluntary subjects under changing circumstances over a sufficiently prolonged period of time. Particularly important would be a study of the effect of different therapists and operators on HIP scores. Moreover, using several induction methods in addition to the HIP would be valuable to see whether patients respond better to some techniques than to others.

The chapter on research findings is inconclusive because the studies presented possess serious methodological flaws. The first informal study involves the prediction of personality typology on the basis of psychological test batteries. Without knowledge of who did the tests, it is impossible to know whether the batteries were comparable. Interrater reliabilities of predictions were also quite low. Moreover, we are not given the criteria on which group judgments were based. Even though these judgments were statistically significant they were not meaningful because they accounted for only 10% of the variance, which the authors themselves note. The investigations focused on the HIP are also unpersuasive because of methodological flaws. In the report on the relationship of low HIP profiles to severe psychopathology, criteria for assigning patients to the health-sickness scale are not specified, and the sample was small. The authors' conclusion from these minimal data that HIP predicts psychopathology simply cannot be drawn from the slight evidence that the two are weakly associated. This is a common error in interpreting the results of positive correlations, which can never be taken as indications of causation.

The last section of the book, by contrast, is excellent. It contains innovative strategems for the control and treatment of a number of conditions, including smoking, obesity, anorexia nervosa, anxiety, concentration problems, insomnia, phobias, pain syndromes, psychosomatic disorders, conversion symptoms, and behavior disorders. These topics are discussed in detail with a good deal of clinical acumen. This section in itself makes the volume a worthwhile addition to the library of any psychotherapist.

In summary, the authors deserve commendation for writing a book that is provocative and challenging. Although the

questions the authors attempt to answer with their clinical and research data regarding hypnotizability as a diagnostic tool, as a means of matching patients and therapists, and as a way of identifying preferred treatment methods are by no means resolved, they offer hypotheses that open up a fertile area for future research. The well-presented HIP is a practical, rapid, easily scored hypnotic method, and the pointers regarding techniques and the phrasing of suggestions for a variety of conditions are valuable.

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Inheritance of Creative Intelligence, *by Jon L. Karlsson, Ph.D., M.D. Chicago, Ill., Nelson-Hall, 1978, 200 pp., $14.95; $7.95 (paper).*

Jon Karlsson has made some important contributions to the genetic study of schizophrenia, applying pedigree and adoption strategies to a population in Iceland. In this book, however, he deals not with the genetics of mental illness as such but with some intriguing hypotheses that, on the genic level, associate intelligence with myopia, creativity with schizophrenia, and giftedness or leadership with alcoholism. As speculations stemming from accepted evolutionary considerations and various biographical and family data, these hypotheses do not lack interest and may serve to spur further research. In the evolutionary sense the concept of selective advantage for gene carriers has been established for sickle-cell disease, and there have been attempts to apply this model to psychiatric illness, especially schizophrenia. However, Karlsson, in his more polemic passages, writes as if his particular hypotheses were proven facts and draws conclusions for social action whose consequences would be serious indeed.

These conclusions may be summarized by the author's questions, "Can society tolerate more mental illness in order to have increased creativity? Should man sacrifice his visual acuity to raise the overall IQ level? and Should alcoholics be supported and their reproduction be encouraged in order to have more effective leaders?" (p. 173). Karlsson also states, "It appears that abundant creative intelligence can be provided only by acceptance of a corresponding increase in mental illness, alcoholism, and myopia" (p. 173).

The book is interlaced with much expressed bitterness toward the psychiatric and academic establishment for what the author considers its antiscientific, antihereditarian bias in apportioning funds for research. To me, certainly not unaware of such trends, this is an anachronistic view of how genetics is favored in United States research psychiatry today. (Contrary to Karlsson's statement on page 129, for example, NIMH does have a unit on psychogenetics.)

There are good and balanced presentations in this book of important topics in population and biochemical genetics. Discussions of the genetics of major psychotic illness are generally informative, although they give little attention to polygenic models involving liability thresholds. The material on mental illness is influenced by the author's opinion that schizophrenia and manic-depressive psychosis are overlapping disorders with many features in common and difficult to separate for purposes of genetic study. This viewpoint also colors the descriptions of mental illness in creative individuals and their families.

Those who wish to explore the author's theories further ought to read this book. References are listed in the back by chapters, but unfortunately some of them are hard to tr down because they are not keyed to the text.

This monograph, in short, is an idiosyncratic essay, of acrimonious and crusading in tone and controversial e among geneticists. It is certain to stimulate the intellectu curious but surely is not an introduction to behavioral netics for the uninitiated.

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A Grammar of Dreams, *by David Foulkes. New York, N. Basic Books, 1978, 441 pp., $20.00.*

In this book Foulkes allies himself with Freud in the m positive sense. Both are students of the mind who use dream to illuminate the workings of the mind. The choice the dream is based on the idea that the dream is the mer activity most free of external stimulation and dominance therefore the purest example of mind function availat Foulkes describes a scoring system for latent struct (SSLS) for coding or summarizing manifest dream cont and its free associations. He intends the SSLS to be a "n ful" approach to the interpretation of dreams and to the velopment of a reliable model of the process of dreamin

He provides a lively and compelling background for cognitive-linguistic synthesis of dreams in his review of conceptual base for a contemporary model of dreaming of the empirical resources available for his synthesis fr work in dream psychology and waking symbolic behav He closes with a very brief glimpse of the potential impli tions and applications his method has for clinical and exp mental dream psychology.

Foulkes argues that a cognitive-linguistic approach dreams captures the best insights of Freud's model of dre formation and his method of dream interpretation. Foul accepts Freud's views as the best approach to the dream dreaming. Importantly, he recognizes that nonmenta models (e.g., "asemantic neural approaches" [1]) cannot dress the central issues in dream psychology and miss point of Freud's contribution.

In SSLS each "sentence" in the dream and in the f associations is coded into seven relational and nine subj categories guided by Hornian and Freudian principles relationship and significant others. These codes are t built up according to very explicit rules to provide a comp interactional network to which a specialized mathemati analysis is applicable. The potential for mathematical an sis opens the possibility for mathematical tests of such c cepts as distortion, displacement, and condensation. The sence of the approach is the avoidance of the arbitrary.

Foulkes belabors the sterility of modern dream resear His assessment of the contribution of modern dream search is debatable and worthy of a separate essay. I h peripherally explored this topic in another context (2).

The work presents a well-grounded and explicit appro to the interpretation and construction of dreams and perh of the mind as well. It is helpful to understand that priv meaning is not the only goal of dream psychology or of approach. Further, the function of the dream, which is concern in dream psychology, is unlikely to be addressed this approach.

Unfortunately, I have a number of major concerns ab the approach. For example, there is no serious empirical plication of this methodology to encourage us to accept

e crucial mathematical methodology is presented so ab-actly that I could not understand it. The circularity of free sociation as a method to explicate dream formation is ted but not adequately addressed. The limited attention en to the affective or dynamic component in favor of the gnitive is regrettable. Lastly, the author's use of nonas-ciative explanations in rule making and his rejection of am-fication techniques require further discussion and testing. This book would be valuable for the student of the mind l for the student of the dream. For the most part, it is a del of how to write a book that integrates the necessary torical and intellectual background to properly present a w contribution.

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radox and Counterparadox: A New Model in the Therapy of Family in Schizophrenic Transaction, *by Mara Selvini Pazoli, M.D., Luigi Boscolo, M.D., Gianfranco Cecchin, D., and Giuliana Prata, M.D.; translated by Elisabeth V. rt. New York, N.Y., Jason Aronson, 1978, 186 pp., .00.*

n this compact and readable book Dr. Palazzoli and her ee co-authors from Milan, Italy, present their work with izophrenic patients and their families. The text is com-sed principally of clinical examples of their method of aking up pathological equilibrium in the family by pre-ting "prescriptions" of counterparadoxical intent. The ily paradox is diagnosed and countered. Their theoretical mework borrows heavily from the work of Watzlawick, kson, and Bateson. The book is more meaningful if those lier writings and concepts are familiar to the reader. Inter-ingly, the bibliography cites no Italian sources other than authors.

Dr. Palazzoli first wrote in Italian 10 years ago about her lytic experiences with anorectic patients and their fami-s. She then turned to working with schizophrenic patients l their families and, with a team of two men and two wom-evolved the techniques described in this book. The anal-s of family interaction are astute. The interactions are lu-ly described. The "prescriptions" are delightfully inge-us and creative. At times one feels the team is utgaming" the family game playing—as indeed they are. One comes away from the book realizing again that it is easy to work with families. Nothing can be taken at face ue. Nothing is as it seems. To keep the family system in ilibrium family members behave in "pathological" ways. at appears to be reality is often not in terms of communi-ion theory. One of the remarkable accomplishments of team in communicating with the families is their ability to ue interpretations of behavior with positive constructive rtones: "You are being generous and giving to your ther by acting crazy." In less experienced hands the tech-ue could be dangerous and destructive.

The reported results are remarkable. The families showed marked improvement after only a few interviews. The families were seen once a month or at even longer intervals 10 times. Very occasionally the team extended the therapy to 20 sessions. Termination was then absolute. This book is valuable reading for any student of family interaction and therapy.

DEBORAH R. COGGINS, M.D.
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Normal Psychology of the Aging Process, revised ed., *edited by Norman E. Zinberg, M.D., and Irving Kaufman, M.D. New York, N.Y., International Universities Press, 1978, 277 pp., $15.00.*

This revised edition includes the proceedings of the 15th anniversary symposium of the Boston Society for Gerontologic Psychiatry. The first edition presented the proceedings of the first symposium. The views of Zinberg, Kaufman, Goldfarb, Berezin, Linden, and Levin on cultural and personality factors, psychosocial and sociophysiological aspects, and intrapsychic and psychodynamic considerations are well expressed, stimulating the reader to agree or disagree with the formulations presented.

The discussions of regression, recession, and libido equilibrium in the psychoses of the aging point to the differing viewpoints of psychoanalytic interpreters. The semantics of regression, object loss, dependency, and libidinal gratification are interpreted with clinical examples.

Parts two and three focus on marital adaptation and a review of current studies on sex and old age. The authors regret that adequate information is not available for determination of the effects of aging on sexuality and the psychological implications of these effects.

I found the book provocative in that some authors' interpretations of clinical examples seemed based on psychoanalytic theory to the exclusion of the physiological changes of aging. For example, Kaufman interprets the hysterialike behavior of an over 70-year-old woman in a beauty parlor as resulting from the "timelessness of the unconscious expression of id derivatives," pointing out that "fear of rape and other sexual and aggressive fears and wishes can occur at all ages." I could not accept this formulation without information about what had been recent fears of this woman. My concern is that such interpretations of intrapsychic dynamics are written as though the student should accept them as fact rather than using formulations to explore alternative bases for what is apparently irrational behavior.

The list of current references provides a source for further reading and points again to the multifactorial complexity of studies of the psychology of the aging process: the cultural, social, economic, physiological, and personality differences that make generalizations about aging individuals fictitious.

SHIRLEY M. COLTHART, M.D.
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The Human Dimension in Psychoanalytic Practice, *edited by Kenneth A. Frank, Ph.D. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1977, 202 pp., $14.75.*

In 1947, Dr. Fanny von Hann-Kende, one of my supervisors at the Columbia University Center for Psychoanalytic Training and Research, remarked that she knew of only one

article on countertransference. It had been written in Hungarian in 1932 and had been translated into German but not into English. In the 1950s, there was a beginning recognition in the literature of the analyst as a thinking, feeling, perceiving human being—a live partner in the patient-therapist dyad. The therapist's internal reactions as well as some of his or her external actions were perceived as countertransference phenomena according to the models of transference and countertransference enunciated by Freud.

Despite some acknowledgment of the "human dimension" in the psychoanalyst in the 1950s and 1960s, there were still many proponents of the "blank screen" perception of analysts, an elite who were immune to the human frailties. However, Otto Will and Harold Searles, among others, have been unabashedly candid about their subjective thoughts and feelings as well as their behavior in the therapeutic setting. They have talked about human feelings and continue to do so, rather than characterizing their responses as countertransference phenomena. In the introduction to *The Human Dimension in Psychoanalytic Practice* the editor quotes Anna Freud in this context: "The analyst and patient are also two real people of equal adult status, in a real, personal relationship to each other." (1).

This volume contains 13 highly personal contributions by competent analysts with a variety of theoretical frames of reference. Searles's contribution considers the role of mature hope in the patient-therapist relationship as well as the role of the conflicting and contradictory hopes and fears in both patient and therapist. He enunciates the importance of the therapist's changing his or her perspective to keep it consistent with the data rather than focusing on the patient's earlier perceptions and expectations. He gives a thumbnail sketch of the first few agonizing sessions with his second analyst, noting how some of his fears were allayed and his hopes engendered. He had entered this second analytic effort after having been summarily dismissed early on by his first analyst.

Fairbairn and Winnicott were the two analysts with whom Guntrip spent time in successive analytic experiences. Guntrip responds to the query, "How complete a result does psychoanalytic therapy achieve?" by noting that theory "is a useful servant but a bad master. . . . In the last resort, good therapists are born not trained and they make the best use of training." Guntrip gives detailed accounts of some of his analytic experiences and his perceptions of the analysts' reactions during and afterward. He sees psychoanalytic therapy as the interaction of the personalities of two people working together toward free spontaneous growth. The analyst as well as the analysand grows. There must be something wrong if an analyst is static when dealing with dynamic personal experiences.

Ralph Greenson's "That Impossible Profession," first published in the *Journal of the American Psychoanalytic Association* in 1966, is included in this volume. The editor notes that Greenson, more than any other prominent orthodox American analyst, has delineated the issues of the psychoanalyst as a person and the real, human relationship between the analyst and the patient. In this paper he characterizes the analyst as having complex and antithetical skills, varied and contradictory traits, and motivations that are difficult to define. He concludes by stressing the crucial importance of a deep, personal, analytic experience and a good personal life outside of one's work. These can render the impossible profession not only possible but rewarding.

Certain patients intimidate their families, school authorities, and, on occasion, therapists. Hilde Bruch notes that the therapist's failure to recognize his or her own anxiety a anger and to deal with the behavior of the patient can sab tage treatment. She gives an example of destructive, agre sive behavior on the part of an adolescent and several e amples of patients with anorexia nervosa who tyrannized their weakness and frightening physical state. Some the pists have believed that resolution of the underlying conflic will bring about appropriate eating habits and weight gain. so doing they have avoided confronting the weight gain iss and the anorexic's intimidating behavior directly. Bruch approach is definitive and effective in neutralizing the tyra ny and restoring health. This includes confrontive measur that are sometimes life-saving, as well as recognizing not o ly the low self-image of the anorexic but also the tremendo power he commands through manipulating his environmen

Harold Kelman's presentation of his philosophy of ps chotherapy is a kind of valediction. He died at age 71 wh the book was in press. His article interrelates his persor and professional histories with his philosophy. He se every patient as suffering from a multidimensional feeling "nobody-was-there-for-me" during infantile development a consequence of distortion of organic modalities. The ta of the therapist is to help the patient to experience these m dalities in an undistorted fashion as well as to experience t therapist in multiple caretaker roles. Kelman elaborates e tensively on his technique, which provides responsivene to needs but avoids subjective involvement with the patien

This short volume is remarkable in the breadth, depth, a comprehensiveness of the articles and the candor and ope ness of the authors. Parts of it will certainly touch vibra chords in every therapist who is alive and growing in t "humbling profession."

REFERENCE

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JAMES P. CATTELL, M.D.
Pittsfield, Mass.

Child Behavior Problems: An Empirical Approach to Mana ment, *by Roger McAuley and Patricia McAuley. New Yo. N.Y., Free Press (Macmillan Publishing Co.), 1978, 233 p, $13.95.*

This small volume is easy reading for the psychiatrist w wishes to begin his or her understanding of behavior thera with children, now that some of the major differences tween behavior therapy and psychotherapy have started dissolve. The approach is clinically oriented and avoids technical language and statistical emphasis that often mal behavior therapy publications so uninteresting to the p chodynamically trained psychiatrist. Good comprehens and individualized history taking is the emphasis; this p vides another common guidepost familiar to the n behavioral clinician. It is clear that the McAuleys were ba cally good clinicians before they integrated behavior thera into their work. Although they emphasize externally obse able behavior, they do not ignore the internal life of youngster.

The underlying theory of the authors' concept of "appl behavior analysis" is that deviant behavior is developed a maintained by its short-term stimulus associations and t the discovery of these associations allows a reshuffling

v the stimuli operate. The emphasis on objective mea-
ement of behavior, especially overt and easily measur-
e behavior, sometimes appears dry and dehumanized be-
se it avoids cognitive and emotional factors, the internal
l interactional life of the youngster, that are not easily
asured. However, this approach adds an important di-
nsion to the good clinician's practice, e.g., having young
ents and their families keep diaries regarding certain tar-
symptoms or problems. Self-recording can produce an
reness of behavior that is a precursor to change.
he authors logically progress from data collection to
lysis of the data to planning and goal setting to implemen-
on of methods to assessment of progress in treatment.
y discuss basic theory and definitions, limitations and ad-
tages, and pros and cons and question the theory with a
l chapter on future directions and problems.
lthough the book is divided into three parts, Assessment,
nning, and Intervention, it could just as easily fall into a
-part division, which is the way it reads. The first is for
ents. The extensive use of parents and teachers as prima-
herapists is a characteristic of behavior therapy because
emphasis is on naturalistic settings. A discussion of dis-
ine that deals with the normal problems of everyday liv-
and point systems and contracts could profitably be read
parents whose discipline tends to be inconsistent.
he second possible division deals with clinical psycho-
hological disorders and the application of behavior thera-
o them. The authors provide case examples for such con-
ons as aggressive destructiveness, stealing, school refus-
stuttering, enuresis, and encopresis. Their balanced
roach is reflected in two case presentations of child
se, one of which was successful and one unsuccessful.
se help to tease out predictors and indications for behav-
therapy and are most instructive regarding limitations.
here is no reason why office psychotherapy, which al-
ly uses many of the techniques described in this book,
not be enriched and complemented by having patients'
ents use some of these techniques. The combined use of
ious forms of interventions such as psychotherapy and
avior therapy seems more and more possible and benefi-
to the young patient and his family.

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Honolulu, Hawaii

ality of Life in Severely and Profoundly Mentally Retarded ple: Research Foundations for Improvement, *edited by C. ward Meyers. Washington, D.C., American Association Mental Deficiency, 1978, 384 pp., $14.95.*

his monograph continues the high level of professional olarship and sophistication demonstrated in two previous lications of the American Association on Mental Defi-ncy. The papers are not addressed to physicians in gener-r even to psychiatrists in particular but primarily to those olved in the behavior and adaptive processes of the se-ely retarded.
he publication comes at a most opportune time for those rged with facilitating the adaptation of this population in community. There has been a marked increase in the ıber of these people in the community in the metropolitan w York area and presumably in most large urban centers ughout the United States. The professionals involved in habilitative process eagerly receive whatever new and ful techniques are suggested by reputable research re-sources and are ready to pursue them at the clinical level. Some innovative and stimulating ideas are contained in this monograph. Techniques for understanding and altering stereotyped behavior, level of involvement of parents in speech programs, other means of nonverbal communication, and extending the use of psychological tools (the Bayley Scales and others) for more refined definition and evaluation of the severely and profoundly retarded population are among others suggested.

The authors are not identified by title or profession but by affiliation with the university or institute where their research was carried out. Some have national reputations and are identifiable, but most are less well known; their backgrounds suggest predominantly behavioral and/or educational psychology, speech pathology, and special pedagogy.

These papers are carefully planned and designed, sophisticated in their use of data and statistics, and apparently rigorously executed. They are all well documented with extensive bibliographies. Regrettably, the publication itself leaves much to be desired. The print seems small and the lines crowded with barely any margin space for those addicted to making notes. Many of the tables were especially difficult to read. Although I believe the book will appeal only to the small audience particularly interested in the severely retarded population, those few may find some stimulating and rewarding ideas, perhaps even motivation for the further research suggested by the contents.

JOSEPH MICHAELS, M.D.
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Videotape Techniques in Psychiatric Training and Treatment, revised ed., *edited by Milton M. Berger, M.D. New York, N.Y., Brunner/Mazel, 1978, 397 pp., $20.00.*

The use of videotape techniques is rapidly expanding in every area of psychiatric interest and expertise. The first edition of this volume was a welcome compendium of authoritative contributions to this expanding new field. This new edition is an attempt to bring us up to date on the rapidly expanding sophisticaation of technique and application, and it succeeds admirably in large measure. As John Spiegel states in his preface to the revised edition, "This is a 'how to' book, written by the growing number of technologically sophisticated therapists, trainers and researchers for those who are incipiently curious, definitely interested, while wishing to be instructed."

The book is organized into sections that deal with different aspects or applications of videotape technique. The first section deals with the technique of confrontation in psychotherapy. Although this section vividly portrays the technique of confrontation and reviews the considerable literature referable to this technique, little is offered in the way of outcome data either on its long-term efficacy or its ability to contribute to a shortening of the psychotherapeutic process. However, the case histories are well presented and to some degree convincing. The section on training is superb and brings together considerable expertise regarding the applicability of videotape to training in clinical psychiatry. The section by Herman Hirsh and Herbert Freed and that of Marsh Gowin and Frank Kline dealing with the use of videotape in supervision of psychotherapy are innovative and well structured. W. Douglas Weir's chapter on evaluating psychiatric learning through videotaped patient interviews is an excellent introduction to the use of videotape material for examination

purposes. However, it is limited in scope and does not include consideration of the utility of videotape-based examinations in program evaluation.

The section on treatment is fascinating because it focuses on the potential use of videotape in almost all forms of therapy, including narcotherapy, child psychotherapy, family therapy, group therapy with schizophrenic patients, marathon and encounter group therapy, individual therapy, and couples therapy. Almost all of the chapters are well written and easily understood. Again, the lack of comparative outcome data demonstrating a clear-cut improvement either in quality of outcome or decreasing length of treatment represents a limitation of the value of the contribution. However, one supposes this is a great deal to ask of an introductory text to a new and expanding field. Perhaps outcome data will be forthcoming.

The section on legal, moral, and ethical considerations presents model forms for signed release and attempts to deal with the issues of privacy and privileged communication as well as other legal implications and complications. Most of the germane issues are raised in a thoughtful way. There is an unfortunate tendency to view legal and ethical concerns over exposure of videotape clinical material as "legalism," but this is balanced by the broad view of the authors.

The final section, titled Technical and Artistic Considerations, should be of considerable help to any department of psychiatry planning or developing a videotape division. I particularly enjoyed the chapter on the technical and artistic aspects of videotape written by Harry Wilmer. One is too often exposed to videotape material that is fascinating in content but so poorly done as to be dull and aesthetically offensive.

This book is a valuable compendium of introductory information on the applications of videotape technique. To quote Lawrence Kolb's preface to the first edition, "As the educational and therapeutic applications of videotape expand and grow, we shall also expect its wider use in research concerned with human behavior." One would hope that some greater emphasis had been placed on research applicability and findings in the present volume, but it is a valuable contribution and recommended introductory reading in an exciting field.

ARTHUR T. MEYERSON, M.D.
New York, N.Y.

Alcoholism and Treatment, *by David J. Armor, J. Michael Polich, and Harriet B. Stambul. New York, N.Y., Wiley-Interscience (John Wiley & Sons), 1978, 343 pp., $16.95.*

Like a phoenix rising from its ashes the Rand Corporation report, originally published in 1976 as a monograph, returns in this volume, expanded with an 80-page index of the reaction to the storm of controversy generated on its first appearance. Its principal contribution will probably be the stimulus it gave to questioning established theory and practice. The authors, two sociologists and a psychologist, were not clinically involved with the subject population and had to rely entirely on prepared material for their statistical analysis and interpretation.

One need not in 1979 rekindle the fires that smoldered three years ago. The interested reader may wish to consult the local and national press of the time or, more succinctly, review the well written and fairly extensive apologia found in Appendix B. In spite of the methodological shortcomings inherent in self-reporting of drinking behavior and in data that are often haphazardly collected and recorded, this follow-up study is one of the largest of its kind, even though it involved a small number of subjects (14 of a group of 25). Its three major findings are worth noting, if only for the reader to check against his or her own clinical experience: 1) more than 70% of even seriously impaired alcoholics improved after treatment, 2) the fact of treatment was more important than the specific type of treatment, provided the treatment was given in sufficient amounts, and 3) the majority of improved clients were drinking moderate amounts of alcohol at levels far below what could be described as alcoholic drinking.

Although the third conclusion was the red flag that inflamed the alcoholism establishment and, especially, recovered alcoholics whose very lives depended on abstinence, clinicians who treat large numbers of alcoholics were relatively unmoved. They were only too well aware of how many patients try to prove wrong therapists who prescribe abstinence by "controlling" their drinking. This may go on for many months without any apparent adverse consequences, but the tenuous sobriety thus maintained is scarcely serene when there is a need to "control" one's drinking, a condition almost pathognomonically different from the social drinking of nonalcoholics.

The longest follow-up of controlled drinking therapy (2 to 55 months) was that of Ewing and Rouse (1), excellent clinicians who were directly involved in the treatment process. Ewing and Rouse found that controlled drinking looked promising in the first 12–18 months but in the longer run was "a universal failure."

One may perhaps forgive sociologists and medical laymen for misunderstanding the degree of immediacy implied in the language of the familiar Alcoholics Anonymous dictum that "an alcoholic is one drink away from a drunk." Of the alcoholic patients who choose to return to some degree of controlled drinking an unknown number may well be successful, just as there are diagnosed cancer cases in remission in the literature. Most physicians with a large alcoholism practice would agree that the alcoholic who can return to drinking behavior without relapsing into dependence is rare. Since we are unable to preselect such individuals, it can scarcely be called responsible practice to recommend a treatment with so little chance of success.

To set the record straight, the authors of this report do not advocate that alcoholics try moderate drinking after treatment. On the contrary, they expressly warn that "alcoholics who have repeatedly failed to moderate their drinking, who have irreversible physical complications due to alcohol, should not drink at all" (p. 295). They also clearly state that their conclusions have no relevance for future policy without further research.

It seems strange that although the authors include comment on much of the criticism of their work, they did not include a memorandum from the Deputy Director of the Division of Special Treatment and Rehabilitation Programs of the National Institute on Alcohol and Alcohol Abuse dated June 18, 1976. This memorandum sheds considerable light on the report's shortcomings. It states that the subjects in the study comprised a more diverse "problem drinking" and less "definitely alcoholic" population than the Rand researchers suggest. It also questions the acceptance of the consumption of 3 oz. of ethanol as normal drinking. The document concludes,

It is very likely that a substantial number of clients in the

samples are misclassified as definite alcoholics when, in fact, many are suffering from a variety of problems related to alcohol abuse but without the severe impairment or physical dependence which characterizes the more commonly held definitions of the definitely alcoholic. . . . Armor et al. have not established that the physically dependent alcoholic person can become a normal drinker, in large part because the assumptions and definitions adopted for the study are unjustified, appear to be arbitrary or self-serving and therefore are unconvincing.

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MAXWELL N. WEISMAN, M.D.
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search Advances in Alcohol and Drug Problems, Vol. 4, *ed-d by Yedy Israel, Frederick B. Glaser, Harold Kalant, bert E. Popham, Wolfgang Schmidt, and Reginald G. iart. New York, N.Y., Plenum Press, 1978, 485 pp., 9.50.*

This book contains an interesting mixture of critical views current research advances regarding various aspects of ohol and drug problems. For example, there are chapters aling with the biological significance of endogenous opiod ptides and the opiate receptors, brain reinforcement cens and the psychoactive drugs, animal studies of alcohol thdrawal reactions, and the role of acetaldehyde in the meolism and reactions of alcohol.

Clinical and social concerns are dealt with in such chaps as a detailed history of the tavern through the ages, pant characteristics as predictors of treatment outcome, the el of consumption and the social consequences of drinkg, alcohol use among North American Blacks, the etiolcal relationship between drug use and criminality, the reults of decriminalization of public drunkenness, and the atment of cigarette dependence.

All of these subjects are discussed in a highly technical d scientific manner that requires concentrated but interting reading. I was amazed that a book could be published quickly with such up-to-date research material and with ch an extensive bibliography of references to the current erature. The editors are to be congratulated for producing professional and scholarly treatise that must have entailed great deal of effort.

FREDERICK LEMERE, M.D.
Seattle, Wash.

andbook of Adolescence: Psychopathology, Antisocial Develment, Psychotherapy, *by Carl P. Malmquist, M.D. New rk, N.Y., Jason Aronson, 1978, 879 pp., $30.00*

Anyone who ventures to capture a whole stage of man in a gle volume must make choices. Malmquist has chosen beviors that bring adolescents to court, and his book is most teresting and valuable in this respect. It also covers a great ny other areas, but in nonforensic matters, particularly in clinical terms, the book is less detailed. Really not a handbook but a series of essays reflecting the author's interests and best read in their entirety, these 879 pages with more than 1,100 references are imposing.

The author's discussions of forensic issues are particularly interesting with respect to adolescent sexual behavior, in which are incorporated the historical background, a broad perspective on sexual crimes and deviations, and a sophisticated view of unsolved problems at the interface of society and therapist. The author's treatment of such issues as censorship and psychopathy is thoughtful and informed, and his heartfelt chapter titled "The Juvenile and the System of Justice" clearly reflects long exposure to the vicissitudes of courts and the inadequacies of treatment and punishment alike for this population.

The clinician may at first be intrigued by the author's decision to include all nosological conditions from neuroses through character disorders to psychoses under the rubric of "antisocial behavior." Inevitably those in forensic work will encounter all varieties of adolescent pathology. However, such an approach may make it difficult to identify patients encountered in clinical practice. Although the title page promises a discussion of psychopathology, antisocial development, and psychotherapy, only the second of these three is treated in depth. Illnesses such as manic depression and symptom complexes such as school phobia are scarcely mentioned, anorexia nervosa rates only a line or two in passing, and even schizophrenia receives relatively brief discussion. Medication is not covered, medical illnesses and their emotional consequences get short shrift, and the discussion of the biological area is somewhat dated throughout the book.

Despite such clinical shortcomings, this is a substantial book. Some of the discussion is extremely complete, going well beyond the adolescent age group; if other aspects are weak, this may reflect the herculean labor such an undertaking must have presented a single author. An introduction might have clarified his expectations for this work, and the book would have benefited from more thorough editing, proofreading, and indexing to render its real contributions in the area of adolescent forensic psychiatry easier to reach.

CHRISTOPHER H. HODGMAN, M.D.
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Motivation: Theories and Principles, *by Robert C. Beck. Englewood Cliffs, N.J., Prentice-Hall, 1978, 459 pp., $12.95.*

Not all efforts to systematize what is known about behavior and its determinants—i.e., the field of psychology—make great use of the concept of motivation. Indeed, B.F. Skinner's classic, *The Behavior of Organisms* (1), which is probably the single most influential treatise on psychology of this century, contains no chapter on motivation; the word does not appear in the index of that volume. Others have found motivation to be a useful integrating concept around which much psychological theory and many psychological principles can be organized. The present volume does this quite well.

This book does not deal systematically with abnormal behavior or topics in clinical psychology and psychiatry. Nor does the author deal with psychodynamic theories or concepts. Rather, the book offers an introduction to basic psychology as a natural science derived mainly from experimental work in animal and human laboratories. Using the core

notion of motivation as a starting point, the author offers succinct, historical introductions and resumés of current experimentally based thinking on a number of related topics. These include the concept of drive, activation and arousal, reinforcement theory, aspects of fear, avoidance and escape learning, experimental aspects of anxiety, conflict and aggression, and other topics. Particularly strong is the chapter on physiology of consummatory behaviors. For example, the section on hunger nicely summarizes the state of knowledge on the role of hypothalamic factors in eating, other brain areas, sensorimotor and motivational deficits, and the more peripheral physiological factors such as stomach distension, osmotic balance, and intestinal volume. The literature on specific hungers is also summarized.

Because of the breadth of this volume, the author deals with no topic in great depth and does not include the most recent experimental findings. However, the volume will be very useful to the clinician or investigator who wants an accurate, comprehensive, understandable, and brief review of one or another aspect of behavior viewed from a motivational perspective.

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JOHN PAUL BRADY, M.D.
Philadelphia, Pa.

Annual Review of the Schizophrenic Syndrome 1976–1977, *edited by Robert Cancro, M.D. New York, N.Y., Brunner/Mazel, 1978, 636 pp., $27.50.*

This impressive and scholarly collection of world literature in the field of schizophrenia research is a valuable reference volume. The editor's encyclopedic knowledge is reflected in his concise and helpful comments about papers that were not included in the 31 chapters. There is a strong tendency toward highly abstract and theoretical papers. This fits with the relative paucity of direct clinical material and with the book's role as a reference work as opposed to a textbook.

Strong emphasis is placed on genetic and family studies of schizophrenia. An outstanding chapter is that of Julian Leff on "Schizophrenia and Sensitivity to the Family Environment." Leff clearly demonstrates the importance of expressed emotion, by which he means criticism and hostility by a parent or spouse at the time of admission of a schizophrenic patient. This variable was found to be significant in terms of relapse and rehospitalization within one year. A standardized test that would allow clinicians to reliably measure this variable would be helpful in formulating post-discharge plans. Another related variable was the patient's spending 35 hours a week or more in direct face-to-face contact with a critical relative. This factor also increased the relapse rate. The findings suggest that under certain circumstances schizophrenic "withdrawal" may help the patient avoid stressful interaction within the family.

Perhaps the finest chapter is that by Robert Heath on "Brain Function and Behavior" reprinted from the *Journal of Nervous and Mental Disease*. Heath gives a clear, well-reasoned overview of the research program in the Department of Psychiatry and Neurology of Tulane University since 1949. The research has focused on developing treatment for patients resistant to commonly used therapies. work has often been controversial, but Heath clearly plains the need for human experimentation (such as electrode implants, done stereotactically) as the logical tension of animal brain research done in the Tulane labo ries. He has demonstrated that the septal region "has ur ingly shown aberrant electrical activity, in the form of sp and slow waves, during psychotic episodes, regardles cause" (p. 197). Heath has made important advances in c lating brain activity with specific emotions. His findings "do not coincide with conventional textbook presentati (p. 197), probably indicating that he continues to work or frontiers of scientific knowledge. He provides an eleg though complex, neuroanatomical summary in his figur (p. 214), a diagram of a monkey brain against the backgrc of stereotaxic coordinates. This chapter is an essential re of how Heath and his colleagues have advanced our un standing of the anatomical-functional substrate for emoti

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Drugs of Abuse: Handbook of Psychopharmacology, Vol. *edited by Leslie L. Iversen, Susan D. Iversen, and Solor H. Snyder. New York, N.Y., Plenum Press, 1978, 412 p $32.50.*

Although this is a review of volume 12 of the *Handboo Psychopharmacology,* it is impossible to do justice to volume without mentioning the scope of the *Handbook* whole. Other volumes have dealt with classes of drugs v significant abuse potential. The title of volume 12 is r leading because one cannot gain a comprehensive overv of abuse by reference to this volume alone. The 14 volu of the *Handbook* have attempted to cover basic neuropl macology (volumes 1–6), behavioral pharmacology in mals (volumes 7–9), and human psychopharmacology (v umes 10–14). Each volume presents an in-depth review given topic by authorities in the field. The *Handbook* s ceeds very well in providing an encyclopedic overview what is known about psychopharmacology.

This volume consists of 7 chapters, each of which i comprehensive review of a given area. These chapters not easy reading for the generalist; most of them are tecl cal, sophisticated, and brief. They do not offer easy planations of complex issues. They presuppose a level technical understanding that most practitioners may have. This is a reference book, a collection of reviews scientific data concerning human psychopharmacology necessarily clinically oriented. For the student and pro sional who wants an overview of the current state of kno edge, the content of this book is well worth the effort of re ing it.

Chapter one, "Drug Self Administration: An Analysis the Reinforcing Effects of Drugs," summarizes the major imal research on drug self-administration. It is a clear straightforward exposition of the philosophy, techniqu and results of such animal research. Unfortunately no tempt is made to present, even speculatively, the impli tions of this research for human behavior, nor are any ref ences made to comparable research in humans.

Chapters two and three, "Analgesics and Their Anta nists: Structure-Activity Relationships" and "Opiates: H man Psychopharmacology," complement each other. T well written discussion of structure-activity relationsh

esents an extraordinary amount of information and is a od guide for anyone who wants to learn more about this pic. Social historical data are interwoven with the latest ormation concerning structure-activity relationships. The thor of this chapter, Jacobson, acknowledges that "unfor- nately, we are without a viable theory that can quan- atively relate molecular structure to biological activity for r multitude of analgesics." Mansky's overview of opiates ings together an impressive amount of information in a adable form. There are many good clinical observations oughout the chapter, and anyone working with narcotic dicts can benefit from this review.

There is no companion chapter on human psycho- armacology for chapter four, "Sedative-Hypnotics: Ani- al Pharmacology," and this is a weakness of this volume. apter five, "Alcohol and Human Behavior" is one of the tter overviews of this topic. It is scholarly, well-written, d packed with information. The authors, Mello and Men- lson, state that "the effects of alcohol on human behavior e diverse and complex and rarely in accord with traditional liefs. . . . Alcohol abuse often results in a distortion of ny of the simplest alcohol-related pleasures. The once sitive effects of alcohol are often transformed into their tithesis."

Finally, chapters six and seven deal with "Cannabis: ructure-Activity Relationships" and "Marihuana: Human fects." There may be a little sophistry involved in calling e drug cannabis when one is discussing structure-activity lationships and marijuana for human effects, but these apters present good overviews of the subject and are valu- le for anyone interested in the subject matter. A system of oss-referencing to other chapters within the *Handbook*, nilar to that done in the *Comprehensive Textbook of Psy- iatry* (1), would have enhanced the value of this series as a hole.

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ie Psychology of the Self: A Casebook, *written with the col- boration of Heinz Kohut, M.D.; edited by Arnold Gold- rg, M.D. New York, N.Y., International Universities ess, 1978, 456 pp., $20.00.*

This book by a group of bold and sensitive clinicians is a llection of six cases. The analysts expose their discomfort, rors, uncertainties, and hesitations as they struggled to ap- y Kohut's formulations to their analytic work with narcis- stic patients. They take Kohut's metapsychological termi- logy and give it context and depth with extensive clinical ta. Their work gives a clear picture of Kohut's under- anding of the dynamics and treatment of narcissism; it also ings into focus many of the questions his formulations ise.

A key aspect of Kohut's theory is the concept of a sepa- te line of development for narcissism with its own stage- lated tasks and human environmental needs (1, 2). Kohut arifies the child's normal exhibitionism and grandiosity and s need for his parents to accept these qualities as they mir- ror his realistic attributes and assets. He suggests that parental "mirroring" supports the child's capacity to develop a coherent sense of self. Phase-appropriate tolerable frustration of mirroring needs allows the child's grandiosity and exhibitionism to be transformed into mature narcissism, ambition, and internally regulated self-esteem. A second crucial aspect of the child's "bipolar self" is his need to lean on idealized parents who can initially accept his idealization. Gradual phase-appropriate disenchantment with idealized parents contributes to the child's development of internalized ideals.

Failure in this careful choreography contributes to defects in the self, which are manifest by unmodulated grandiosity or exhibitionism, a tendency to fragmentation or overstimulation, empty depressions, narcissistic relationships with others, and continuing dependency on others to support fragile self-esteem.

Through a study of the transferences in these six cases, the analysts present a picture of the nature of parental failure in supporting the child's narcissistic development. The parents are seen as preoccupied with themselves, unaccepting or unavailable, devaluing of the child or of themselves, intrusive, exhibitionistic, and unempathic. As the analysts attempted to understand their patients, they saw how classical analytic interventions themselves became the sources in the transference for the repetitive trauma the patients feared the most. Oedipal interpretations were heard by these patients as echoes of their parents' attempts to lay their own meanings on the child. Often, the patient's angry and desperate response to these interpretations took the form of, "All you do is interpret, you won't take me seriously. You don't believe me." The patients' extraordinary sensitivity, vulnerability, and susceptibility to humiliation and fragmentation required a shift in the analysts' interpretive focus.

With Kohut's new perspective, these analysts were able to interpret their patients' responses as repetitions of early experiences of nonacceptance and intrusion. The analysts' acceptance and interpretation of their patients' need for mirroring responses and of their need for an idealization of the analyst evoked confirming early memories and a developing acceptance and perspective in the patients of their unintegrated narcissistic needs. In many of the cases the clinical material demonstrates how the analysts' empathic failures were used by the patients to recognize their earlier trauma and work it through in a new context so that they could internalize a more stable and coherent sense of themselves. This process resulted in significant improvement in the patients' creative capacities, ambitions, and sense of independence.

Despite the richness and openness of the case material, important questions about these clinicians' conceptualizations of the analytic experience remain. In many of the cases, interpretation alone appeared to be sufficient. However, in others direct gratification (e.g., admiration, loaning money) was used by the analysts without adequate clarification of how the intervention was understood. It is important to know how an analyst reaches the determination that a patient cannot stand the deprivation of an interpretation without being disruptively humiliated. In addition, the authors' focus on narcissistic issues leaves the reader with a somewhat monochromatic view of these patients. Many alternative formulations are raised only to be too rapidly dismissed; one is left wondering about alternative or additional perspectives from which to view the material.

Finally, it remains unclear how a mutually agreed on termination can be reached when the patient does not relate to the analyst as a whole object. For several of these cases,

although many narcissistic issues had been worked through, much seemed to remain untouched at termination. It would be helpful to know how these analysts determined when to relinquish their attempts to move beyond the narcissistic transference with the patient.

This is an evocative book. If many issues remain unclear it is because the concepts are still in their formative stages. The contributors of these case reports invite us to struggle with these issues along with them. They have given us the basic data to help us examine their thinking.

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The Therapeutic Interaction: A Synthesis, *by Robert Langs, M.D. New York, N.Y., Jason Aronson, 1977, 195 pp., $50.00.*

Robert Langs belongs to a group of psychoanalysts who have stressed what I would characterize as the hyperalert, hypertechnical, almost suspicious approach to the psychoanalytic process. Since the early 1970s Langs' productivity has been staggering, including five lengthy personally authored volumes as well as an impressive list of edited books on clinical psychoanalysis and its application. However, it is difficult to assess the impact of these works on the profession at large, particularly because they tend to fall between various groups of clinicians. Langs' obsessional concern with maintaining the psychoanalytic framework and detailing the interaction between psychoanalyst and patient is certainly beyond the concern of most psychiatric psychotherapists. Classically trained psychoanalysts tend to turn to more established texts on the issues of psychoanalytic technique.

In my view, Langs' approach to psychoanalysis and psychoanalytic psychotherapy is far from comfortable or satisfactory. If critics of psychoanalysis have tended to be overly harsh in their criticism or dismissive in their judgments of psychoanalysis, psychoanalysts like Langs have erred in the direction of overvaluing the psychoanalyst's power by attributing to psychoanalysis a monopoly on curative factors for emotionally based illnesses while insisting on flawless technique as essential. Langs represents the extreme end of this spectrum of psychoanalysts who attribute excessive importance to technique.

Even more significant than Langs' willingness to dismiss other approaches to psychotherapy and psychoanalysis is the effect of his "purist" approach to psychoanalytic technique. At the end of this brief volume he states, "Looking back over this synthesis, I am reminded again of the virtual impossibility of the analyst to maintain constant mastery over the incredibly complex relationship between himself and the analysand." This attempt to maintain complete and constant mastery or, more precisely, control over all aspects of the analytic relationship is what seems to me to be a major limitation of the author's approach. In his hands psychoanalysis becomes an endeavor of such technical difficulty and virtuosity as to defy practice by reasonably intelligent, well-educated psychoanalysts. Pitfalls are everywhere. The analyst is continually in danger of being misled or seduced by the patient, and the patient must beware of the analyst's countertransference. The analyst must scrutinize every aspect of his or her interaction with the patient to what appears to me to be a crippling extent. This constant stress of the need for purity and vigilance gives much of this volume the feel of a moral tract.

In his closing paragraphs Langs anticipates criticism from other analysts because of his tendency to stress interaction aspects of the analytic process. However, he does not seem aware of the possibility that criticism of his ideas might come from another source, namely, the degree of technical rigidity he advocates. Langs has also felt it important to characterize the interactional aspect of the psychoanalytic process in terms of projective identification and introjective identification. He borrows these terms freely from the Kleinian school of psychoanalysis while maintaining that his is a classical stance on the technique of psychoanalysis. These concepts, which seem to me to be questionable at best, are then used further in the service of technical rigidification and suspiciousness of the patient and the analyst.

I think there is considerable irony in the fact that although Langs writes extensively about the therapeutic alliance, his adherence to the concept of projective identification and introjective identification is a major barrier to anything approaching basic trust in the psychotherapeutic relationship. This then seems to me to be a book greatly marred by psychoanalytic chauvinism. It is unlikely to win friends for psychoanalysis or to help individuals who, while not practicing psychoanalysis proper, use findings from psychoanalysis and modified psychoanalytic techniques in conducting psychotherapy. Those searching for a nonchauvinistic, helpful clarification of how to be both psychoanalytically oriented and human will not find it in this volume. I think, in fact, that it would have a negative effect on an individual's clinical flexibility.

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C.G. Jung: The Haunted Prophet, *by Paul J. Stern. New York, N.Y., Delta (Dell Publishing Co.), 1977, 262 pp., $3. (paper).*

Thanks to three-quarters of a century of research into the depths of the psyche, we are very familiar with the phenomenon of transference, the putting onto another person of our own psychic attitudes and contents of which we are unaware. Chief among such projections are those which originate in our own earliest past that have long been forgotten and repressed into the unconscious—feelings about parents, siblings, and other significant members of our early environment. We know well that our transference of past unresolved emotions into significant present-day relationships can be strongly tinged with negative or positive flavor. No one is immune to this natural process of projection. The only way this autonomous process can be altered is through the intervention of conscious understanding and the assimilation of the unconscious contents in transference.

Analysts themselves fall victim to transference, frequently in a positive way to their psychological mentor or negatively against a founder of an opposing school of depth psychology. The hallmarks of unresolved transference clearly announce that projections are taking place. We feel especially agitated about the person onto whom we are projecting—that person has gotten under our skin and we strongly overreact. We feel

mpulsively gripped by our reactions and neither measure
modify the tone of adoration, if the transference is posi-
, or of captious, even vitriolic attack if the transference is
ative. We are caught in unconscious identification with
person onto whom the transference has been made. We
not extricate ourselves and give the impression of thrash-
about in an irrational manner to get free.

aul Stern's book on Jung bears all the marks of negative
sference. His pages abound in sarcastic remarks and be-
ing commentary. His authoritative tone and frequent use
what look like precise details, in some cases with quota-
s, are all the more remarkable because the book has no
tnote data whatsoever. The reader is left wondering how
ch of what Stern asserts as fact stems from his endow-
nt of his subject with his own negative reactions.

he reader may be even more puzzled about the source of
author's disproportionately intense reactions to Jung and
y he even bothered to write this screed. The book is not
ful as an introduction to Jung's thought or as a critical
luation of the analytical procedures with which it is asso-
ed. There may be some value in the work for those
ght in too passionately positive a transference to Jung
his ideas. For them, Stern's too passionately negative
ctions might suggest a better balance.

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**ld Abuser: A Study of Child Abusers in Self-Help Group
rapy,** *by Marilyn C. Collins, Ph.D. Littleton, Mass., 3 Publishing Co., 1978, 124 pp., $12.50.*

ommiseration can offer comfort to group participants. A
pite from suffering and a source of strength, comfort de-
s from joining a body greater than the individual self. The
ole becomes more than the sum of its parts. This principle
been institutionalized in various self-help movements
r the past several decades. Perhaps the best known is
oholics Anonymous. We can assume that Parents Anon-
ous borrowed their name and, perhaps, their ideology
n Alcoholics Anonymous.

his book offers a description of the Parents Anonymous
vement as reported by a social scientist who served as a
nsor for a group of mothers who had abused their chil-
n. The author draws on her experience as a participant-
erver to assess the causes and suggest a solution for the
blem of child abuse. Her technique of quoting interviews
1 abusing mothers lends a refreshing vitality to data that
ht otherwise be buried by the obscure phraseology that
vails when the text turns from clinical material to a dis-
sion of ideas.

he author characterizes the therapeutic consequences of
icipation in Parents Anonymous as following six stages
he "moral career of a child abuser." Starting from stage
, "being different and feeling guilty," the participants
n alternative behavior patterns to physically abusing
r children. The telephone is heavily relied on as a means
ontacting group members whenever the impulse to abuse
ild arises. This technique, used extensively in Alcoholics
onymous, appears to work well for members of Parents
onymous as well.

he author states that the modification of abusive behav-
depends on "coming out" as a child abuser. A public
laration among a group of peers constitutes an essential
fessional technique as a step toward attaining stage six,
oral self-acceptance and becoming a recruiter."

The ultimate therapeutic goal of the Parents Anonymous program is the attainment of a "career of affiliation." This is defined by the author as being "an experienced child abuser, skilled as a lay therapist." The author faults the efforts of other workers for having failed to offer this form of solution:

> Few "rehabilitative" programs for child abusers at any level, whether part of the private or public sector, offer the opportunity to their participants to graduate from the stigma that defined their need of rehabilitation to begin with. The major flaw in these programs is their lack of an affiliation clause. (p. 108)

In her approach to this complex problem, the author relies on the writings of Becker, Goffman, and other social scientists, several of whom are not listed in the bibliography although their work is cited in the text. The psychiatrist reader of this book will find scant reference to individual dynamics, although "lowered self-esteem" is cited as a cause of abusive behavior. Although the author emphasizes that this problem is not limited to the lower socioeconomic classes, she makes no effort to search for an explanation that might transcend the limitations of social theory.

The book raises several points about which there could be extended discussion. The substitution of one activity for another is a well-recognized technique for modifying behavior. It has been used to alter alcoholism, drug addiction, phobias, and sexual inhibitions. Substituting disulfiram, methadone, inspirational phrases, and erotic imagery can be employed to counter temporarily the threat posed by unmastered appetites. However, substitution of actions, no matter how effective an adjunct in changing the social dimension of behavior, should not be equated with a transformation of motivation.

The goal of a "career of affiliation" presents another questionable item. The status of *ex,* whether drunk, con, or child abuser, is not in itself a qualification to treat others afflicted with the condition. Child abuse is a discrete clinical syndrome manifesting a pervasive, deep, and pressing problem, the mastery of aggression. The tendency to formulate simple solutions for this complex issue, although understandable, is fraught with hazards. Simple solutions suggest that the problem is simple. It is not. Child abuse draws on issues of human pathology from the deepest levels of individual development and social adaptation. An understanding of the phenomenon is not likely to be granted those who choose to pursue a simplistic approach.

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Society, Stress, and Disease, Vol. 3: The Productive and Reproductive Age—Male/Female Roles and Relationships, *edited by Lennart Levi, M.D. New York, N.Y., Oxford University Press, 1978, 279 pp., $41.00.*

This volume is one of an increasing wave of books growing from the conviction that if one convenes a symposium of well-known names in a given field the presentations should be published. This volume is a convincing counterargument.

Within its modest page length it contains 37 chapters by nearly as many different authors as well as a synopsis of the general discussion. There are a few exceptional chapters. Two by Henry and associates correlate the breakdown of normal male/female role differentiation in experimental mu-

rine populations with total disruption of reproductive patterns, maternal care, offspring survival, and social control of aggression ensuring colony survival. They further document the correlation of such stress with physiological disturbance and physical pathology and discuss the data suggesting that these findings may extrapolate to humans. Liljestrom offers a sensible discussion of the different stresses generated in role-differentiated versus role-shared young nuclear families, indicating that the trade-off of stresses for both parents and children does not strongly favor either pattern. Gebhard presents previously unpublished data from the Kinsey research on subjectively perceived sources of marital stress.

Unfortunately, these presentations represent exceptions. Most of the chapters are so brief that they permit generalizations and truisms more appropriate to high school texts or provide sociological documentation of the obvious. With few exceptions the format does not encourage the presentation of new or detailed data or carefully reasoned and documented broad conclusions. Mead's three pages of generalizations about cross-cultural determinants of male/female roles, followed by five pages of tiny print references that are not noted in the text is an unbecoming exercise in condescension.

The book does have a predominant theme—the plea for psychosocial equality for women—but here too there are more sorrows than joys in the presentation despite the philosophical acceptability of that goal. The advocacy of sociopolitical goals in a scientific forum can lead to carelessness in the supports one calls on. Blatantly undocumented statements such as, "Most female neuroses are a result of societal demands and discrimination rather than the supposed mental illness of the individual," are adduced as scientific support. There is often a fuzzy indistinction between "equal" and "identical." State-supported social engineering to produce androgeny in young children is presented as an unquestioned good, with the implication that it is scientifically supported but without attention even to the cautionary data cited in this volume.

It is difficult to identify an audience for whom this book could be recommended. It is too uneven and confusing to benefit the unsophisticated student and offers too little new data or sound and careful scholarship for the knowledgeable professional in the field of male/female roles and traits.

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Notizen zu Martin Heidegger, *by Karl Jaspers; edited by Hans Saner. Munich, Germany, R. Piper & Co., 1978, 342 pp., 29.30 deutsch marks.*

Karl Jaspers wrote notes on Martin Heidegger for more than 30 years, from 1928 to 1964. These were written with the intention of producing a comprehensive critique of Heidegger, but Jaspers, who died in 1969, did not complete the work, nor did he edit these notes; these have been done by one of his assistants.

Jaspers and Heidegger first met in 1920. Both hoped that a friendship might develop because they felt united in the desire to combat the traditional formalistic philosophy taught in German universities at that time. In the case of Jaspers, this was related to the fact that he had no formal training in philosophy and had entered this field through the "back door" of medicine, psychiatry, and psychology. His *General Psychopathology* had appeared in 1913.

A hesitant and somewhat ambivalent friendship seer have existed in fact until 1933. During this time their philosophical works were published: Heidegger's *Sein Zeit* (1927) and Jaspers' three-volume *Philosophie* (1 The relationship was interrupted by the effects of poli events, which were quite different for the two. Heide pronounced himself publicly to be a convinced nationa cialist (Jaspers says Heidegger was no opportunist) and came the first national socialist rector of his university (F burg). After a year, apparently, Heidegger was discarde the party (Jaspers states Heidegger was entirely naïve pc cally as well as in all aspects of practical life such as finar and in science and medicine).

Jaspers, on the other hand, although he initially had clear ideas about the new government, quickly got into flict with it. From 1937 on he was not allowed to teacl publish; during the war both he and his Jewish wife wer constant danger of being arrested. After 1945, Jaspers fered to reconcile with Heidegger if he would make a pu statement acknowledging his earlier error; this was not fo coming, and Heidegger later stated publicly that it ne would. This resulted in the abandoning of further attempt reconciliation between the two philosophers.

The notes largely represent Jaspers' attempt to underst the reasons for this failure. He found them in both Heic ger's philosophy and his personality. We learn in the edit introduction that, as might be expected, the reasons ar part to be sought on Jaspers' side, who was quite sensitiv criticisms by his colleague. Jaspers continued to be ambi lent about Heidegger, but his negative opinions tendec become stronger as the years passed by. The principal ferences between them, as he saw it, were in the degre personal involvement in philosophy. He saw Heidegge attempting to construct an objective philosophical edific if it were a branch of science and independent of the one had constructed it. Jaspers considered this a fatal error f philosopher: "This is akin to founding a religion." viewed Heidegger as a sort of magician with a very powe and beautiful writing style that could become a domir force to the detriment of the subject matter. Heidegger quired a much larger following than Jaspers, and Jas thought that he had strong authoritarian inclinations. Her of course the suspected link with the political involvem

The fact remains that Jaspers considered Heidegger only person of comparable rank in his field. At one poin even considered that Heidegger may have had more im tant things to say: "But this only one was my polite ene The powers we served were incompatible. Soon it appe that we could not even talk to each other." Still, he no when someone attacked Heidegger, he found himself fending him, "not because I agree with him but because type of critique does not touch the points which seem to to be the philosophically essential ones."

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Coding the Therapeutic Process: Emblems of Encounter Manual for Counsellors and Therapists, *by Murray Cox. N York, N.Y., Pergamon Press, 1978, 77 pp., $7.50 (paper*

Structuring the Therapeutic Process: Compromise Chaos—the Therapist's Response to the Individual and Group, *by Murray Cox. New York, N.Y., Pergamon Pr 1978, 292 pp., $10.00 (paper).*

Dr. Cox has written two outstanding books for psychotherapists in all disciplines. He writes as an individual and group therapist in private practice and as a psychotherapist at Broadmoor, a hospital for those who "require treatment under conditions of special security on account of their dangerous, violent, or criminal propensity." Thus, what Dr. Cox says in these books is derived from an unusually diverse experience.

Structuring the Therapeutic Process is a literate, wise, and witty book. It is also eclectic. Dr. Cox has gathered a rich harvest from many sources. He is less concerned about schools of thought than he is about what he feels is the "core dynamic of psychotherapy," i.e., emotional disclosure. Facilitation of disclosure occurs, no matter what therapeutic orientation is used, when there is congruence of perspective, when the therapist can view the world from within the patient. The patient becomes more trusting as the therapist comes closer to risking, in Dr. Cox's words, "his own ontological security."

There is authenticity and honesty in Dr. Cox's message. Therapists who are already aware of the fact that they reach therapeutic turning points when they risk their own vulnerability will be supported by the framework he provides. Other therapists will be encouraged to try to use their own emotional responsivity for intervention.

The position presented does not support casual statements by the therapist. It is the patient who associates, not the therapist. Dr. Cox presents a structure for filtering out what is said through a therapeutic vigilance that comes from receptivity to the patient's needs, talents, and, occasionally, desperation. The therapist primarily wants to help the patient; what he or she learns about himself or herself stands apart from and is always secondary to that goal.

The author does not convey a sense of working against tide and wind; there is a unique pleasure in such encounters. He writes, for example, of the necessity to have patients express those moments which give pleasure as much as those which give pain. He points to the possibility that some therapists are threatened by a patient's joy and thus may tend to never elicit disclosures about it. He addresses in depth the problems of transference and countertransference as he describes what is for him the essential characteristic of the therapist—emotional accessibility and tolerance for any and all of the patient's disclosures. There is the difficult and paradoxical task of mastering affective neutrality while maintaining accessibility to such feelings.

Coding the Therapeutic Process, the companion volume, is focused on a visual display system (VDS), a device for notation applicable to encounters with patients. A VDS can be a helpful guideline and marker when a therapist attempts to grasp and retain the dynamic content of patient interactions over time. This book also has many of Dr. Cox's insights into the process of psychotherapy. Perhaps it would be improved by an extended version presenting further examples. The notation used has great promise as an extremely helpful conceptual tool for the group therapist in clinical work and research. Therapists who have been concerned with problems of notation will benefit from the ideas contained in this volume.

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ɔoks Received

's listing acknowledges the receipt of recent books. Books particular interest to the readers of this journal will be iewed as space permits, and copies of the reviews will be t to the publishers. Books cannot be returned to the blishers.

vances in Neurochemistry, Vol. 3, *edited by B.W. Agranoff d M.H. Aprison. New York, N.Y., Plenum Press, 1978, pp., $32.50.*

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od Disorders: The World's Major Public Health Problem, *ted by Frank J. Ayd, Jr., M.D., and Irving J. Taylor, D. Baltimore, Md., Ayd Medical Publications, 1978, 261 , $18.50 (paper).*

e Psychiatric Foundations of Medicine, Vol. 1: Dimensions Behavior, *edited by George U. Balis, M.D., Leon Wurm- , M.D., Ellen McDaniel, M.D., and Robert G. Grenell, .D. Boston, Mass., Butterworth Publishers, 1978, 530 pp., .95.*

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e Psychiatric Foundations of Medicine, Vol. 4: Clinical Psy- pathology, *edited by George U. Balis, M.D., Leon Wurm- , M.D., Ellen McDaniel, M.D., and Robert G. Grenell, .D. Boston, Mass., Butterworth Publishers, 1978, 618 pp., .95.*

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Developmental Processes: Heinz Werner's Selected Writings. Vol. 1: General Theory and Perceptual Experience; Vol. 2: Cognition, Language, and Symbolization, *edited by Sybil S. Barten and Margery B. Franklin. New York; N.Y., International Universities Press, 1978, 549 pp., $35.00 for both volumes.*

The Neurophysiological Aspects of Human Mental Activity, *by N.P. Bechtereva. New York, N.Y., Oxford University Press, 1978, 181 pp., $11.95.*

Beyond the Double Bind: Communication and Family Systems, Theories, and Techniques with Schizophrenics, *edited by Milton M. Berger, M.D. New York, N.Y., Brunner/Mazel, 1978, 254 pp., $16.95.*

The Painted Message, *by Otto Billig and B.G. Burton-Bradley. Cambridge, Mass., Schenkman Publishing Co. (New York, N.Y., Halsted Press, John Wiley & Sons, distributor), 1978, 255 pp., $19.50.*

"So the Witch Won't Eat Me": Fantasy and the Child's Fear of Infanticide, *by Dorothy Bloch. Boston, Mass., Houghton Mifflin Co., 1978, 242 pp., $9.95.*

Brain Systems and Psychological Concepts, *by John Boddy. New York, N.Y., John Wiley & Sons, 1978, 449 pp., $32.00.*

Social Origins of Depression: A Study of Psychiatric Disorder in Women, *by George W. Brown and Tirril Harris. New York, N.Y., Free Press (Macmillan Publishing Co.), 1978, 389 pp., $19.95.*

Event-Related Brain Potentials in Man, *edited by Enoch Callaway, Patricia Tueting, and Stephen H. Koslow. New York, N.Y., Academic Press (Harcourt Brace Jovanovich), 1978, 626 pp., $22.00.*

Child Abuse and Neglect in Residential Institutions: Selected Readings on Prevention, Investigation, and Correction. *Washington, D.C., U.S. Department of Health, Education, and Welfare, 1978, unnumbered pages, no price listed (paper).*

Principles of Psychopharmacology, 2nd ed., *edited by William G. Clark, Ph.D., and Joseph del Giudice, M.D. New York, N.Y., Academic Press (Harcourt Brace Jovanovich), 1978, 957 pp., $27.50.*

Coding the Therapeutic Process: Emblems of Encounter. A Manual for Counsellors and Therapists, *by Murray Cox. New York, N.Y., Pergamon Press, 1978, 77 pp., $7.50 (paper).*

Structuring the Therapeutic Process: Compromise with Chaos—The Therapist's Response to the Individual and the Group, *by Murray Cox. New York, N.Y., Pergamon Press, 1978, 292 pp., $10.00 (paper).*

Jungian Psychotherapy: A Study in Analytical Psychology, *by Michael Fordham. New York, N.Y., John Wiley & Sons, 1978, 175 pp., no price listed.*

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Psychiatric, Psychogenic, and Somatopsychic Disorders Handbook: A Laboratory and Clinical Guide to the Medical Management of Emotional and Intellectual Pathology, *by A. James Giannini, M.D., and Henry R. Black, M.D., with Roger L. Goettsche, M.D. Garden City, N.Y., Medical Examination Publishing Co. (Excerpta Medica), 1978, 316 pp., $18.00 (spiral-bound).*

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The Growth of the Child: Reflections on Human Development, *by Jerome Kagan. New York, N.Y., W.W. Norton & Co., 1978, 283 pp., $12.95.*

A Social Interactional Theory of Emotions, *by Theodore D. Kemper. New York, N.Y., Wiley-Interscience (John Wiley & Sons), 1978, 434 pp., $19.95.*

Extrasensory Perception: Advances in Parapsychological Research, Vol. 2, *edited by Stanley Krippner, Mary Lou Carlson, Montague Ullman, and Robert O. Becker. New York, N.Y., Plenum Press, 1978, 295 pp., $22.50.*

Hysteria: The Elusive Neurosis. Psychological Issues Monograph 45, *by Alan Krohn. New York, N.Y., International Universities Press, 1978, 337 pp., $20.00.*

Child Study Through Observation and Participation: A Workbook, *by Kay Kuzma, Ed.D., with Paul Roesel, Ed.S. Palo Alto, Calif., R & E Research Associates, 1978, 207 pp., $8.00 (paper).*

Kinship and Casework: Family Networks and Social Intervention, enlarged ed., *by Hope Jensen Leichter and William E. Mitchell. New York, N.Y., Teachers College Press, 1978, 347 pp., no price listed (paper).*

The Art of Medicating Psychiatric Patients, *by John R. Lion, M.D. Baltimore, Md., Williams & Wilkins Co., 1978, 146 pp., $13.95 (paper).*

Young, Sober and Free, *written and compiled by Shelly M. shall. Center City, Minn., Hazelden Books, 1978, 137 p, $4.95 (paper).*

Hazards of Medication: A Manual on Drug Interactions, Cc traindications, and Adverse Reactions with Other Prescribi and Drug Information, 2nd ed., *edited by Eric W. Marti Ph.D. Philadelphia, Pa., J.B. Lippincott Co., 1978, 666 p $39.50.*

Laugh After Laugh: The Healing Power of Humor, *by Re mond A. Moody, Jr., M.D. Jacksonville, Fla., Headwat Press (Philadelphia, Pa., J.B. Lippincott Co., distributo 1978, 128 pp., $7.95.*

Progress in Clinical and Biological Research, Vol. 24A: Tr Research, Part A: Psychology and Methodology, *edited Walter E. Nance, Gordon Allen, and Paolo Parisi. N York, N.Y., Alan R. Liss, 1978, 259 pp., $28.00.*

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Progress in Clinical and Biological Research, Vol. 24C: Tr Research, Part C: Clinical Studies, *edited by Walter Nance, Gordon Allen, and Paolo Parisi. New York, N. Alan R. Liss, 1978, 275 pp., $30.00.*

Living and Loving After Divorce, *by Catherine Napolita and Victoria Pellegrino. New York, N.Y., Signet (N American Library), 1977, 241 pp., $1.95 (paper).*

Creating Community Acceptance for Handicapped People, *Roberta Nelson. Springfield, Ill., Charles C Thomas, 19 209 pp., $14.75.*

The Woman Patient: Medical and Psychological Interfac Vol. 1: Sexual and Reproductive Aspects of Women's Hea Care, *edited by Malkah T. Notman, M.D., and Carol C. N delson, M.D. New York, N.Y., Plenum Press, 1978, 358 p $19.50.*

The Religious Care of the Psychiatric Patient, *by Wayne Oates. Philadelphia, Pa., Westminster Press, 1978, 237 p $12.50.*

Historical Explorations in Medicine and Psychiatry, *edited Hertha Riese. New York, N.Y., Springer Publishing C 1978, 227 pp., $22.95.*

The Making and Breaking of a Nicotinic, Alcoholic Depress (A Completely True Story), *by Phil Rivers. New York, N. Vantage Press, 1978, 129 pp., $6.95.*

Sound Minds in a Soundless World, *by Luther D. Robins M.D., Sc.D. Washington, D.C., U.S. Government Print Office, 1978, 140 pp., $3.95 (paper).*

Pediatric Neurology and Neurosurgery, *edited by Richard Thompson, M.D., and John R. Green, M.D. New Yo N.Y., SP Medical & Scientific Books (Spectrum P lications), 1978, 417 pp., $30.00.*

Aging: The Process and the People, *edited by Gene Usdin, M.D., and Charles K. Hofling, M.D. New York, N.Y., Brunner/Mazel, 1978, 239 pp., $15.00.*

Existential-Phenomenological Alternatives for Psychology, *by Ronald S. Valle and Mark King. New York, N.Y., Oxford University Press, 1978, 376 pp., $17.95; $9.95 (paper).*

Neuropsychology: A Clinical Approach, *by Kevin Walsh, M.B.B.S., M.Sc. New York, N.Y., Churchill Livingstone, 1978, 363 pp., $25.00; $15.95 (paper).*

Psychiatry for Lawyers, revised ed., *by Andrew S. Watson, M.D. New York, N.Y., International Universities Press, 1978, 451 pp., $25.00.*

Programs of the Brain: Based on the Gifford Lectures, 1975–1977, *by J.Z. Young. New York, N.Y., Oxford University Press, 1978, 300 pp., $14.95.*

The Doctor Tree: Developmental Stages in the Growth of Physicians, *by Ralph N. Zabarenko and Lucy M. Zabarenko. Pittsburgh, Pa., University of Pittsburgh Press, 1978, 173 pp., $9.95.*

OFFICIAL ACTIONS

Position Statement on Abortion

This statement was approved by the Assembly of District Branches at its October 15, 1978, meeting and by the Board of Trustees at its December 10, 1977, meeting. This final draft was drawn up by a subcommittee[1] appointed by the Reference Committee to collate an Area I Action Paper and information provided by the Committee on Women, the Council on National Affairs, the Council on Children, Adolescents, and Their Families, and the American Academy of Child Psychiatry.

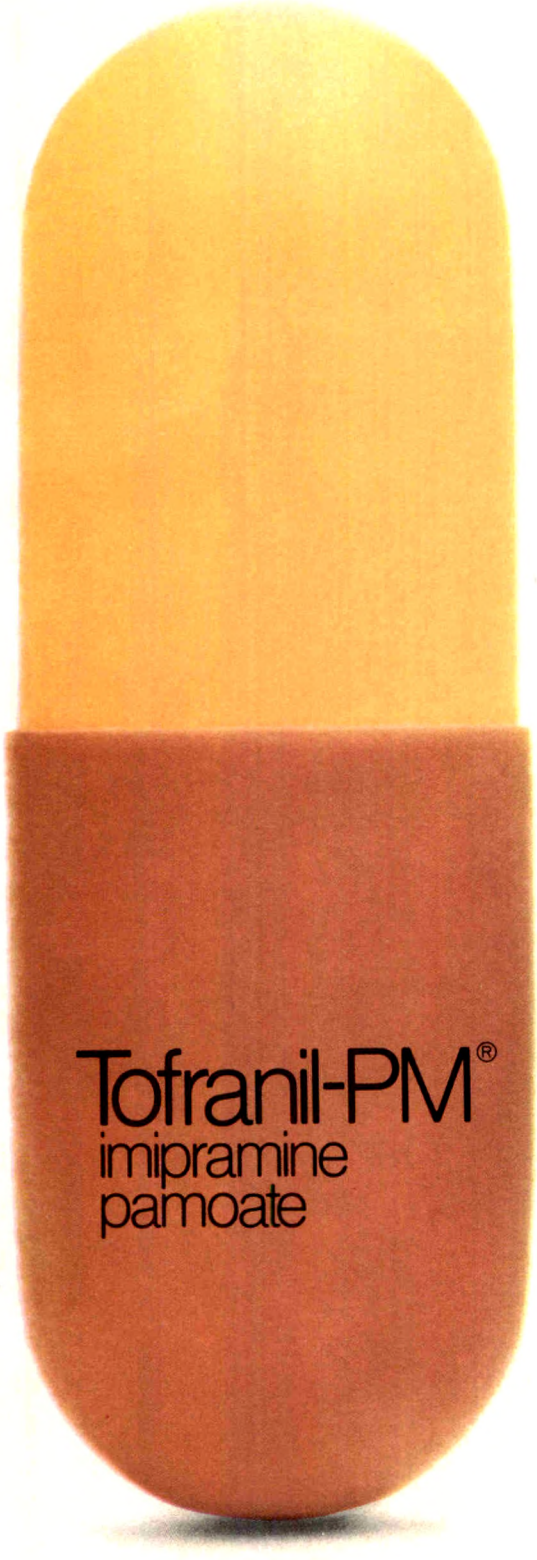
THE EMOTIONAL CONSEQUENCES of unwanted pregnancy on parents and their offspring may lead to long-standing life distress and disability, and the children of unwanted pregnancies are at high risk for abuse, neglect, mental illness, and deprivation of the quality of life. Pregnancy that results from undue coercion, rape, or incest creates even greater potential distress or disability in the child and the parents. The adolescent most vulnerable to early pregnancy is the product of adverse sociocultural conditions involving poverty, discrimination, and family disorganization, and statistics indicate that the resulting pregnancy is laden with medical complications which threaten the well-being of mother and fetus. The delivery that ensues from teenage pregnancy prone to prematurity and major threats to the health mother and child, and the resulting newborns have a high percentage of birth defects, developmental difficulties, and poorer life and health expectancy than the average for o society. Such children are often not released for adoptic and thus get caught in the web of foster care and welfa systems, possibly entering lifetimes of dependency and cos ly social interventions. The tendency of this pattern to pa from generation to generation is very marked and th serves to perpetuate a cycle of social and educational failur mental and physical illness, and serious delinquency.

Because of these considerations, and in the interest public welfare, the American Psychiatric Association 1) o poses all constitutional amendments, legislation, and regul tions curtailing family planning and abortion services to a segment of the population; 2) reaffirms its position that abc tion is a medical procedure in which physicians should r spect the patient's right to freedom of choice—psychiatris may be called on as consultants to the patient or physician those cases in which the patient or physician requests su consultation to expand mutual appreciation of motivati and consequences; and 3) affirms that the freedom to act interrupt pregnancy must be considered a mental health i perative with major social and mental health implications

[1]The subcommittee included Edward H. Futterman, M.D., chairperson of the Council on Children, Adolescents, and Their Families; James M. Stubblebine, M.D., chairperson of the Council on Mental Health Services; Harold M. Visotsky, M.D., chairperson of the Council on National Affairs (1975–1978); Jeanne Spurlock, M.D., staff liaison; and Jay Cutler, staff legal counsel.

ıany patients

HE RIGHT ANTIDEPRESSANT



No tricyclic antidepressant works more effectively to brighten the mood, relieve sleep disturbances, and ameliorate mild anxiety.[1,2] And none appears to be better balanced[3] in terms of the biogenic amine theory of depression, which relates depression to functional deficiencies of the catecholamines.

For Tofranil-PM is almost equally effective in inhibiting the postsynaptic uptake of both serotonin and norepinephrine, the two neurotransmitters most often implicated in depression, while other tricyclic antidepressants tested are heavily skewed towards one or the other.*

*Nortriptyline, a chemical derivative of amitriptyline, also exhibits balanced action, but does not appear as effective in treating serotonin-mediated depression as imipramine.

Please see last page for a brief summary of the prescribing information.

Geigy

for many patients
with mixed
anxiety-depression

THE RIGH

Geigy

COMBINATION...



While Tofranil-PM alone exerts a sedative effect in depression, some patients with mixed anxiety-depression require the added control of a minor tranquilizer. For such patients, you can confidently prescribe diazepam (or virtually any other antianxiety agent) adjunctively with Tofranil-PM —unsurpassed among tricyclic antidepressants for its mood-brightening activity.[1, 2]

Tofranil-PM is effective when used alone or in combination regimens. Virtually any combination of psychotropics is possible—a flexibility difficult to achieve with fixed-combination antidepressant regimens.

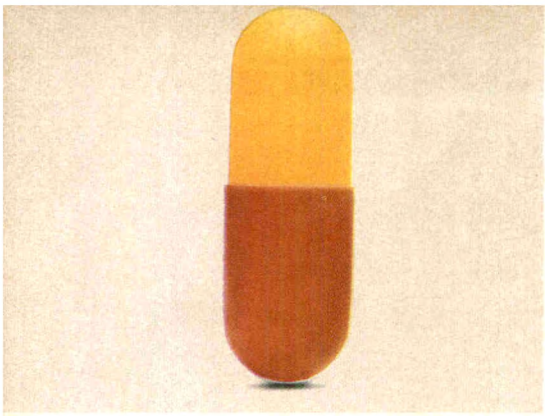
The decision of which drugs to combine, and at what dosages, remains entirely in your hands . . . where it belongs.

Please see last page for a brief summary of the prescribing information.

Tofranil-PM®...plus Valium® or any antianxiety agent

imipramine pamoate

(diazepam)



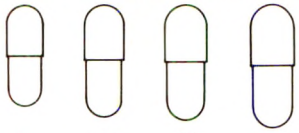
for many patients, the right antidepressant alone... or the basis of the right combination

Tofranil-PM® imipramine pamoate ...unsurpassed antidepressant action

For the majority of patients with neurotic depression, Tofranil-PM may be the only drug needed to produce a satisfactory brightening of mood.[1, 2] A recent report[4] notes that the two leading single-entity tricyclic antidepressants—imipramine pamoate (Tofranil-PM) and amitriptyline—provide equally effective antidepressant activity.

Better quality sleep—usually without morning "hangover"

Recent evaluation confirms that "... patients treated with imipramine pamoate tended ($P < .09$) to have a better quality of sleep than those treated with amitriptyline."[4]



75 mg 100 mg 125 mg 150 mg

Each capsule contains imipramine pamoate equivalent to 75, 100, 125 or 150 mg of imipramine hydrochloride.

Many patients show good results on Tofranil-PM 75 mg h.s.; however, the most effective daily dose may be the 150-mg capsule.

Tofranil-PM®

imipramine pamoate

References

1. Lippman S: Depression—A good approach for the non-psychiatrist. II—Treatment of choice. Res and Staff Phys 24 (7):100-104, 1978.
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4. Goldberg HL, Finnerty RJ: Which tricyclic for depressed outpatients, imipramine pamoate or amitriptyline? Dis Nerv Syst 38:785-9, 1977.

Tofranil-PM®

brand of imipramine pamoate

Capsules of 75 mg.
Capsules of 100 mg.
Capsules of 125 mg.
Capsules of 150 mg.
For oral administration

Each 75-mg. capsule contains imipramine pamoate equivalent to 75 mg. of imipramine hydrochloride.
Each 100-mg. capsule contains imipramine pamoate equivalent to 100 mg. of imipramine hydrochloride.
Each 125-mg. capsule contains imipramine pamoate equivalent to 125 mg. of imipramine hydrochloride.
Each 150-mg. capsule contains imipramine pamoate equivalent to 150 mg. of imipramine hydrochloride.

Brief Summary of Prescribing Information

Indications For the relief of symptoms of depression. Endogenous depression is more likely to be alleviated than other depressive states.

Contraindications The concomitant use of monoamine oxidase inhibiting compounds is contraindicated. Hyperpyretic crises or severe convulsive seizures may occur in patients receiving such combinations. The potentiation of adverse effects can be serious, or even fatal. When it is desired to substitute Tofranil-PM, brand of imipramine pamoate, in patients receiving a monoamine oxidase inhibitor, as long an interval should elapse as the clinical situation will allow, with a minimum of 14 days. Initial dosage should be low and increases should be gradual and cautiously prescribed.
The drug is contraindicated during the acute recovery period after a myocardial infarction. Patients with a known hypersensitivity to this compound should not be given the drug. The possibility of cross-sensitivity to other dibenzazepine compounds should be kept in mind.

Warnings *Usage in Pregnancy:* Safe use of imipramine during pregnancy and lactation has not been established; therefore, in administering the drug to pregnant patients, nursing mothers, or women of childbearing potential, the potential benefits must be weighed against the possible hazards. Animal reproduction studies have yielded inconclusive results. There have been clinical reports of congenital malformation associated with the use of imipramine, but a causal relationship has not been confirmed.
Extreme caution should be used when this drug is given to:

- patients with cardiovascular disease because of the possibility of conduction defects, arrhythmias, myocardial infarction, strokes and tachycardia;
- patients with increased intraocular pressure, history of urinary retention, or history of narrow-angle glaucoma because of the drug's anticholinergic properties;
- hyperthyroid patients or those on thyroid medication because of the possibility of cardiovascular toxicity;
- patients with a history of seizure disorder because this drug has been shown to lower the seizure threshold;
- patients receiving guanethidine or similar agents since Tofranil-PM, brand of imipramine pamoate, may block the pharmacologic effects of these drugs.

Since imipramine may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks, such as operating an automobile or machinery, the patient should be cautioned accordingly.
Usage in Children: Tofranil-PM, brand of imipramine pamoate, should not be used in children of any age because of the increased potential for acute overdosage due to the high unit potency (75 mg., 100 mg., 125 mg. and 150 mg.). Each capsule contains imipramine pamoate equivalent to 75 mg., 100 mg., 125 mg. or 150 mg. imipramine hydrochloride.

Precautions It should be kept in mind that the possibility of suicide in seriously depressed patients is inherent in the illness and may persist until significant remission occurs. Such patients should be carefully supervised during the early phase of treatment with Tofranil-PM, brand of imipramine pamoate, and may require hospitalization. Prescriptions should be written for the smallest amount feasible.
Hypomanic or manic episodes may occur, particularly in patients with cyclic disorders. Such reactions may necessitate discontinuation of the drug. If needed, Tofranil-PM, brand of imipramine pamoate, may be resumed in lower dosage when these ep relieved. Administration of a tranquilizer ful in controlling such episodes.
Prior to elective surgery, imipramine shoul tinued for as long as the clinical situation
An activation of the psychosis may occa observed in schizophrenic patients and reduction of dosage and the addition of phenothiazine.
In occasional susceptible patients or in t ing anticholinergic drugs (including antip agents) in addition, the atropine-like effe come more pronounced (e.g., paralytic i supervision and careful adjustment of dc quired when this drug is administered co with anticholinergic or sympathomimetic
Avoid the use of preparations, such as d and local anesthetics, which contain any pathomimetic amine (e.g., adrenalin, nor since it has been reported that tricyclic a sants can potentiate the effects of catec
Patients should be warned that the conc of alcoholic beverages may be associate exaggerated effects.
Both elevation and lowering of blood suc have been reported.
Concurrent administration of imipramine troshock therapy may increase the haza treatment should be limited to those pati whom it is essential, since there is limite experience.

Adverse Reactions Note: Although the follows includes a few adverse reactions not been reported with this specific drug macological similarities among the tricyc depressant drugs require that each of th be considered when imipramine is admi
Cardiovascular: Hypotension, hypertens dia, palpitation, myocardial infarction, ar heart block, stroke, falls.
Psychiatric: Confusional states (especia derly) with hallucinations, disorientation, anxiety, restlessness, agitation; insomnia mares; hypomania; exacerbation of psy
Neurological: Numbness, tingling, pares tremities; incoordination, ataxia, tremors neuropathy; extrapyramidal symptoms; alterations in EEG patterns; tinnitus.
Anticholinergic: Dry mouth, and, rarely, a sublingual adenitis; blurred vision, distu accommodation, mydriasis; constipation ileus; urinary retention, delayed micturiti the urinary tract.
Allergic: Skin rash, petechiae, urticaria, photosensitization (avoid excessive exp light); edema (general or of face and tor fever; cross-sensitivity with desipramine
Hematologic: Bone marrow depression agranulocytosis; eosinophilia; purpura; cytopenia. Leukocyte and differential cc be performed in any patient who develc sore throat during therapy; the drug shc continued if there is evidence of patholc phil depression.
Gastrointestinal: Nausea and vomiting, epigastric distress, diarrhea; peculiar ta abdominal cramps, black tongue.
Endocrine: Gynecomastia in the male; t largement and galactorrhea in the fema or decreased libido, impotence; testicul elevation or depression of blood sugar l
Other: Jaundice (simulating obstructive function; weight gain or loss; perspiratic urinary frequency; drowsiness, dizzines and fatigue; headache; parotid swelling
Withdrawal Symptoms: Though not indi addiction, abrupt cessation of treatmen longed therapy may produce nausea, h malaise.

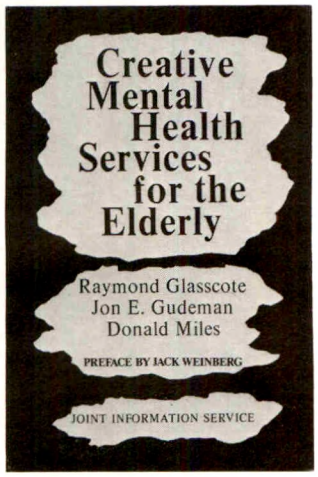
How Supplied Tofranil-PM, brand of im pamoate, is available as follows:
75-mg., coral-colored capsules equival imipramine hydrochloride in bottles of 3 1,000 capsules.
100-mg., dark yellow/coral-colored cap alent to 100 mg. imipramine hydrochlori of 30, 100 and 1,000 capsules.
125-mg., light yellow/coral-colored cap lent to 125 mg. imipramine hydrochlorid 30, 100 and 1,000 capsules.
150-mg., coral-colored capsules equiva mg. imipramine hydrochloride in bottles and 1,000 capsules.
667120 (9/75) 98-146-840-A
For complete details, including descript dosage and administration, and overdo see full prescribing information.

GEIGY Pharmaceuticals
Division of CIBA-GEIGY Corporation
Ardsley, New York 10502

Geigy

The Joint Information Service of the American Psychiatric Association and the Mental Health Association releases Two New Volumes That

Focus on the Elderly



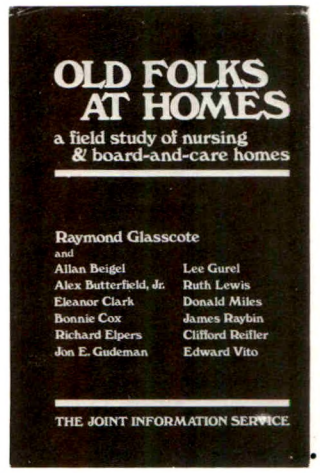
This carefully documented book reports a field study of ten programs, four in America and six in England and Scandinavia, which serve as practical demonstrations of creative mental health services for the elderly. They include:

- a novel "Neighborhood Family" providing vigorous support to elderly residents of several trailer parks
- a high school for the elderly which sends its students abroad for study trips
- a "Human Development Project" that focuses on responding to the psychological needs of the elderly
- a carefully coordinated system of "respite hospitalization," which promotes the health of the elderly while allowing maximum use of hospital beds and family resources
- a "Lucy Booth," patterned after the Peanuts comic strip, which provides easy access to many kinds of services
- and many other innovative, successful approaches.

Each program is subjected to intensive scrutiny. The resulting document becomes a handbook, and, as well, a forum through which some of the world's most experienced practitioners of "the psychiatry of old age" present their views.

Dr. Wilma Donohue, director of the International Center for Social Gerontology, characterizes this volume as "a masterful job of putting everything in relief . . . the guidelines for action are right here."

190 pages. Casebound. Price $8.50.



Concerned about the very bad reputation that nursing homes have received from many quarters in recent years, the Joint Information Service set up a field study to visit a systematically chosen sample of nursing homes, and board-and-care homes as well, to see to what extent the care of and the quality of life for the patients differed from the geriatrics units of state hospitals. Altogether, sixty nursing homes and 31 board-and-care homes in ten locales were visited, in each case by the senior author accompanied by two mental health professionals.

The teams were surpised to learn that care and quality of life in many of the nursing and board-and-care homes they visited were better—sometimes dramatically better—than that in the mental health facilities and often at lower cost.

This unique publication provides an overview of all facilities visited plus vignettes of approximately half. Federal and state rules and regulations are also expertly synthesized into brief and readable style.

148 pages. Casebound. Price $6.50

Publication Sales Division
American Psychiatric Association
1700 18th St., N.W., Washington, D.C. 20009

Please send: _____ **copies of *Creative Mental Health Services for the Elderly* @ $8.50 per copy**
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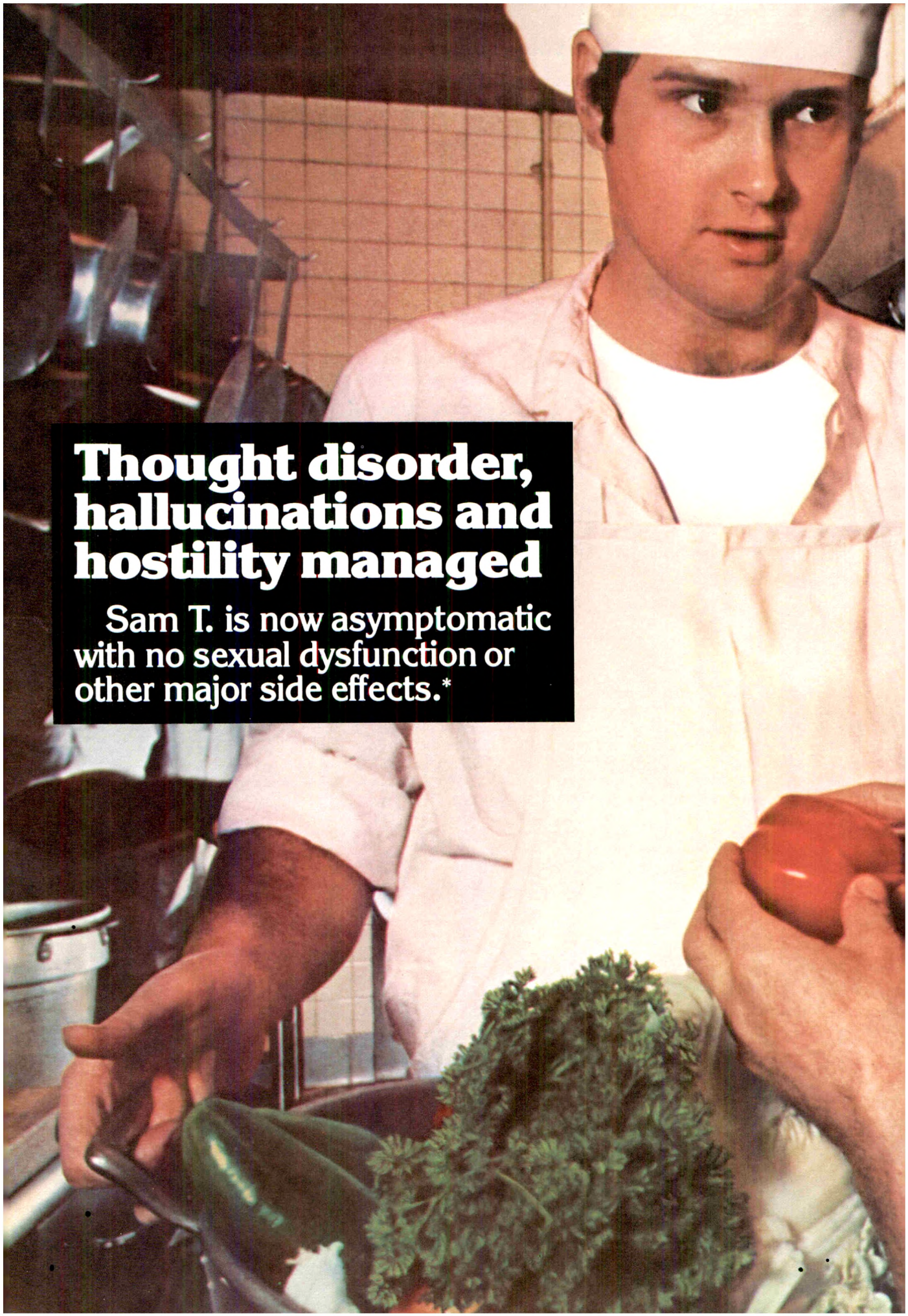
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AJP


Thought disorder, hallucinations and hostility managed
Sam T. is now asymptomatic with no sexual dysfunction or other major side effects.*



LOXITANE® Loxapine Succinate

Permits rapid management of Sam's hostility and excited behavior

Thirty-six year old salad maker, claiming to be the "Wild Man of the West" was brought to the hospital by his kitchen supervisor. History revealed prior psychiatric hospitalizations and therapies including psychotropic drugs. Hypertension was noted as a complication of earlier treatment with antipsychotic drugs. Diagnosis: chronic schizophrenia, acute exacerbation. He responded promptly to LOXITANE® C Loxapine HCl Concentrate 50 mg stat, followed by LOXITANE® Loxapine Succinate capsules 30 mg t.i.d.

Permits aggressive titration for rapid establishment of optimal management of Sam's thought disorder and hallucinations

During first week, LOXITANE dosage increased to 50 mg q.i.d. Significant improvement in thought disorder noted by third week. Auditory hallucinations and delusions much less frequent. Patient calmer and more cooperative.

Permits sustained symptom management with minimal risk of serious adverse effects, such as sexual dysfunction

Patient revealed that while on outpatient status, he failed to take his previously prescribed antipsychotic for a ten-month period after he met and married his girlfriend. He stated the drug "didn't let me be a man." No sexual dysfunction reported with LOXITANE.

Side effects are usually mild, transitory and easily managed

Patient felt anxious because his "heart was beating so fast," and he "could not sit still." Increased pulse rate and restlessness subsided following decrease of LOXITANE to 20 mg q.i.d.

Consistence in schizophrenic symptom management and tolerance promotes patient compliance

All admitting symptomatology under control by the fourth week. By the sixth week, he was anxious to return home. Stated "this medicine doesn't make me feel drowsy like the other did." Discharged by the eighth week. He returned to work fully managed on LOXITANE 20 mg t.i.d., and free from recurrence of previously reported side effects.

*Painful ejaculation and urine containing sperm have not been reported. Endocrine disturbances such as galactorrhea and menstrual irregularities have only been rarely reported. Other sexual side effects infrequently reported. Although not reported to date, possibility of hepatic, renal, ocular, or phototoxicity cannot be ruled out at this time. Transient liver enzyme changes not definitely related to LOXITANE have been reported. Not an actual patient, but a simulation to illustrate the action of LOXITANE as reported in clinical studies.

See LOXITANE prescribing information on following page for indications, warnings and precautions and for more detailed information concerning side effects.

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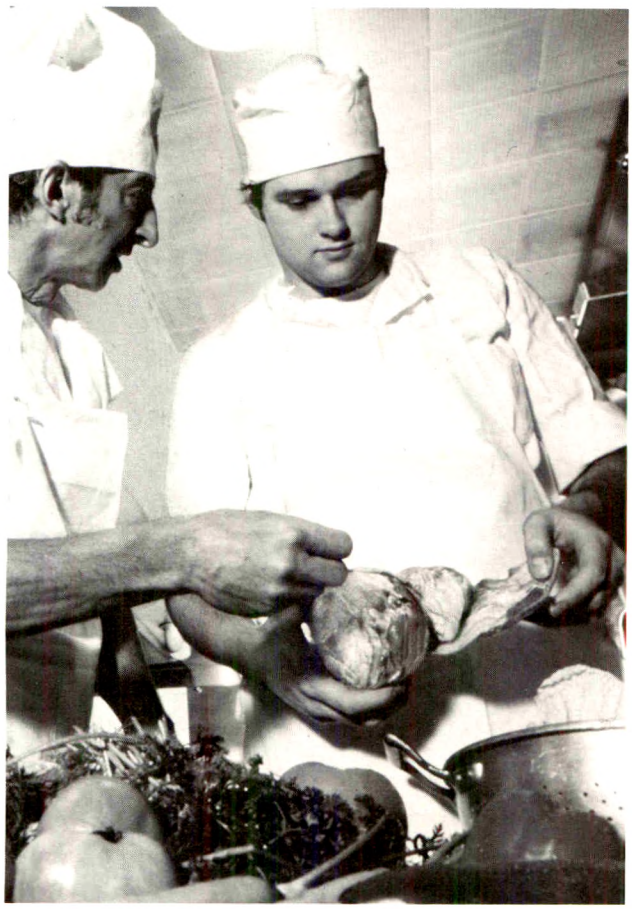
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Lederle

Capsules:
5 mg, 10 mg,
25 mg, 50 mg

Maximum management... minimum risk

Maximum management... minimum risk



Loxitane®
LOXAPINE SUCCINATE
Lederle

Recommended Daily Dosage

Initial Dosage	MILD 10 mg b.i.d.	MODERATE 10 mg t.i.d or q.i.d.	SEVERE 25 mg b.i.d.
First 7 to 10 days	Increase dosage until psychotic symptoms are controlled. Dosage should not exceed 250 mg/day. Usual dosage during titration: 50 to 150 mg/day.		
Maintenance Dosage	Adjust to lowest effective level. Usual maintenance dosage: 60 to 100 mg/day. Many patients are controlled with dosages as low as 20 to 60 mg/day.		

Brief Summary
LOXITANE® Loxapine Succinate Capsules
LOXITANE® C Loxapine Hydrochloride Oral Concentrate
INDICATIONS: Manifestations of schizophrenia.
CONTRAINDICATIONS: Comatose or severely depressed states; hypersensitivity to the drug.
WARNINGS: Safe use during pregnancy or lactation has not been established; weigh potential benefits to possible hazards. Not recommended for use in children under 16. May impair mental and/or physical abilities especially during early therapy; warn ambulatory patients about activities requiring alertness and concomitant use of alcohol or other CNS depressants. Not recommended for management of behavioral complications in mentally retarded patients.
PRECAUTIONS: Use with extreme caution in patients with a history of convulsive disorders; use with caution in patients with cardiovascular disease or in those with glaucoma or a tendency to urinary retention particularly when on concomitant anticholinergic medication. Loxapine has an antiemetic effect in animals which might occur in man masking signs of overdosage of toxic drugs and obscuring intestinal obstruction or brain tumor. Since possible ocular toxicity cannot be excluded, observe carefully for pigmentary retinopathy and lenticular pigmentation.
ADVERSE REACTIONS: CNS effects, other than extrapyramidal, infrequent. Mild drowsiness may occur at beginning of therapy or upon dosage increase, usually subsides with continued therapy. Sedation, dizziness, faintness, staggering gait, muscle twitching, weakness and confusional states have been reported. Extrapyramidal reactions often occur early in treatment manifested by Parkinson-like symptoms (tremor, rigidity, excessive salivation, masked facies, akathisia) controllable by dosage reduction or antiparkinson drugs at usual dosages. Dystonic and dyskinetic reactions, while less frequently occurring, may be more severe, requiring dosage reduction or temporary withdrawal plus appropriate counteractive drugs. Persistent Tardive Dyskinesia may appear during prolonged therapy or following discontinuance, the risk greater in the elderly, especially females, on high dosage. Symptoms, persistent and in some patients apparently irreversible, are characterized by rhythmical involuntary movement of the tongue, face, mouth and jaw sometimes accompanied by involuntary movement of extremities. Since there is no known effective treatment, discontinue all antipsychotic drugs if symptoms appear. Reinstitution of treatment, increased dosage, or switching to another agent may mask syndrome. The syndrome may not develop if medication is stopped when fine vermicular movements of the tongue first appear. Cardiovascular Effects: Tachycardia, hypotension, hypertension, lightheadedness and syncope. ECG changes, not known to be related to loxapine use, have been reported. Skin: Dermatitis, edema of face, pruritus, seborrhea. Possible photosensitivity and/or phototoxicity; skin rashes of unknown etiology seen in a few patients in hot summer months. Anticholinergic: Dry mouth, nasal congestion, constipation, blurred vision (more likely to occur with concomitant use of antiparkinson agents). Other: Nausea, vomiting, weight gain or loss, dyspnea, ptosis, hyperpyrexia, flushed facies, headache, paresthesia, polydipsia. Rarely, galactorrhea and menstrual irregularity of unknown etiology.

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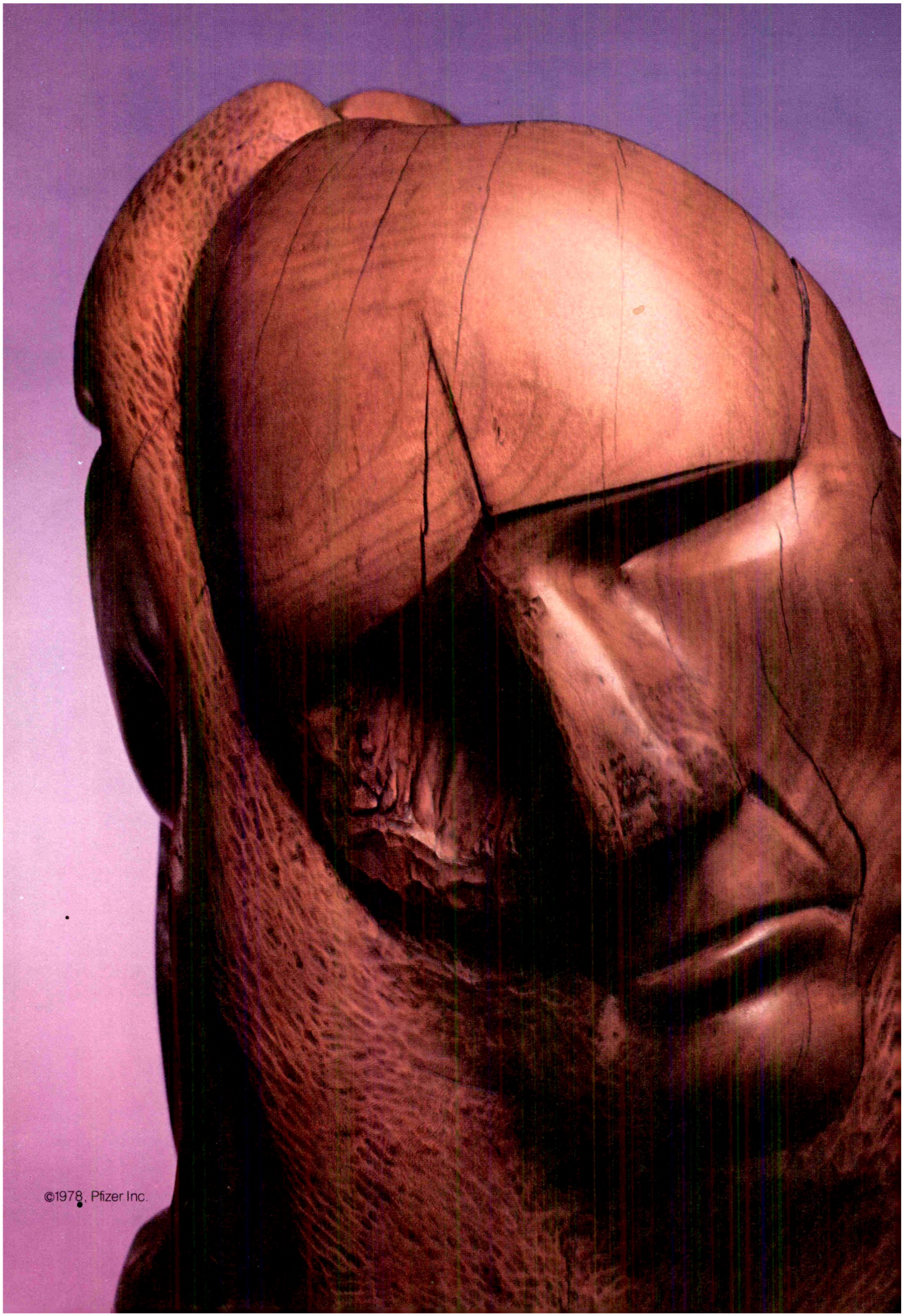
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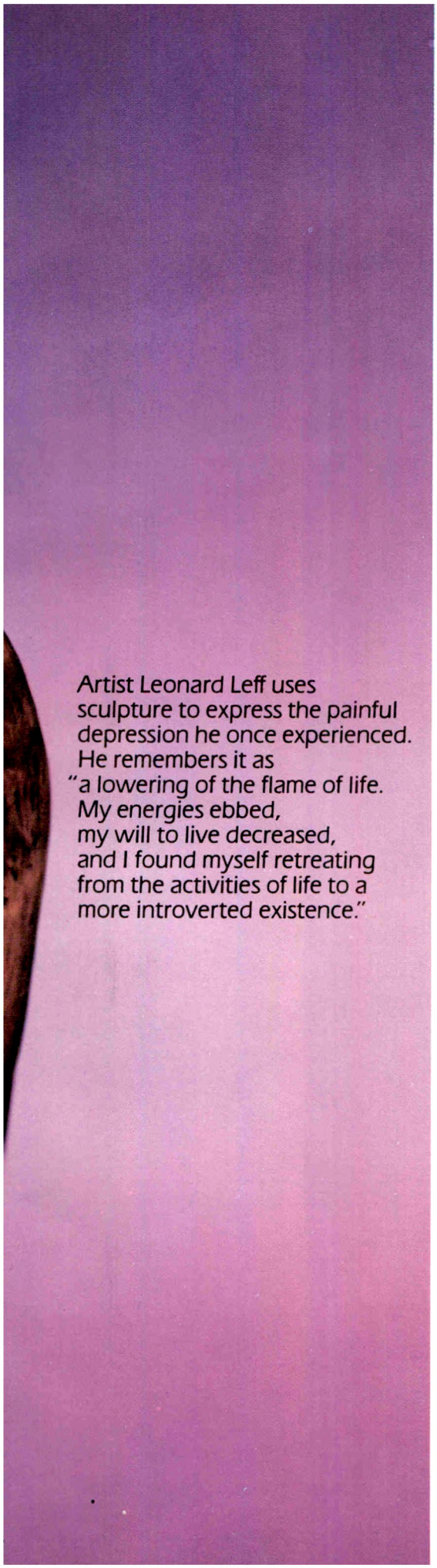
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Artist Leonard Leff uses sculpture to express the painful depression he once experienced. He remembers it as "a lowering of the flame of life. My energies ebbed, my will to live decreased, and I found myself retreating from the activities of life to a more introverted existence."

WHEN DEPRESSION EXPRESSES ITSELF

SINEQUAN®
(DOXEPIN HCl)

ANTIDEPRESSANT EFFECTIVENESS with convenient once-a-day *h.s.* dosage*

150-MG CAPSULE†

Also available in:
100-mg, 75-mg, 50-mg, 25-mg, 10-mg CAPSULES and ORAL CONCENTRATE, 10 mg/ml, in 120-ml (4-oz) bottles

*The total daily dosage of Sinequan, up to 150 mg, may be administered on a once-a-day schedule without loss of effectiveness.

† The 150-mg capsule strength is intended for maintenance therapy only and is not recommended for initiation of treatment.

See Brief Summary on next page for information on contraindications, warnings, precautions and adverse reactions.

ANTIDEPRESSANT EFFECTIVENESS

SINEQUAN®
(DOXEPIN HCl)

150-mg CAPSULE*

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which may improve patient compliance. The tota daily dosage, up to 150 mg per day, may be giver on a once-a-day schedule without loss of effectiveness. Sinequan may also be given on a divided dosage schedule, up to 300 mg per day.

PROMINENT SEDATIVE EFFECT

which may help to relieve the difficulty in falling and staying asleep, and the early-morning awakening often associated with depression.

ESTABLISHED ANTIANXIETY ACTIVITY

to help alleviate the anxiety which often accompanies clinical depression.

USUALLY WELL TOLERATED

At doses up to 150 mg per day, Sinequan does not generally affect the antihypertensive activity of guanethidine and related compounds. Tachycardia and hypotension have been reported occasionally. Drowsiness is the most commonly o served side effect. Dry mouth, blurred vision, consti pation and urinary retention have been reported.

EXTENDED RANGE OF DOSAGE STRENGTHS

for flexibility in individualizing therapy.

*The 150-mg capsule strength is intended for maintenance therapy only and is not recommended for initiation of treatment.

BRIEF SUMMARY
SINEQUAN® (doxepin HCl) Capsules/Oral Concentrate

Contraindications. Contraindicated in individuals who have shown hypersensitivity to the drug, and in patients with glaucoma or a tendency to urinary retention. These disorders should be ruled out, particularly in older patients. Possibility of cross sensitivity with other dibenzoxepines should be kept in mind.

Warnings. The once-a-day dosage regimen of SINEQUAN (doxepin HCl) in patients with intercurrent illness or patients taking other medications should be carefully adjusted. This is especially important in patients receiving other medications with anticholinergic effects.

Usage in Geriatrics: The use of SINEQUAN on a once-a-day dosage regimen in geriatric patients should be adjusted carefully based on the patient's condition.

Usage in Pregnancy: Reproduction studies performed in animals have shown no evidence of harm to the animal fetus. Since there is no experience in pregnant women receiving this drug, safety in pregnancy has not been established. There are no data with respect to the secretion of the drug in human milk and its effect on the nursing infant.

Usage in Children: Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

MAO Inhibitors: Serious side effects and even death have been reported following the concomitant use of certain drugs with MAO inhibitors. Therefore, MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with this drug. The exact length of time may vary and is dependent upon the particular MAO inhibitor being used, the length of time it has been administered and the dosage involved.

Usage with Alcohol: It should be borne in mind that alcohol ingestion may increase the danger inherent in any intentional or unintentional SINEQUAN overdosage. This is especially important in patients who may use alcohol excessively.

Precautions. Since drowsiness may occur with the use of this drug, patients should be warned of that possibility and cautioned against driving a car or operating dangerous machinery while taking this drug.

Patients should also be cautioned that their response to alcohol may be potentiated.

Since suicide is an inherent risk in any depressed patient, and may remain so until significant improvement has occurred, patients should be closely supervised during the early course of therapy. Prescriptions should be written for the smallest feasible amount.

Should increased symptoms of psychosis or shift to manic symptomatology occur, it may be necessary to reduce dosage or add a major tranquilizer to the dosage regimen.

Adverse Reactions. NOTE: Some of the adverse reactions noted below have not been specifically reported with SINEQUAN use. However, due to the close pharmacological similarities among the tricyclics, the reactions should be considered when prescribing SINEQUAN.

Anticholinergic Effects: Dry mouth, blurred vision, constipation, and urinary retention have been reported. If they do not subside with continued therapy, or become severe, it may be necessary to reduce the dosage.

Central Nervous System Effects: Drowsiness is the most commonly noticed side effect. This tends to disappear as therapy is continued. Other infrequently reported CNS side effects are confusion, disorientation, hallucinations, numbness, paresthesias, ataxia, and extrapyramidal symptoms and seizures.

Cardiovascular: Cardiovascular effects including hypotension and tachycardia have been reported occasionally.

Allergic: Skin rash, edema, photosensitization, and pruritus have occasionally occurred.

Hematologic: Eosinophilia has been reported in a few patients. There have been occasional reports of bone marrow depression manifesting as agranulocytosis, leukopenia, thrombocytopenia, and purpura.

Gastrointestinal: Nausea, vomiting, indigestion, taste disturbances, diarrhea, anorexia, and aphthous stomatitis have been reported. (See anticholinergic effects.)

Endocrine: Raised or lowered libido, testicular swelling, gynecomastia in males, enlargement of breasts and galactorrhea in the female, raising or lowering of blood sugar levels have been reported with tricyclic administration.

Other: Dizziness, tinnitus, weight gain, sweating, chills, fatigue, weakness, flushing, jaundice, alopecia, and headache have been occasionally observed as adverse effects.

Dosage and Administration. For most patients with illness of mild to moderate severi starting daily dose of 75 mg is recommended. Dosage may subsequently be increase decreased at appropriate intervals and according to individual response. The usual optir dose range is 75 mg/day to 150 mg/day.

In more severely ill patients higher doses may be required with subsequent gradual increa 300 mg/day if necessary. Additional therapeutic effect is rarely to be obtained by exceedi dose of 300 mg/day.

In patients with very mild symptomatology or emotional symptoms accompanying org disease, lower doses may suffice. Some of these patients have been controlled on doses as as 25-50 mg/day.

The total daily dosage of SINEQUAN (doxepin HCl) may be given on a divided or once-a dosage schedule. If the once-a-day schedule is employed the maximum recommended do 150 mg/day. This dose may be given at bedtime. **The 150 mg capsule strength is intende maintenance therapy only and is not recommended for initiation of treatment.**

Antianxiety effect is apparent before the antidepressant effect. Optimal antidepressant e may not be evident for two to three weeks.

Overdosage.

A. Signs and Symptoms

1. Mild: Drowsiness, stupor, blurred vision, excessive dryness of mouth.

2. Severe: Respiratory depression, hypotension, coma, convulsions, cardiac arrhythmias tachycardias.

Also: urinary retention (bladder atony), decreased gastrointestinal motility (paralytic ile hyperthermia (or hypothermia), hypertension, dilated pupils, hyperactive reflexes.

B. Management and Treatment

1. Mild: Observation and supportive therapy is all that is usually necessary.

2. Severe: Medical management of severe SINEQUAN overdosage consists of aggres supportive therapy. If the patient is conscious, gastric lavage, with appropriate precautior prevent pulmonary aspiration, should be performed even though SINEQUAN is rapidly absor The use of activated charcoal has been recommended, as has been continuous gastric la with saline for 24 hours or more. An adequate airway should be established in comatose pati and assisted ventilation used if necessary. EKG monitoring may be required for several c since relapse after apparent recovery has been reported. Arrhythmias should be treated with appropriate antiarrhythmic agent. It has been reported that many of the cardiovascular and symptoms of tricyclic antidepressant poisoning in adults may be reversed by the slow in venous administration of 1 mg to 3 mg of physostigmine salicylate. Because physostigmin rapidly metabolized, the dosage should be repeated as required. Convulsions may respor standard anticonvulsant therapy; however, barbiturates may potentiate any respiratory dep sion. Dialysis and forced diuresis generally are not of value in the management of overdos due to high tissue and protein binding of SINEQUAN.

Supply. SINEQUAN is available as capsules containing doxepin HCl equivalent to: 10 mg, 75 and 100 mg doxepin: bottles of 100, 1000, and unit-dose packages of 100 (10 x 10's). 25 mg 50 mg doxepin: bottles of 100, 1000, 5000, and unit-dose packages of 100 (10 x 10's). 150 doxepin: bottles of 50, 500, and unit-dose packages of 100 (10 x 10's). SINEQUAN Concentrate (10 mg/ml) is available in 120 ml bottles with an accompanying dropper calibr at 5 mg, 10 mg, 15 mg, 20 mg, and 25 mg. Each ml contains doxepin HCl equivalent to 1(doxepin. Just prior to administration, SINEQUAN Oral Concentrate should be diluted approximately 120 ml of water, whole or skimmed milk, or orange, grapefruit, tomato, prur pineapple juice. SINEQUAN Oral Concentrate is not physically compatible with a numb carbonated beverages. For those patients requiring antidepressant therapy who ar methadone maintenance, SINEQUAN Oral Concentrate and methadone syrup can be m together with Gatorade®, lemonade, orange juice, sugar water, Tang®, or water; but not grape juice. Preparation and storage of bulk dilutions is not recommended.

More detailed professional information available on request.

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The American Psychiatric Association announces publication of two volumes reporting the Conference on Education of Psychiatrists

held at Lake of the Ozarks, Missouri, June 9-15, 1975. Organized by the A.P.A. in cooperation with the American Academy of Child Psychiatry, the American Association of Chairmen of Departments of Psychiatry, and the American Association of Directors of Psychiatric Residency Training.

1. PSYCHIATRIC EDUCATION: PROLOGUE TO THE 1980's, Library Bound, 544 pages, $15.00.

This is a substantive, highly readable, report of the Conference, written by Anne H. Rosenfeld with an Editorial Board comprising: Ewald W. Busse, M.D., Chairperson of the Conference, Walter E. Barton, M.D., Bernard Holland, M.D., Donna Norris, M.D., Melvin Sabshin, M.D., Jeanne Spurlock, M.D., and Robert L. Robinson, M.A. (Staff Consultant).

The volume contains ten chapters covering the topical structure of the Conference: Goals and Values; Competence, Evaluation, and Quality Control; The Resident; Faculty; Educational Methods: Means in the Service of Ends; The Program Environment; Research; The Economics of Residency Training; Psychiatry in Its Social Context; and Retrospect and Prospect. It also contains nine appendices describing the organization of the Conference and its participants, together with selected model programs and related informational materials of value.

2. THE WORKING PAPERS OF THE 1975 CONFERENCE ON EDUCATION OF PSYCHIATRISTS Paper bound, 432 pages, $12.50.

Sixty two leading psychiatric educators served on seven preparatory commissions, one for each of seven topic areas, and prepared advance working papers as a basis for Conference deliberations. The topics and Commission Chairpersons were: Social Issues and Needs (James N. Sussex, M.D.); Goals and Objectives (Morton F. Reiser, M.D.); The Resident (David R. Hawkins, M.D.); Content, Methodology, Faculty and Environment (Milton Greenblatt, M.D.); Research (Daniel X. Freedman, M.D.); Economics (Herzl Spiro, M.D.); Evaluation and Quality Control (Eric Pfeiffer, M.D.). The Chairpersons, together with the Conference Chairperson, served as the Editorial Board for this volume.

Papers commenting on the reports of the commissions, published here for the first time, were presented at the Conference by Melvin Sabshin, M.D., Eugene B. Brody, M.D., Jeanne Spurlock, M.D., Jack R. Ewalt, M.D., Robert J. Stoller, M.D., James R. Comer, M.D., and Bernard Holland, M.D.

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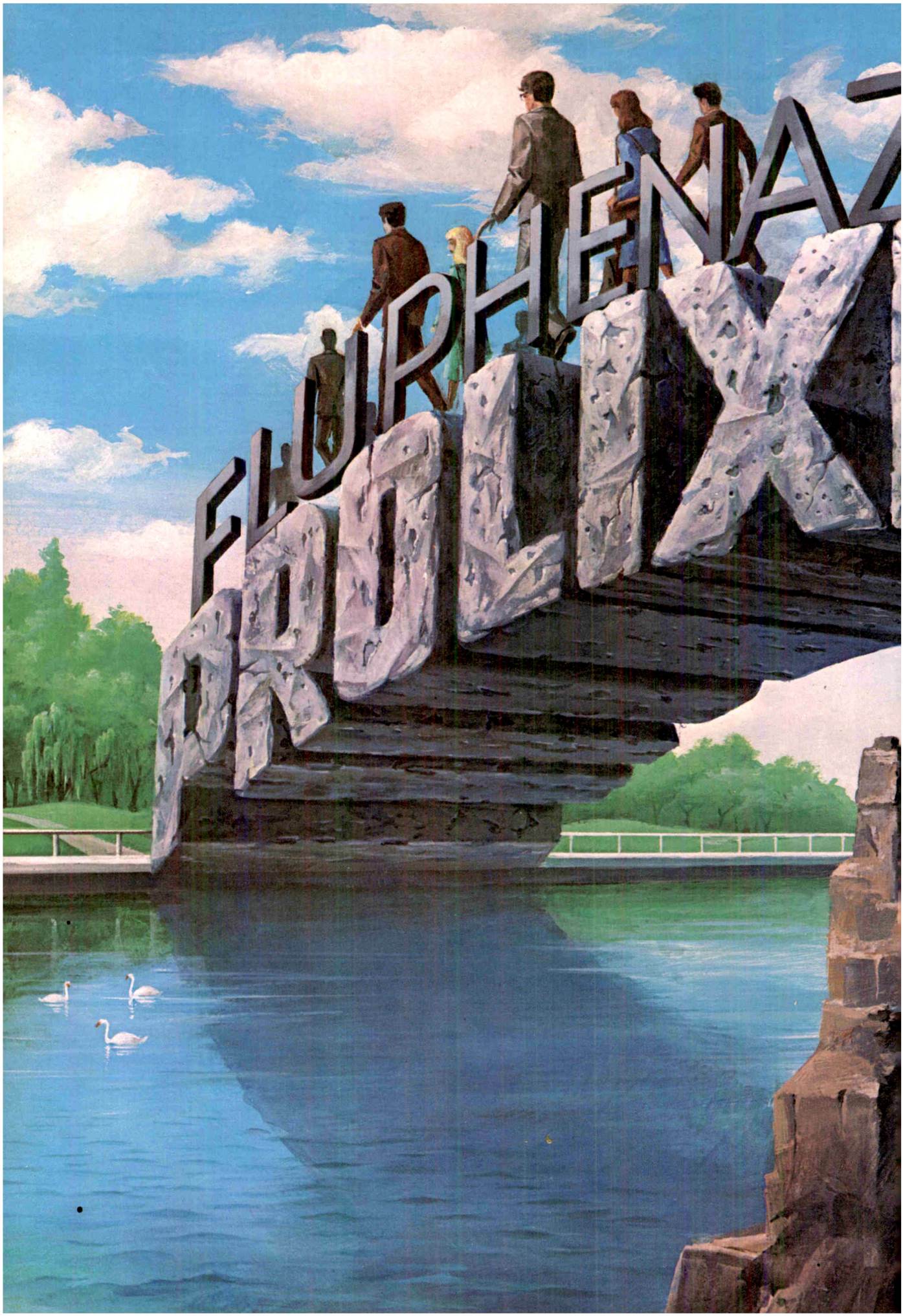
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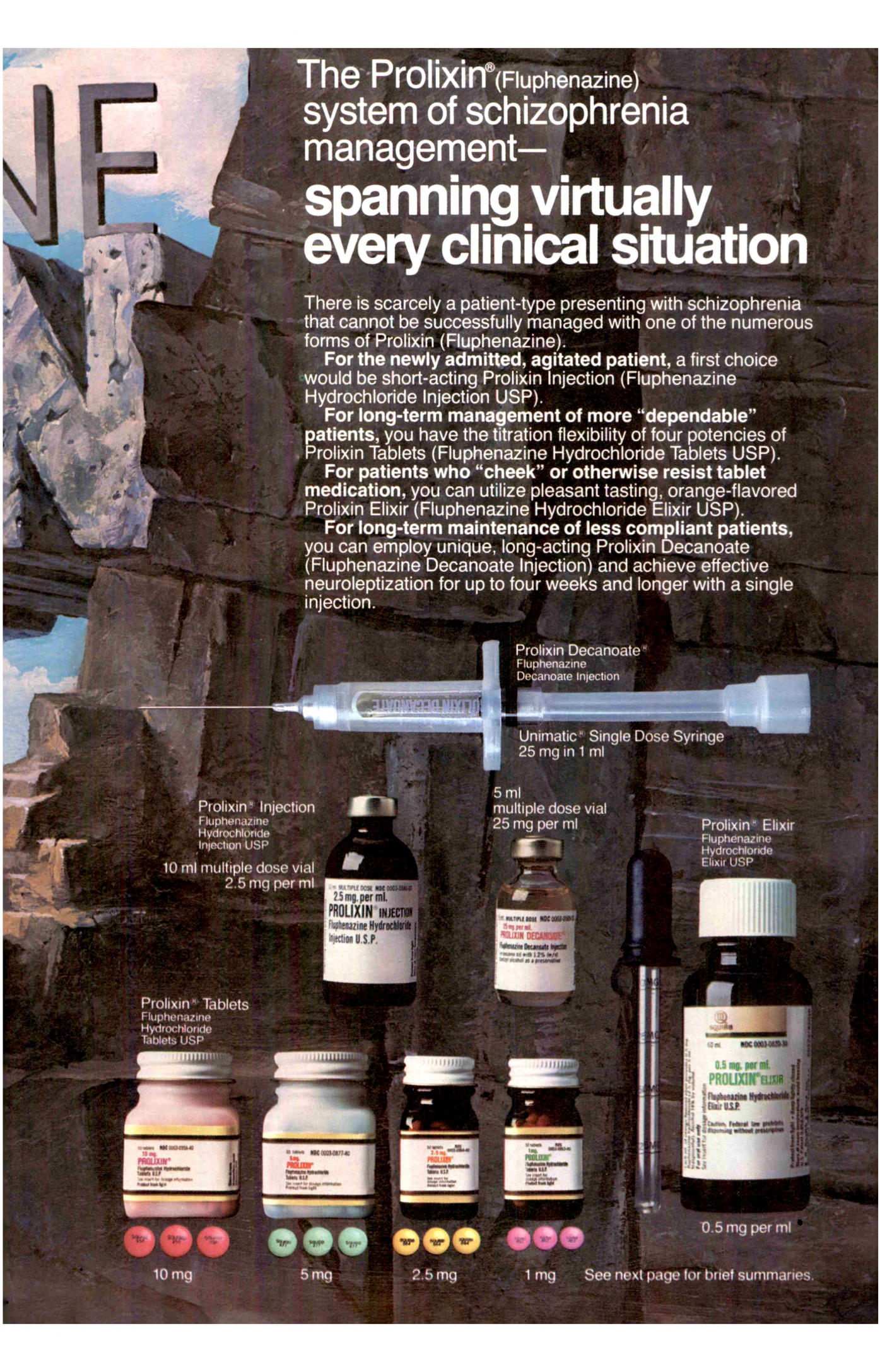
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For the newly admitted, agitated patient, a first choice would be short-acting Prolixin Injection (Fluphenazine Hydrochloride Injection USP).

For long-term management of more "dependable" patients, you have the titration flexibility of four potencies of Prolixin Tablets (Fluphenazine Hydrochloride Tablets USP).

For patients who "cheek" or otherwise resist tablet medication, you can utilize pleasant tasting, orange-flavored Prolixin Elixir (Fluphenazine Hydrochloride Elixir USP).

For long-term maintenance of less compliant patients, you can employ unique, long-acting Prolixin Decanoate (Fluphenazine Decanoate Injection) and achieve effective neuroleptization for up to four weeks and longer with a single injection.



See next page for brief summaries.

PROLIXIN® (Fluphenazine Hydrochloride)
TABLETS/ELIXIR/INJECTION

Prolixin Tablets (Fluphenazine Hydrochloride Tablets USP) provide 1, 2.5, 5, or 10 mg fluphenazine hydrochloride per tablet. Prolixin Elixir (Fluphenazine Hydrochloride Elixir USP) provides 0.5 mg fluphenazine hydrochloride per ml (2.5 mg per 5 ml teaspoonful) with 14% alcohol by volume. Prolixin Injection (Fluphenazine Hydrochloride Injection USP) provides 2.5 mg fluphenazine hydrochloride per ml; it contains 0.1% methylparaben and 0.01% propylparaben as preservatives.

CONTRAINDICATIONS: In presence of suspected or established subcortical brain damage. In patients who have a blood dyscrasia or liver damage, or who are receiving large doses of hypnotics, or who are comatose or severely depressed. In patients who have shown hypersensitivity to fluphenazine; cross-sensitivity to phenothiazine derivatives may occur.

WARNINGS: Mental and physical abilities required for driving a car or operating heavy machinery may be impaired by use of this drug. Potentiation of effects of alcohol may occur. Safety and efficacy in children have not been established because of inadequate experience in use in children.

Usage in Pregnancy: Safety for use during pregnancy has not been established; weigh possible hazards against potential benefits if administering this drug to pregnant patients.

PRECAUTIONS: Caution must be exercised if another phenothiazine compound caused cholestatic jaundice, dermatoses or other allergic reactions because of the possibility of cross-sensitivity. When psychotic patients on large doses of a phenothiazine drug are to undergo surgery, hypotensive phenomena should be watched for; less anesthetics or central nervous system depressants may be required. Because of added anticholinergic effects, fluphenazine may potentiate the effects of atropine.

Use fluphenazine cautiously in patients exposed to extreme heat or phosphorus insecticides; in patients with a history of convulsive disorders since grand mal convulsions have occurred; and in patients with special medical disorders such as mitral insufficiency or other cardiovascular diseases, and pheochromocytoma. Bear in mind that with prolonged therapy there is the possibility of liver damage, pigmentary retinopathy, lenticular and corneal deposits, and development of irreversible dyskinesia.

There is sufficient experimental evidence to conclude that chronic administration of antipsychotic drugs which increase prolactin secretion has the potential to induce mammary neoplasms in rodents under the appropriate conditions. There are recognized differences in the physiological role of prolactin between rodents and humans. Since there are, at present, no adequate epidemiological studies, the relevance to human mammary cancer risk from prolonged exposure to fluphenazine hydrochloride and other antipsychotic drugs is not known.

Periodic checking of hepatic and renal functions and blood picture should be done. Monitor renal function of patients on long-term therapy; if BUN becomes abnormal, discontinue fluphenazine. "Silent pneumonias" are possible.

Abrupt Withdrawal: In general, phenothiazines do not produce psychic dependence. However, gastritis, nausea and vomiting, dizziness, and tremulousness have been reported following abrupt cessation of high dose therapy; reports suggest that these symptoms can be reduced if concomitant antiparkinsonian agents are continued for several weeks after the phenothiazine is withdrawn.

ADVERSE REACTIONS: *Central Nervous System*—Extrapyramidal symptoms are most frequently reported. Most often these symptoms are reversible, but they may be persistent. They include pseudoparkinsonism, dystonia, dyskinesia, akathisia, oculogyric crises, opisthotonos, hyperreflexia. The incidence and severity of such reactions will depend more on individual patient sensitivity, but dosage level and patient age are also determinants. As these reactions may be alarming, the patient should be forewarned and reassured. These reactions can usually be controlled by administration of an anti-parkinsonian drug such as benztropine mesylate and by subsequent reduction in dosage.

Persistent Tardive Dyskinesia: As with all antipsychotic agents, persistent and sometimes irreversible tardive dyskinesia may appear in some patients on long-term therapy or may occur after discontinuation of drug. The risk seems greater in elderly patients, especially females, on high dosages. The syndrome is characterized by rhythmical involuntary movements of tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and may be accompanied by involuntary movements of extremities. There is no known effective therapy for tardive dyskinesia; usually the symptoms are not alleviated by antiparkinsonism agents. If the symptoms appear, discontinuation of all antipsychotic agents is suggested. The syndrome may be masked if treatment is reinstituted, or drug dosage increased, or a different antipsychotic agent used. Reports are that fine vermicular movements of the tongue may be an early sign of the syndrome which may not develop if medication is stopped at that time.

Phenothiazine derivatives have been known to cause restlessness, excitement, or bizarre dreams; reactivation or aggravation of psychotic processes may be encountered. If drowsiness or lethargy occur, the dosage may need to be reduced. Dosages, far in excess of the recommended amounts, may induce a catatonic-like state.

Autonomic Nervous System—Hypertension and fluctuations in blood pressure have been reported. Although hypotension is rarely a problem, patients with pheochromocytoma, cerebral vascular or renal insufficiency or severe cardiac reserve deficiency such as mitral insufficiency appear to be particularly prone to this reaction and should be observed carefully. Supportive measures including intravenous vasopressor drugs should be instituted immediately should severe hypotension occur; Levarterenol Bitartrate Injection is the most suitable drug; *epinephrine should not be used* since phenothiazine derivatives have been found to reverse its action. Nausea, loss of appetite, salivation, polyuria, perspiration, dry mouth, headache and constipation may occur. Reducing or temporarily discontinuing the dosage will usually control these effects. Blurred vision, glaucoma, bladder paralysis, fecal impaction, paralytic ileus, tachycardia, or nasal congestion have occurred in some patients on phenothiazine derivatives.

Metabolic and Endocrine—Weight change, peripheral edema, abnormal lactation, gynecomastia, menstrual irregularities, false results on pregnancy tests, impotency in men and increased libido in women have occurred in some patients on phenothiazine therapy.

Allergic Reactions—Itching, erythema, urticaria, seborrhea, photosensitivity, eczema and exfoliative dermatitis have been reported with phenothiazines. The possibility of anaphylactoid reactions should be borne in mind.

Hematologic—Blood dyscrasias including leukopenia, agranulocytosis, thrombocytopenic or nonthrombocytopenic purpura, eosinophilia, and pancytopenia have been observed with phenothiazines. If soreness of the mouth, gums or throat or any symptoms of upper respiratory infection occur and confirmatory leukocyte count indicates cellular depression, therapy should be discontinued and other appropriate measures instituted immediately.

Hepatic—Liver damage manifested by cholestatic jaundice, particularly during the first months of therapy, may occur; treatment should be discontinued. A cephalin flocculation increase, sometimes accompanied by alterations in other liver function tests, has been reported in patients who have had no clinical evidence of liver damage.

Others—Sudden deaths have been reported in hospitalized patients on phenothiazines. Previous brain damage or seizures may be predisposing factors. High doses should be avoided in known seizure patients. Shortly before death, several patients showed flare-ups of psychotic behavior patterns. Autopsy findings have usually revealed acute fulminating pneumonia or pneumonitis, aspiration of gastric contents, or intramyocardial lesions. Although not a general feature of fluphenazine, potentiation of central nervous system depressants such as opiates, analgesics, antihistamines, barbiturates, and alcohol may occur.

Systemic lupus erythematosus-like syndrome, hypotension severe enough to cause fatal cardiac arrest, altered electrocardiographic and electroencephalographic tracings, altered cerebrospinal fluid proteins, cerebral edema, asthma, laryngeal edema, and angioneurotic edema; with long-term use, skin pigmentation and lenticular and corneal opacities have occurred with phenothiazines.

For full prescribing information, consult package inserts.

HOW SUPPLIED: *Tablets*—1 mg in bottles of 50 and 500; 2.5 mg and 5 mg in bottles of 50 and 500 and in Unimatic® single-dose cartons of 100; 10 mg in bottles of 50 and 500. *Elixir*—in bottles of 473 ml (1 pint) and in 60 ml dropper-assembly bottles with dropper calibrated at 0.5 ml (0.25 mg), 1 ml (0.5 mg), 1.5 ml (0.75 mg), and 2 ml (1 mg). *Injection*—in multiple-dose vials of 10 ml.

PROLIXIN DECANOATE®
Fluphenazine Decanoate Injection

Prolixin Decanoate (Fluphenazine Decanoate Injection) provides 25 mg fluphenazine deca per ml in a sesame oil vehicle with 1.2% (w/v) benzyl alcohol as a preservative.

CONTRAINDICATIONS: In presence of suspected or established subcortical brain dama patients who have a blood dyscrasia or liver damage, or who are receiving large dos hypnotics, or who are comatose or severely depressed. In patients who have shown hype sitivity to fluphenazine; cross-sensitivity to phenothiazine derivatives may occur.

Not intended for use in children under 12.

WARNINGS: Mental and physical abilities required for driving a car or operating heav chinery may be impaired by use of this drug. Physicians should be alert to the possibilit severe adverse reactions may occur which require immediate medical attention. Potentia effects of alcohol may occur. Safety and efficacy in children have not been established be of inadequate experience in use in children.

Usage in Pregnancy: Safety for use during pregnancy has not been established; possible hazards against potential benefits if administering this drug to pregnant patier

PRECAUTIONS: Caution must be exercised if another phenothiazine compound caused c tatic jaundice, dermatoses or other allergic reactions because of the possibility of sensitivity. When psychotic patients on large doses of a phenothiazine drug are to un surgery, hypotensive phenomena should be watched for; less anesthetics or central ne system depressants may be required. Because of added anticholinergic effects, flupher may potentiate the effects of atropine.

Use fluphenazine decanoate cautiously in patients exposed to extreme heat or phosp insecticides; in patients with a history of convulsive disorders since grand mal convulsions occurred; and in patients with special medical disorders such as mitral insufficiency or cardiovascular diseases, and pheochromocytoma. Bear in mind that with prolonged th there is the possibility of liver damage, pigmentary retinopathy, lenticular and corneal dep and development of irreversible dyskinesia.

Fluphenazine decanoate should be administered under the direction of a physician e enced in the clinical use of psychotropic drugs. Periodic checking of hepatic and renal fun and blood picture should be done. Renal function of patients on long-term therapy sho monitored; if BUN becomes abnormal, treatment should be discontinued. "Silent pneum are possible.

There is sufficient experimental evidence to conclude that chronic administration of a chotic drugs which increase prolactin secretion has the potential to induce mammary neop in rodents under the appropriate conditions. There are recognized differences in the phy ical role of prolactin between rodents and humans. Since there are, at present, no ade epidemiological studies, the relevance to human mammary cancer risk from prolonged exp to fluphenazine decanoate and other antipsychotic drugs is not known.

ADVERSE REACTIONS: *Central Nervous System*—Extrapyramidal symptoms are mo quently reported. Most often these symptoms are reversible, but they may be persistent include pseudoparkinsonism, dystonia, dyskinesia, akathisia, oculogyric crises, opistho hyperreflexia. Muscle rigidity sometimes accompanied by hyperthermia has been re withdrawal of fluphenazine decanoate. One can expect a higher incidence of such rea with fluphenazine decanoate than with less potent piperazine derivatives or straight phenothiazines. The incidence and severity will depend more on individual patient sensitiv dosage level and patient age are also determinants. As these reactions may be alarmir patient should be forewarned and reassured. These reactions can usually be control administration of an antiparkinsonian drug such as benztropine mesylate and by subse reduction in dosage.

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Phenothiazine derivatives have been known to cause restlessness, excitement, or dreams; reactivation or aggravation of psychotic processes may be encountered. If drow or lethargy occur, the dosage may need to be reduced. Dosages, far in excess of the mended amounts, may induce a catatonic-like state.

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Metabolic and Endocrine—Weight change, peripheral edema, abnormal lac gynecomastia, menstrual irregularities, false results on pregnancy tests, impotency in m increased libido in women have occurred in some patients on phenothiazine therapy.

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Systemic lupus erythematosus-like syndrome, hypotension severe enough to caus cardiac arrest, altered electrocardiographic and electroencephalographic tracings, cerebrospinal fluid proteins, cerebral edema, asthma, laryngeal edema, and angion edema; with long-term use, skin pigmentation and lenticular and corneal opacities have o with phenothiazines. Local tissue reactions occur only rarely with injections of fluph decanoate.

For full prescribing information, consult package insert.

HOW SUPPLIED: 1 ml Unimatic® single-dose preassembled syringes and cartridge-units and 5 ml vials.

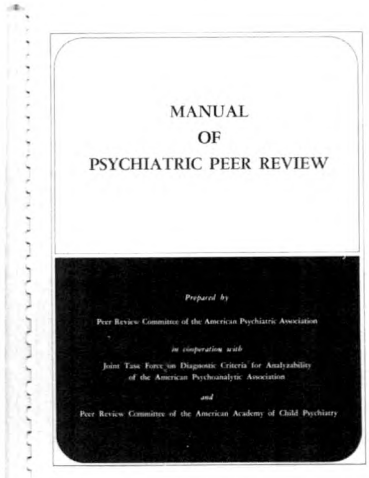
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Announcing Publication . . .

MANUAL OF PSYCHIATRIC PEER REVIEW

by the Peer Review Committee of the American Psychiatric Association, Donald G. Langsley, M.D., Chairperson in cooperation with the Joint Task Force on Diagnostic Criteria for Analyzability of the American Psychoanalytic Association, William Offenkrantz, M.D., Chairperson, and the Peer Review Committee of the American Academy of Child Psychiatry. Larry B. Silver, M.D., Chairperson.



"This Manual provides a set of guidelines for district branches of APA and their peer review committees which should be adapted and modified in terms of local criteria, standards, and practices. The guidelines are not intended to be hard and fast rules. They are screening criteria, and not regulations to control practice. They are so designed that they can be utilized by non-physicians to identify those cases that can profitably be reviewed by psychiatrists serving on peer review committees. This document should be viewed as transitional, subject to revision as experience is gained, and continually updated in response to advances in psychiatric knowledge and changes in patterns of practice. . . . The application of these standards, and refinement through experience must rest with the peer review committees of district branches. . . . It should help them to move ahead in the development of a peer review for American psychiatry."

Robert W. Gibson, M.D.
President

Part I titled PSYCHIATRIC PEER REVIEW contains sections on Basic Relationships and Responsibilities, Appointment of Peer Review Committees, Funding, Operations and Procedures, Medical Care Evaluation Studies, Appeals Processes, Special Problems, Confidentiality, Educational Aspects, Medical Ethics, Advocacy, Interdisciplinary Aspects, with Appendices on Model Screening Format for Inpatient Treatment for Alcoholism, Model Screening Format for Outpatient Treatment, Sample Form for Requesting Extension of Office Treatment, Claims Review Guidelines Suggestions, APA Position Papers on Peer Review, Bibliography.

Part II titled PSYCHOANALYTIC PEER REVIEW has sections on Steps in Peer Review for Psychoanalysis, Guidelines for Model Criteria Sets, Pilot Application of Guidelines for Model Criteria Sets, Sample Peer Review Form, Sample data from Pilot Applications of Guidelines, and Glossary of Terms Used.

Part III titled PEER REVIEW AND CHILD PSYCHIATRY contains sections of Special Considerations, Legal Rights of Children, Issue of Confidentiality, Conclusions, References, and Appendices on Model Screen Criteria Format for Intermediate Care, for Acute Care in a Short Stay Facilities, and for Partial Hospitalization, Outpatient Diagnostic Evaluation Procedures, and Outpatient Treatment for Children and Adolescents.

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ANXIETY

SIMPLE SYMPTOM OR SYNDROME



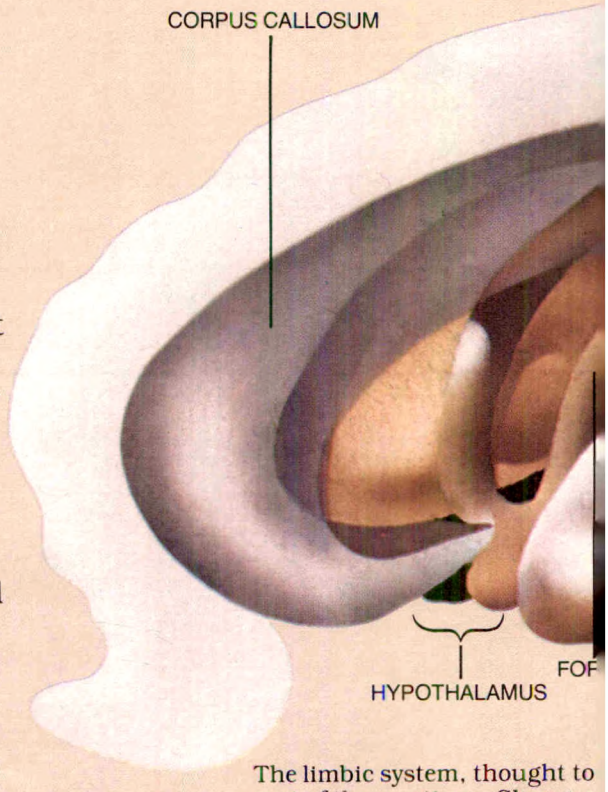
Some psychiatrists consider anxiety a simple symptom complex

Some psychiatrists maintain that anxiety is a self-limiting symptomatic reaction to stressful circumstances, whether physical or psychological, that occurs when the magnitude of the stress surpasses the patient's ability to cope. Most stressors are a form of loss, but in some patients just the anticipation of loss can be emotionally disabling. Anxiety is the response when the patient's internal milieu is threatened by change, whether it is perceived as desirable or undesirable.[1]

1. *Stress Without Distress.* Hans Selye, interviewed by McCrie R: *Pract Psychol for Physicians* 3:52-56, Aug 1976

Others suggest it is a syndrome, a true disorder[2]

Psychiatrists who view anxiety as syndrome theorize that its etiolog and/or maintenance is primarily pendent on events within the cen nervous system. They point out t certain individuals appear to be i ently more anxiety prone than others—seem not only to perceive wider range of situations as threa ing but to respond to these situat with more pronounced levels of C arousal and subjective anxiety. O searcher,[3] who makes a sharp dis



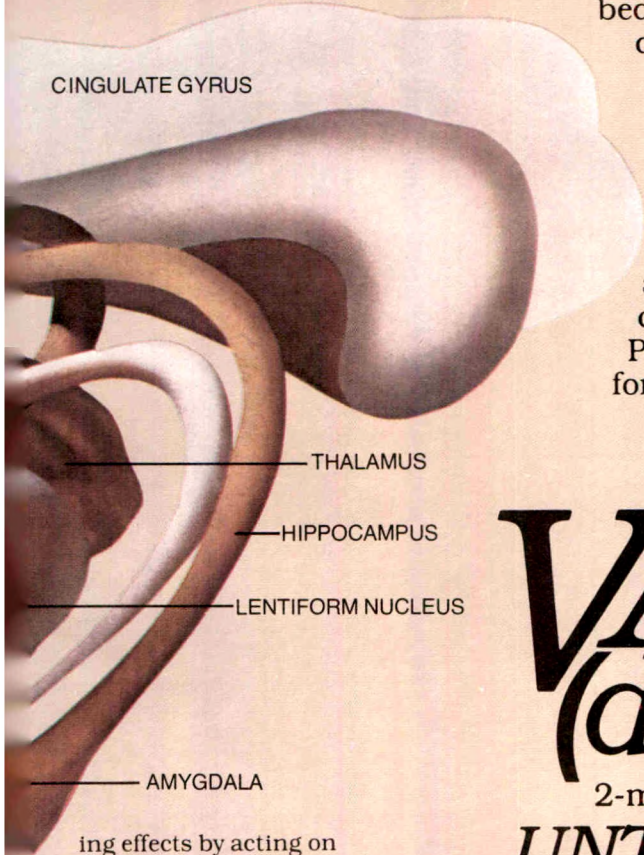
The limbic system, thought to seat of the emotions. Changes limbic system function could l peripheral overarousal—inter by the patient as anxiety. Stud animals and a limited study in man beings support the prem Valium (diazepam) produces i

ROCHE

n between *state* anxiety (A-state), nsitory emotional reactions to ess, and *trait* anxiety (A-trait), a rel- vely permanent, characteristic way esponding to stress, has found rked individual differences in the subjects perceive external stress- Among other differences: high rait persons tend to be more self- recatory, more afraid of failure. us, they perceive ego-involving situ- ons as more "dangerous" than do A-trait individuals—and often nifest more intense levels of ap- hension and other common psychic ptoms of state anxiety.

sher S: Anxiety: What causes it and how to it, a panel discussion reprinted in *Med Opin* 25, July 1977

ielberger CD: Anxiety: State-Trait-Process, in *s and Anxiety*, vol. 1, edited by Spielberger CD, son IG. New York, John Wiley & Sons, 1975



ing effects by acting on parts of the limbic system, the nus and hypothalamus.[4] ver, further research will be sary to establish the modes and of action of Valium (diazepam) man beings.

ier MAB, Crandall PH, Walsh GO: *Exp* 51:241-258, Apr 1976

Symptom or syndrome, anxiety responds to Valium (diazepam)

When a patient's coping mechanisms are insufficient to deal with his level of anxiety, Valium (diazepam) can promptly and effectively relieve the anxiety and its somatic symptoms. Initial calming often occurs in hours. After several days of Valium therapy, anxiety relief is pronounced and sustained. And as the anxiety is reduced, accompanying tension, insomnia-like symptoms and somatic symptoms are also relieved.

Available in three scored tablet strengths, Valium allows you to set, adjust and readjust dosage as appropriate. For patients prone to anxiety at bedtime, you may want to add an *h.s.* dose to a *b.i.d.* or *t.i.d.* regimen.

Valium is usually well tolerated; side effects more severe than drowsiness, fatigue or ataxia are rare. As with all CNS-acting medications, patients should be cautioned against drinking alcohol or operating dangerous machinery while on Valium therapy. Periodic reassessment of the need for Valium is recommended.

VALIUM® (diazepam) IV

2-mg, 5-mg, 10-mg scored tablets

UNTIL THE PATIENT CAN COPE AGAIN



Please see following page for a summary of product information.

VALIUM® (diazepam)

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Tension and anxiety states; somatic complaints which are concomitants of emotional factors; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome; convulsive disorders (not for sole therapy).

The effectiveness of Valium (diazepam) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

Supplied: Valium® (diazepam) Tablets, 2 mg, 5 mg and 10 mg—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50, available singly and in trays of 10.

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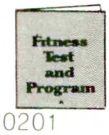


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As Canada's fourth largest province, Alberta (1,954,500 population) is experiencing a phase of economic growth primarily based on its energy resources. Its residents enjoy a vigorous cultural life and a high standard of facilities and services. The development of the Health Sciences Centre (Phase I cost is U.S. $85,000,000) will make its capital city, Edmonton, a major centre in health care research.

Rapid expansion of Forensic Psychiatric Services in Alberta and the erection of a new Forensic Facility at Alberta Hospital, Edmonton, have resulted in the creation of three new positions. The psychiatrists will be expected to allot their time to work with Alberta Hospital Forensic Services and the Federal and Provincial Correctional Institutions, which are all in close proximity. If the successful appointees desire they may conduct sessions at the Forensic Out-Patient Service located in downtown Edmonton.

In addition, general and specialty positions exist in various large and small urban locations with Alberta Mental Health Services. Most are team-oriented, community or part time community positions backed by excellent medical and paramedical staff and services.

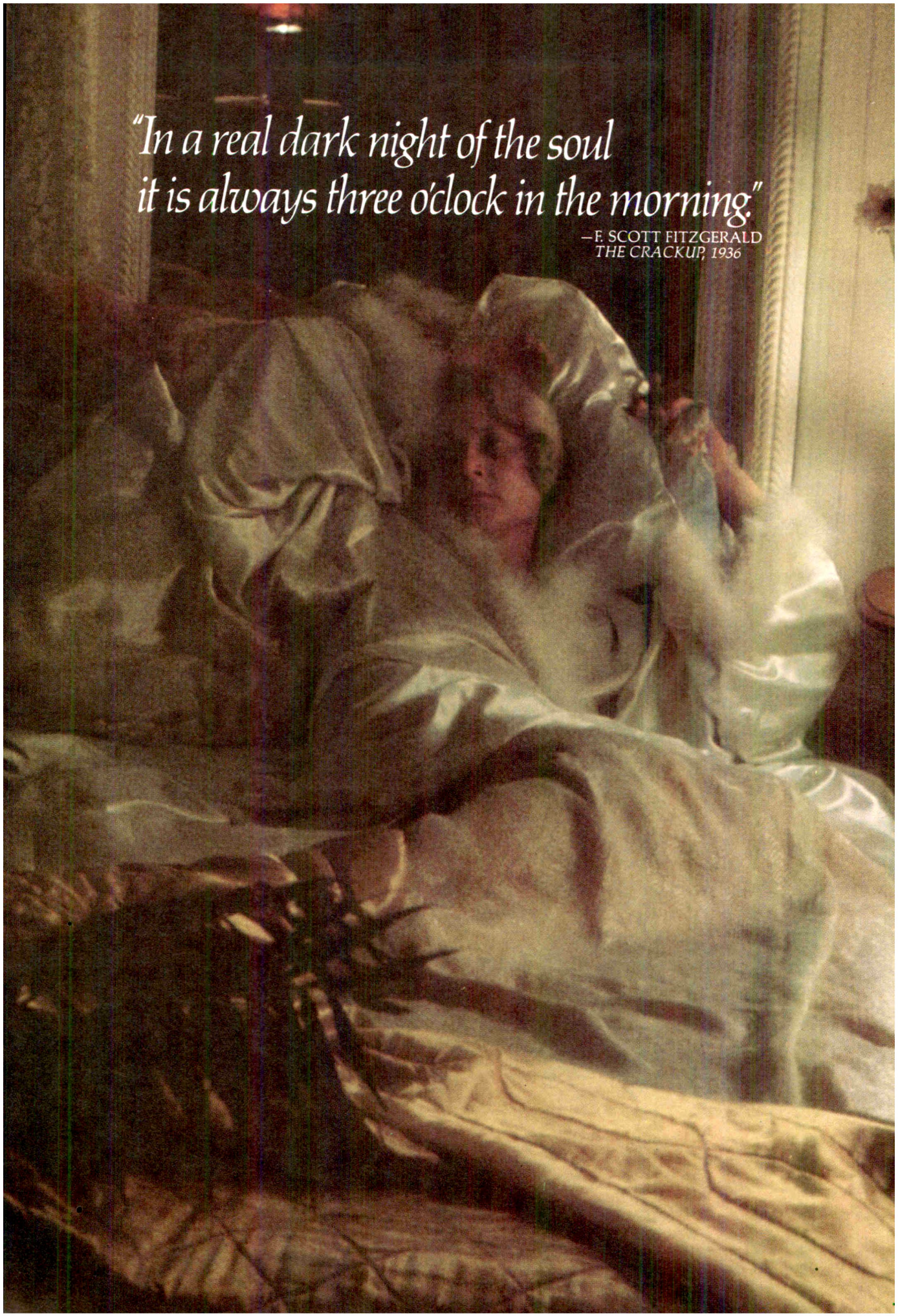
Basic requirement is eligibility to practice medicine in Alberta. Advanced training is desirable and recognized in our salary schedule.

Salary range is from U.S. $25,000 to U.S. $48,430, plus duty roster pay. Financial arrangements include removal expenses. (Exchange calculated as of December 13, 1978.)

For further information, call Dr. David R. Cornish (collect) at (403) 973-2438 OR write, in confidence, to:

Dr. David R. Cornish, Medical Director, Alberta Hospital
Box 307, Edmonton, Alberta T5J 2J7

Alberta


"In a real dark night of the soul
it is always three o'clock in the morning."
—F. SCOTT FITZGERALD
THE CRACKUP, 1936

Insomnia
a shade of blue that often accompanies depression

And, in anxiety/depression, Adapin® (doxepin HCl) often helps restore disturbed sleep patterns, such as early morning wakening, with a single daily dose at bedtime. Adapin quickly relieves the patient's anxiety, gradually brightens his mood and outlook, with optimal antidepressant response usually evident within two to three weeks.

Brief Summary of Prescribing Information
ADAPIN® (doxepin HCl) Capsules

Indications—Relief of symptoms of anxiety and depression.

Contraindications—Glaucoma, tendency toward urinary retention, or hypersensitivity to doxepin.

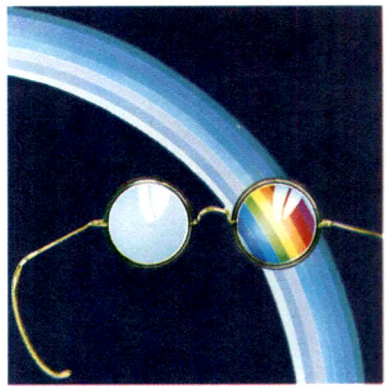
Warnings—Adapin has not been evaluated for safety in pregnancy. No evidence of harm to the animal fetus has been shown in reproductive studies. There are no data concerning secretion in human milk, nor on effect in nursing infants.
Usage in children under 12 years of age is not recommended. MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with this drug, as serious side-effects and death have been reported with the concomitant use of certain drugs and MAO inhibitors.
In patients who may use alcohol excessively potentiation may increase the danger inherent in any suicide attempt or overdosage.

Precautions—Drowsiness may occur and patients should be cautioned against driving a motor vehicle or operating hazardous machinery. Since suicide is an inherent risk in depressed patients they should be closely supervised while receiving treatment. Although Adapin has shown effective tranquilizing activity, the possibility of activating or unmasking latent psychotic symptoms should be kept in mind.

Adverse Reactions—Dry mouth, blurred vision and constipation have been reported. Drowsiness has also been observed.
Adverse effects occurring infrequently include extrapyramidal symptoms, gastrointestinal reactions, secretory effects such as heating, tachycardia and hypotension. Weakness, dizziness, fatigue, weight gain, edema, paresthesias, flushing, chills, tinnitus, claustrophobia, decreased libido, rash and pruritus may also occur.

Dosage and Administration—In mild to moderate anxiety and/or depression: 25 mg t.i.d. Increase or decrease the dosage according to individual response. Daily dosage, up to 150 mg may be taken at one time without loss of effectiveness. Usual optimum daily dosage is 75 mg to 150 mg per day not to exceed 300 mg per day.
Antianxiety effect usually precedes the antidepressant effect by two or three weeks.

How Supplied—Each capsule contains doxepin, as the hydrochloride: 10 mg, 25 mg, 50 mg and 100 mg capsules in bottles of 100 and 1000.
For complete prescribing information please see package insert or PDR.



When they see life in shades of blue... help them see life in all its colors.

Adapin®
(doxepin HCl)

single daily dose recommended h.s.



10 mg capsules
25 mg capsules
50 mg capsules
100 mg capsules

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ROCHESTER, NEW YORK 14623

Molindone HCl

Lidone™ treats the classic target symptoms



with less likelihood of certain troublesome effects

Lidone lets you control key schizophrenic symptoms,

- **Significant gains have been reported** following use of Lidone for patients experiencing hallucinations, conceptual disorganization, hostility, unusual thought content, and other schizophrenic manifestations.
- **Progress has also been noted** in the social outlook of patients. Emotional withdrawal has lessened with the Lidone therapy. Many patients have improved their social behavior and interpersonal relationships.

yet keep a low side effects profile

- **There have been no reports of persistent** tardive dyskinesia, excessive weight gain, impotence, lens opacities, or skin pigmentation. And the incidence of significant hypotension is rare (below 1% in clinical studies). Other side effects are similar to comparable agents, and seldom require discontinuation of therapy.

Lidone™
Molindone HCl

5, 10, 25 mg capsules

ABBOTT

Please see overleaf for prescribing information

TM—TRADEMARK

8063258 R

Prescribing Information for

Lidone™ (molindone hydrochloride)

DESCRIPTION — LIDONE (molindone hydrochloride) is a dihydroindolone compound which is not structurally related to the phenothiazines, the butyrophenones or the thioxanthenes.

LIDONE is 3-ethyl-6, 7-dihydro-2-methyl-5-(morpholinomethyl) indol-4(5H)-one hydrochloride. It is a white crystalline powder, freely soluble in water and alcohol and has a molecular weight of 312.67.

ACTIONS — LIDONE (molindone hydrochloride) has a pharmacological profile in laboratory animals which predominantly resembles that of major tranquilizers causing reduction of spontaneous locomotion and aggressiveness, suppression of a conditional response and antagonism of the bizarre stereotyped behavior and hyperactivity induced by amphetamines. In addition, LIDONE antagonizes the depression caused by the tranquilizing agent tetrabenazine.

In human clinical studies tranquilization is achieved in the absence of muscle relaxing or incoordinating effects. Based on EEG studies, LIDONE exerts its effect on the ascending reticular activating system.

Human metabolic studies show LIDONE (molindone hydrochloride) to be rapidly absorbed and metabolized when given orally. Unmetabolized drug reached a peak blood level at 1.5 hours. Pharmacological effect from a single oral dose persists for 24 to 36 hours. There are 36 recognized metabolites with less than 2 to 3% unmetabolized LIDONE being excreted in urine and feces.

INDICATIONS — LIDONE (molindone hydrochloride) is indicated in the management of the manifestations of schizophrenia.

CONTRAINDICATIONS — LIDONE (molindone hydrochloride) is contraindicated in severe central nervous system depression (alcohol, barbiturates, narcotics, etc.) or comatose states, and in patients with known hypersensitivity to the drug.

WARNINGS

Usage in Pregnancy: Studies in pregnant patients have not been carried out. Reproduction studies have been performed in the following animals:

Pregnant Rats Oral Dose —
20 mg/kg/day — 10 days, no adverse effect
40 mg/kg/day — 10 days, no adverse effect

Pregnant Mice Oral Dose —
20 mg/kg/day — 10 days, slight increase resorptions
40 mg/kg/day — 10 days, slight increase resorptions

Pregnant Rabbits Oral Dose —
5 mg/kg/day — 12 days, no adverse effect
10 mg/kg/day — 12 days, no adverse effect
20 mg/kg/day — 12 days, no adverse effect

Animal reproductive studies have not demonstrated a teratogenic potential. The anticipated benefits must be weighed against the unknown risks to the fetus if used in pregnant patients.

Nursing Mothers: Data are not available on the content of LIDONE (molindone hydrochloride) in the milk of nursing mothers.

Usage in Children: Use of LIDONE (molindone hydrochloride) in children below the age of twelve years is not recommended because safe and effective conditions for its usage have not been established.

PRECAUTIONS — Some patients receiving LIDONE (molindone hydrochloride) may note drowsiness initially and they should be advised against activities requiring mental alertness until their response to the drug has been established.

Increased activity has been noted in patients receiving LIDONE. Caution should be exercised where increased activity may be harmful.

LIDONE does not lower the seizure threshold in experimental animals to the degree noted with more sedating antipsychotic drugs. However, convulsive seizures have been reported in a few instances.

LIDONE has an antiemetic effect in animals. A similar effect may occur in humans and may obscure signs of intestinal obstruction or brain tumor.

ADVERSE REACTIONS

CNS Effects — The most frequently occurring effect is initial drowsiness that generally subsides with continued usage of the drug or lowering of the dose.

Noted less frequently were depression, hyperactivity and euphoria.

Neurological Extrapyramidal Reactions — Extrapyramidal reactions noted below may occur in susceptible individuals and are usually reversible with appropriate management.

Akathisia — Motor restlessness may occur early.

Parkinson Syndrome — Akinesia, characterized by rigidity, immobility and reduction of voluntary movements and tremor, have been observed. Occurrence is less frequent than akathisia.

Dystonic Syndrome — Prolonged abnormal contractions of muscle groups occur infrequently. These symptoms may be managed by the addition of a synthetic antiparkinson agent (other than L-dopa), small doses of sedative drugs, and/or reduction in dosage.

Autonomic Nervous System — Occasionally blurring of vision, tachycardia, nausea, dry mouth and salivation have been reported. Urinary retention and constipation may occur particularly if anticholinergic drugs are used to treat extrapyramidal symptoms.

Hematological — There have been rare reports of leucopenia and leucocytosis. If such reactions occur, treatment with LIDONE may continue if clinical symptoms are absent. Alterations of blood glucose, liver function tests, B.U.N., and red blood cells have not been considered clinically significant.

Metabolic and Endocrine Effects — Alteration of thyroid function has not been significant. Amenorrhea has been reported infrequently. Resumption of menses in previously amenorrheic women has been reported. Initially heavy menses may occur. Lactation associated with LIDONE therapy has not been reported. Increase in libido has been noted in some patients. Impotence has not been reported. Although both weight gain and weight loss have been in the direction of normal or ideal weight, excessive weight gain has not occurred with LIDONE.

Cardiovascular — Rare, transient, non-specific T wave changes have been reported on E.K.G. Association with a clinical syndrome has not been established. Rarely has significant hypotension been reported.

Ophthalmological — Lens opacities and pigmentary retinopathy have not been reported where patients have received LIDONE (molindone hydrochloride). In some patients, phenothiazine induced lenticular opacities have resolved following discontinuation of the phenothiazine while continuing therapy with LIDONE.

Skin — Early, non-specific skin rash, probably of allergic origin, has occasionally been reported. Skin pigmentation has not been seen with LIDONE usage alone.

LIDONE (molindone hydrochloride) has certain pharmacological similarities to other antipsychotic agents. Because adverse reactions are often extensions of the pharmacological activity of a drug, all of the known pharmacological effects associated with other antipsychotic drugs should be kept in mind when LIDONE is used. Upon abrupt withdrawal after prolonged high dosage an abstinence syndrome has not been noted.

Tardive Dyskinesia — Although rarely reported with LIDONE (molindone hydrochloride) symptoms were reversible upon discontinuation of therapy.

Tardive dyskinesia associated with other agents has appeared in some patients on long-term therapy and has also appeared after drug therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movement may be involuntary movements of extrer

There is no known effective treatment dyskinesia; antiparkinsonism agents u not alleviate the symptoms of this syndr suggested that all antipsychotic agents b tinued if these symptoms appear. Should essary to reinstitute treatment, or inc dosage of the agent, or switch to a diffe psychotic agent, the syndrome may be m has been reported that fine vermicular m of the tongue may be an early sign of the and if the medication is stopped at that syndrome may not develop.

DOSAGE AND ADMINISTRATION — I maintenance doses of LIDONE (molindor chloride) should be individualized.

Initial Dosage Schedule

The usual starting dosage is 50 to 75 mg
- Increase to 100 mg/day in three or f
- Based on severity of symptomatolog may be titrated up or down dependi dividual patient response.
- An increase to 225 mg/day may be re patients with severe symptomatolog
- Some chronic, treatment-resistant may require up to 400 mg/day; (how long-term safety of 400 mg/day has established).

Elderly or debilitated patients should be st lower dosage.

Maintenance Dosage Schedule

1. Mild — 5 mg to 15 mg three or four tim
2. Moderate — 10 mg to 25 mg three or fou day.
3. Severe — 225 mg/day may be required.

Dosage may be administered once a day.

DRUG INTERACTIONS — Potentiation administered concurrently with LIDONE done hydrochloride) has not been reporte tionally, animal studies have not shown i toxicity when LIDONE is given concurren representative members of three classes (i.e., barbiturates, chloral hydrate and ant son drugs).

MANAGEMENT OF OVERDOSAGE — matic, supportive therapy should be the r

Gastric lavage is indicated for the redu absorption of LIDONE (molindone hydroc which is freely soluble in water.

Since the adsorption of LIDONE (molinc drochloride) by activated charcoal has n determined, the use of this antidote must sidered of theoretical value.

Emesis in a comatose patient is contrain Additionally, while the emetic ef apomorphine is blocked by LIDONE in anim blocking effect has not been determi humans.

A significant increase in the rate of rer unmetabolized LIDONE from the body by diuresis, peritoneal or renal dialysis woul expected. (Only 2% of a single ingested LIDONE is excreted unmetabolized in the However, poor response of the patient may use of these procedures.

While the use of laxatives or enemas m based on general principles, the amount metabolized LIDONE in feces is less than trapyramidal symptoms have responded to of diphenhydramine (Benadryl*) and th thetic anticholinergic antiparkinson agen Artane*, Cogentin*, Akineton*).

HOW SUPPLIED — LIDONE (molindo drochloride) capsules are supplied in bottle in the following dosage strengths and colo

5 mg (**NDC** 0074-5542-13) blue and crear
10 mg (**NDC** 0074-5543-13) red and cream
25 mg (**NDC** 0074-5544-13) brown and cre

TM — Trademark
*Benadryl — Trademark, Parke Davis and Co.
*Artane — Trademark, Lederle Laboratories
*Cogentin — Trademark, Merck Sharp & Dohme
*Akineton — Trademark, Knoll Pharmaceutical C

ABBOTT LABORATORIE
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EBRUARY 1979

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Helps weather
the manic storm

maintenance therapy with Eskalith® 300 mg capsules and of lithium carbonate

In manic-depressive patients with a history of mania, 'Eskalith'

prevents or diminishes the intensity of future episodes

can increase the intervals between episodes

helps patients function within normal limits

Before prescribing, see complete prescribing information in SK&F literature or PDR. The following is a brief summary.

WARNING
Lithium toxicity is closely related to serum lithium levels, and can occur at doses close to therapeutic levels. Facilities for prompt and accurate serum lithium determinations should be available before initiating therapy.

Indications: Treatment of manic episodes of manic-depressive illness. Maintenance therapy prevents or diminishes the intensity of subsequent episodes in manic-depressive patients with a history of mania.

Warnings: Lithium should generally not be given to patients with significant renal or cardiovascular disease, severe debilitation or dehydration, sodium depletion, or to patients receiving diuretics.

Lithium therapy has been reported in some cases to be associated with morphologic changes in the kidneys.

Caution patient and family to watch for diarrhea, vomiting, tremor, mild ataxia, drowsiness, or muscular weakness as signs of lithium toxicity, and to discontinue therapy and contact a physician should they occur. Patients receiving combined therapy with lithium and an antipsychotic should be monitored closely for early evidence of neurologic toxicity and treatment discontinued promptly if such signs appear. Caution patients about activities requiring alertness (e.g., operating vehicles or machinery).

Lithium should not be used in pregnancy, especially during the first trimester, unless potential benefits outweigh possible hazards.

Not recommended for children under 12.

Precautions: Lithium tolerance is greater during the acute manic phase and decreases when manic symptoms subside.

Lithium therapy may lead to sodium depletion. Normal diet (including salt) and adequate fluid intake (2500-3000 ml.) must be maintained, at least during initial stabilization period. Protracted sweating or diarrhea can decrease tolerance; in such cases, administer supplemental fluid and salt.

Sweating, diarrhea, and concomitant infection with elevated temperatures may require temporary reduction or cessation of dosage.

Adverse Reactions: Mild to moderate toxic reactions may occur at serum lithium levels from 1.5 to 2.5 mEq./l., and moderate to severe reactions at levels from 2.0 to 2.5 mEq./l. Fine hand tremor, polyuria, and mild thirst may occur during initial therapy and persist. Transient and mild nausea and general discomfort also appear during initial therapy. These effects usually subside with continued treatment or temporary reduction or cessation of dosage. If persistent, discontinue dosage.

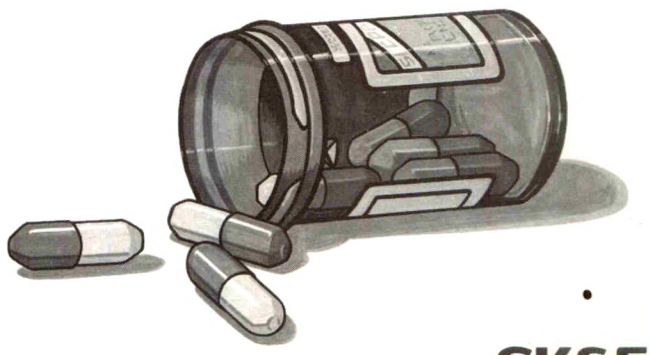
Diarrhea, vomiting, drowsiness, muscular weakness, and lack of coordination may be early signs of toxicity and may occur at levels below 2.0 mEq./l. At higher levels, ataxia, giddiness, tinnitus, blurred vision, and a large output of dilute urine may be seen. Serum levels above 3.0 mEq./l. may produce a complex clinical picture, involving multiple organs and systems. Serum levels should not exceed 2.0 mEq./l. during acute phase.

The following reactions appear to be related to serum lithium levels, including levels within the therapeutic range: **Neuromuscular**—tremor, muscle hyperirritability (fasciculations, twitching, clonic movements of whole limbs), ataxia, choreo-athetotic movements, hyperactive deep tendon reflex; **Central Nervous System**—blackout spells, epileptiform seizures, slurred speech, dizziness, vertigo, incontinence of urine or feces, somnolence, psychomotor retardation, restlessness, confusion, stupor, coma; **Cardiovascular**—cardiac arrhythmia, hypotension, peripheral circulatory collapse; **Gastrointestinal**—anorexia, nausea, vomiting, diarrhea; **Genitourinary**—albuminuria, oliguria, polyuria, glycosuria; **Dermatologic**—drying and thinning of hair, alopecia, anesthesia of skin, chronic folliculitis, exacerbation of psoriasis, xerosis cutis; **Autonomic**—blurred vision, dry mouth; **Thyroid Abnormalities**—euthyroid goiter and/or hypothyroidism (including myxedema) with lower T_3 and T_4. I^{131} uptake may be elevated; **EEG Changes**—diffuse slowing, widening of the frequency spectrum, potentiation and disorganization of background rhythm; **EKG Changes**—reversible flattening, isoelectricity or inversion of T-waves; **Miscellaneous**—fatigue, lethargy, transient scotomata, dehydration, weight loss, tendency to sleep.

Reactions unrelated to dosage include: transient EEG and EKG changes, leukocytosis, headache, diffuse nontoxic goiter with or without hypothyroidism, transient hyperglycemia, generalized pruritus with or without rash, cutaneous ulcers, albuminuria, worsening of organic brain syndromes, excessive weight gain, edematous swelling of ankles or wrists, thirst or polyuria, sometimes resembling diabetes insipidus, and metallic taste. A single case of a syndrome resembling Raynaud's has been reported.

How Supplied: 300 mg. capsules in bottles of 100.

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Philadelphia, PA

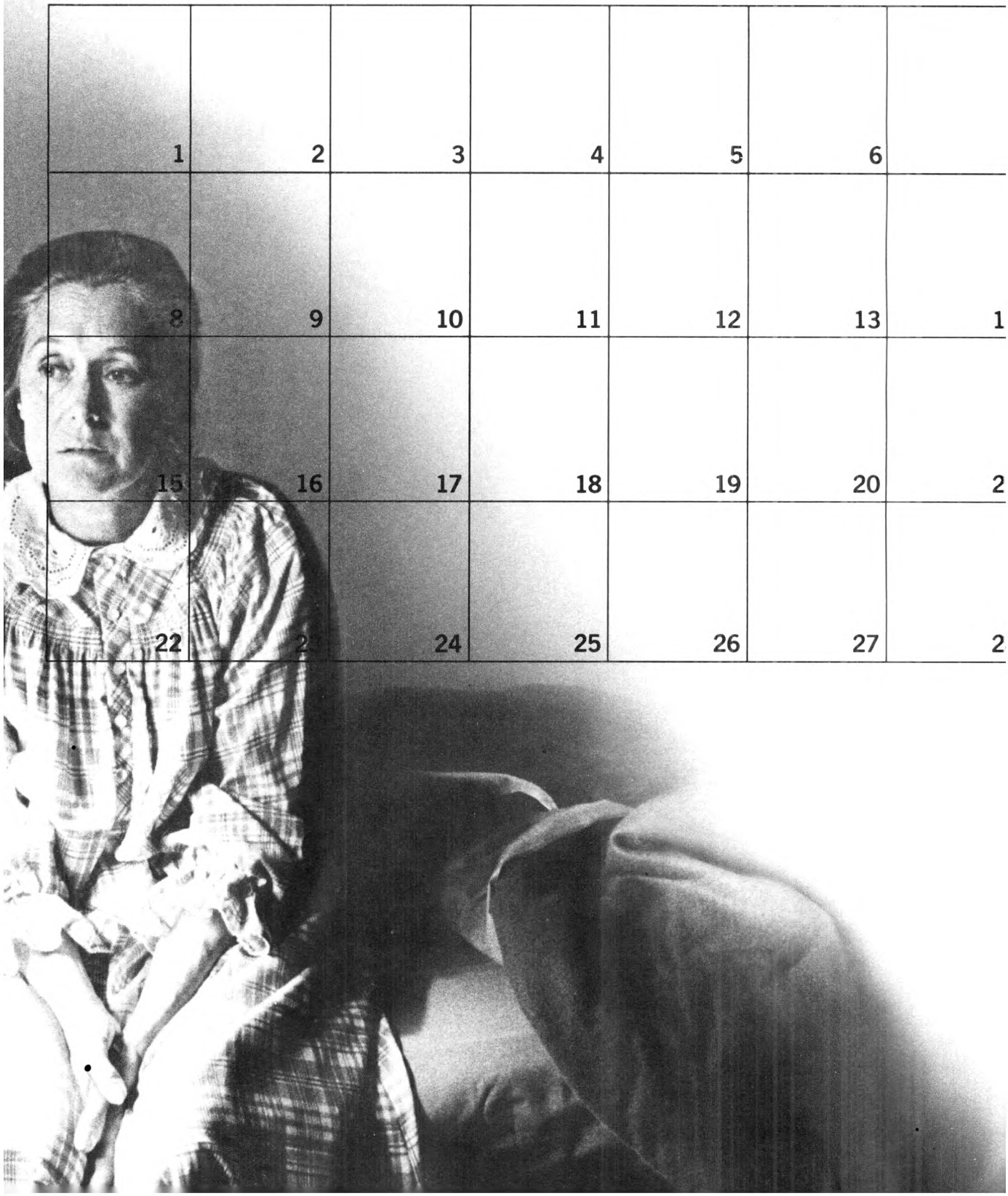


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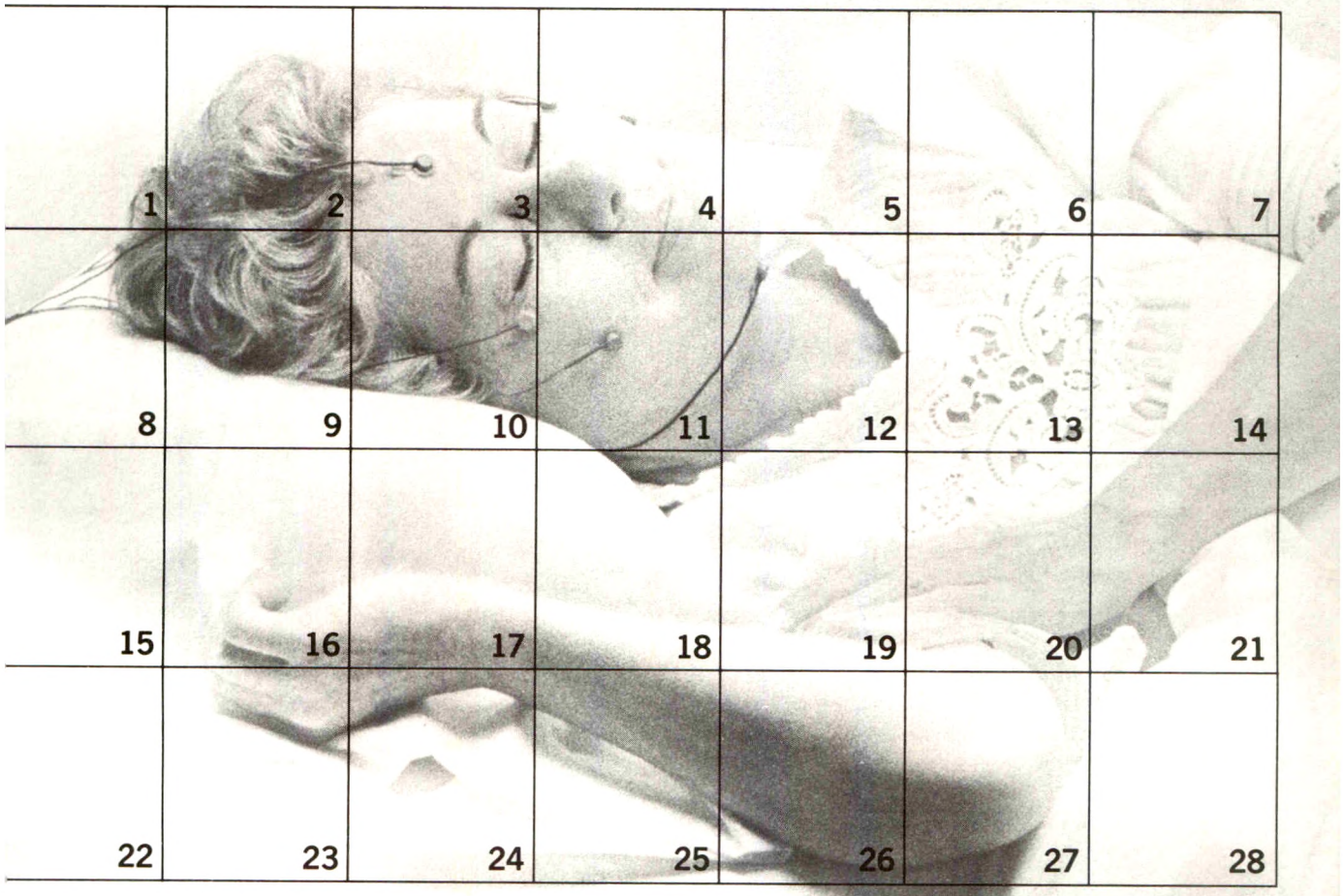
INSOMNIA

that continues for up to two weeks outlasts most hypnotics.



ONLY **DALMANE**® (flurazepam HCl) C-IV

›rovides significant efficacy for chronic insomniacs from the first through 28 consecutive nights of investigation.



ective investigations prove the continuing efficacy of mane 30 mg *h.s.* during a 4-week medication period.[1-3] ontrast, a recently published sleep laboratory study orted the absence of a statistically significant hypnotic ct for chloral hydrate (1 Gm), ethchlorvynol (500 mg),* ethimide (500 mg), methaqualone (250 and 400 mg) secobarbital (100 mg) at the end of two weeks of inistration.[4] Another study demonstrated that total e time in insomniacs receiving pentobarbital (100 mg) rned to premedication baseline levels within 14 nights.[2] ecause insomnia is often transient, the prolonged inistration of Dalmane is seldom necessary. If therapy st be extended, periodic blood counts and liver and ney function tests should be performed.

her recently published study showed that ethchlorvynol (500 mg) did esult in a statistically significant reduction of sleep latency, minutes ake time after sleep onset, number of nighttime awakenings and unt of wakefulness at medication nights 1-3 or at medication ts 12-14.[5]

Less risk of accidental or purposeful overdosage with Dalmane (flurazepam HCl)

"Flurazepam has a higher therapeutic index (lethal: hypnotic dose) than the other sedative-hypnotics; thus, there is less risk of accidental overdosage or potential use as a suicide agent."[6]

As with all medications of its type, Dalmane should be administered with caution to severely depressed patients or those with any evidence of latent depression. Protective measures should be taken for patients with suicidal tendencies.

Please see following page for a summary of product information.

ROCHE

FOR AN UNSURPASSED RECORD OF EFFECTIVENESS AND SAFETY

DALMANE®
(flurazepam HCl) C IV

30-MG AND 15-MG CAPSULES

One 30-mg capsule *h.s.*— usual adult dosage
(15 mg may suffice in some patients).
One 15-mg capsule *h.s.*— recommended dosage for elderly or debilitated patients.

Before prescribing Dalmane (flurazepam HCl), please consult complete product information, a summary of which follows:
Indications: Effective in all types of insomnia characterized by difficulty in falling asleep, frequent nocturnal awakenings and/or early morning awakening; in patients with recurring insomnia or poor sleeping habits; in acute or chronic medical situations requiring restful sleep. Objective sleep laboratory data have shown effectiveness for at least 28 consecutive nights of administration. Since insomnia is often transient and intermittent, prolonged administration is generally not necessary or recommended.
Contraindications: Known hypersensitivity to flurazepam HCl.
Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).

Usage in Pregnancy: **Several studies of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) suggest increased risk of congenital malformations during the first trimester of pregnancy. Dalmane, a benzodiazepine, has not been studied adequately to determine whether it may be associated with such an increased risk. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage.
Precautions: In elderly and debilitated, limit dosage to 15 mg to reduce risk of oversedation, dizziness, confusion and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in patients who are severely depressed, or with latent depression or suicidal tendencies, or with impaired renal or hepatic function. Periodic blood counts and liver and kidney function tests are advised during repeated therapy.
Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity, and elevated SGOT, SGPT, total and direct bilirubins and alkaline phosphatase.
Dosage: Individualize for maximum beneficial effect.
Adults: 30 mg usual dosage; 15 mg may suffice in some patients.
Elderly or debilitated patients: 15 mg initially until response is determined.
Supplied: Capsules containing 15 mg or 30 mg flurazepam HCl.

REFERENCES:
1. Kales A, *et al: Clin Pharmacol Ther 19*:576-583, May 1976
2. Kales A, *et al: Clin Pharmacol Ther 18*:356-363, Sep 1975
3. Dement WC, Guilleminault C, Zarcone V: Progress in Clinical Sleep Research. Scientific exhibit at the American Medical Association, Atlantic City NJ, Jun 14-18, 1975
4. Kales A, *et al: J Clin Pharmacol 17*:207-213, Apr 1977
5. Kripke DF, Lavie P, Hernandez J: *Psychopharmacology* 56:221-223, 1978
6. *AMA Drug Evaluations*, Ed 3. Littleton Mass, Publishing Sciences Group, Inc., 1977, p. XXXIII

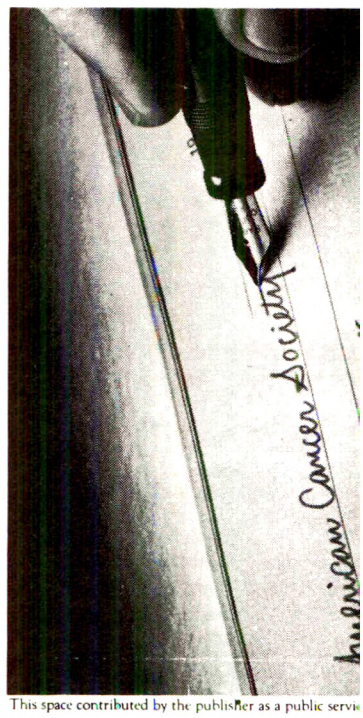
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in from research laboratories all over the world.
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CAPSULES,
SINGLE DOSE TABLETS

Brief Summary

ACTIONS — Pharmacologically, clorazepate dipotassium has the characteristics of the benzodiazepines. It has depressant effects on the central nervous system. The primary metabolite, nordiazepam, quickly appears in the blood stream. The serum half-life is about 2 days. The drug is metabolized in the liver and excreted primarily in the urine. (See CLINICAL PHARMACOLOGY section.)

INDICATIONS — TRANXENE is indicated for the symptomatic relief of anxiety associated with anxiety neurosis, in other psychoneuroses in which anxiety symptoms are prominent features, and as an adjunct in disease states in which anxiety is manifested.

TRANXENE is indicated for the symptomatic relief of acute alcohol withdrawal.

The effectiveness of TRANXENE in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should reassess periodically the usefulness of the drug for the individual patient.

CONTRAINDICATIONS — TRANXENE is contraindicated in patients with a known hypersensitivity to the drug, and in those with acute narrow angle glaucoma.

WARNINGS — TRANXENE is not recommended for use in depressive neuroses or in psychotic reactions.

Patients on TRANXENE should be cautioned against engaging in hazardous occupations requiring mental alertness, such as operating dangerous machinery including motor vehicles.

Since TRANXENE has a central nervous system depressant effect, patients should be advised against the simultaneous use of other CNS-depressant drugs, and cautioned that the effects of alcohol may be increased.

Because of the lack of sufficient clinical experience, TRANXENE is not recommended for use in patients less than 18 years of age.

Physical and Psychological Dependence:

Withdrawal symptoms (similar in character to those noted with barbiturates and alcohol) have occurred following abrupt discontinuance of clorazepate. Symptoms of nervousness, insomnia, irritability, diarrhea, muscle aches and memory impairment have followed abrupt withdrawal after long-term use of high dosage.

Caution should be observed in patients who are considered to have a psychological potential for drug dependence.

Evidence of drug dependence has been observed in dogs and rabbits which was characterized by convulsive seizures when the drug was abruptly withdrawn or the dose was reduced; the syndrome in dogs could be abolished by administration of clorazepate.

Usage in Pregnancy:

An increased risk of congenital malformations associated with the use of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) during the first trimester of pregnancy has been suggested in several studies. TRANXENE, a benzodiazepine derivative, has not been studied adequately to determine whether it, too, may be associated with an increased risk of fetal abnormality. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physician about the desirability of discontinuing the drug.

Usage during Lactation:

TRANXENE should not be given to nursing mothers since it has been reported that nordiazepam is excreted in human breast milk.

PRECAUTIONS — In those patients in which a degree of depression accompanies the anxiety, suicidal tendencies may be present and protective measures may be required. The least amount of drug that is feasible should be available to the patient.

Patients on TRANXENE for prolonged periods should have blood counts and liver function tests periodically. The usual precautions in treating patients with impaired renal or hepatic function should also be observed.

In elderly or debilitated patients, the initial dose should be small, and increments should be made gradually, in accordance with the response of the patient, to preclude ataxia or excessive sedation.

ADVERSE REACTIONS — The side effect most frequently reported was drowsiness. Less commonly reported (in descending order of occurrence) were: dizziness, various gastrointestinal complaints, nervousness, blurred vision, dry mouth, headache, and mental confusion. Other side effects included insomnia, transient skin rashes, fatigue, ataxia, genitourinary complaints, irritability, diplopia, depression and slurred speech.

There have been reports of abnormal liver and kidney function tests and of decrease in hematocrit.

Decrease in systolic blood pressure has been observed.

DOSAGE AND ADMINISTRATION

For the symptomatic relief of anxiety:

TRANXENE is administered orally. The capsules may be given in divided doses. The usual daily dose is 30 mg. The dose should be adjusted gradually within the range of 15 to 60 mg. daily in accordance with the response of the patient. In elderly or debilitated patients it is advisable to initiate treatment at a daily dose of 7.5 to 15 mg.

TRANXENE capsules may also be administered as a single dose daily at bedtime; the recommended initial dose is 15 mg. After the initial dose, the response of the patient may require adjustment of subsequent dosage. Lower doses may be indicated in the elderly patient. Drowsiness may occur at the initiation of treatment and with dosage increment.

TRANXENE-SD tablets (22.5 mg.) may be administered as a single dose every 24 hours. This tablet is intended as an alternate dosage form for the convenience of patients stabilized on a dose of 7.5 mg. capsules three times a day. TRANXENE-SD tablets should not be used to initiate therapy.

TRANXENE-SD HALF STRENGTH tablets (11.25 mg.) may be administered as a single dose every 24 hours.

For the symptomatic relief of acute alcohol withdrawal:

Recommended schedule: 1st 24 hours, 30 mg. TRANXENE initially, followed by 30 to 60 mg. in divided doses; 2nd 24 hours, 45 to 90 mg. in divided doses; 3rd 24 hours, 22.5 to 45 mg. in divided doses; 4th day, 15 to 30 mg. in divided doses. Thereafter gradually reduce to 7.5 to 15 mg. daily, and discontinue as soon as condition is stable. Maximum daily dose is 90 mg. Avoid excessive reductions in total drug on successive days.

DRUG INTERACTIONS — If TRANXENE is to be combined with other drugs acting on the central nervous system, careful consideration should be given to the pharmacology of the agents to be employed. Animal experience indicates that TRANXENE prolongs the sleeping time after hexobarbital or after ethyl alcohol, increases the inhibitory effects of chlorpromazine, but does not exhibit monoamine oxidase inhibition. Clinical studies have shown increased sedation with concurrent hypnotic medications. The actions of the benzodiazepines may be potentiated by barbiturates, narcotics, phenothiazines, monoamine oxidase inhibitors or other antidepressants.

If TRANXENE is used to treat anxiety associated with somatic disease states, careful attention must be paid to possible drug interaction with concomitant medication.

MANAGEMENT OF OVERDOSAGE — Overdosage is usually manifested by varying degrees of CNS depression ranging from slight sedation to coma. As in the management of overdosage with any drug, it should be borne in mind that multiple agents may have been taken.

There are no specific antidotes for the benzodiazepines. The treatment of overdosage should consist of the general measures employed in the management of overdosage of any CNS depressant. Gastric evacuation either by the induction of emesis, lavage, or both, should be performed immediately. General supportive care, including frequent monitoring of the vital signs and close observation of the patient, is indicated. Hypotension, though rarely reported, may occur with large overdoses. In such cases the use of agents such as Levophed® (levarterenol) or Aramine® (metaraminol) should be considered.

While reports indicate that individuals have survived overdoses of TRANXENE (clorazepate dipotassium) as high as 450 to 675 mg., these doses are not necessarily an accurate indication of the amount of drug absorbed since the time interval between ingestion and the institution of treatment was not always known. Sedation in varying degrees was the most common physiological manifestation of TRANXENE overdosage. Deep coma when it occurred was usually associated with the ingestion of other drugs in addition to TRANXENE.

CLINICAL PHARMACOLOGY — Studies in healthy men have shown that TRANXENE has depressant effects on the central nervous system. Prolonged administration of single daily doses as high as 120 mg. was without toxic effects. Abrupt cessation of high doses was followed in some patients by nervousness, insomnia, irritability, diarrhea, muscle aches, or memory impairment.

Absorption — Excretion:

After oral administration of TRANXENE, there is essentially no circulating parent drug. Nordiazepam, its primary metabolite, quickly appears in the blood stream. In 2 volunteers given 15 mg. (50 μC) of ^{14}C-TRANXENE, about 80% was recovered in the urine and feces within 10 days. Excretion was primarily in the urine with about 1% excreted per day on day 10.

HOW SUPPLIED — TRANXENE (clorazepate dipotassium) is supplied as capsules in three dosage strengths:

3.75 mg. capsules (gray with white cap) in bottles of 100 (NDC 0074-3417-13) and 500 (NDC 0074-3417-53). Also available in ABBO-PAC® unit dose packages of 100 capsules (NDC 0074-3417-11).

7.5 mg. capsules (gray with maroon cap) in bottles of 30 (NDC 0074-3418-30), 100 (NDC 0074-3418-13) and 500 (NDC 0074-3418-53). Also available in ABBO-PAC unit dose packages of 100 capsules (NDC 0074-3418-11).

15 mg. capsules (all gray) in bottles of 100 (NDC 0074-3419-13) and 500 (NDC 0074-3419-53). Also available in ABBO-PAC unit dose packages of 100 capsules (NDC 0074-3419-11).

TRANXENE-SD single dose tablets are supplied in two dosage strengths:

TRANXENE-SD 22.5 mg. tablets (tan-colored) in bottles of 100 (NDC 0074-2997-13).

TRANXENE-SD HALF STRENGTH 11.25 mg. tablets (blue-colored) in bottles of 30 (NDC 0074-2699-30) and 100 (NDC 0074-2699-13).

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*Of course patients should be cautioned against hazardous tasks requiring mental alertness.

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See overleaf for prescribing information.

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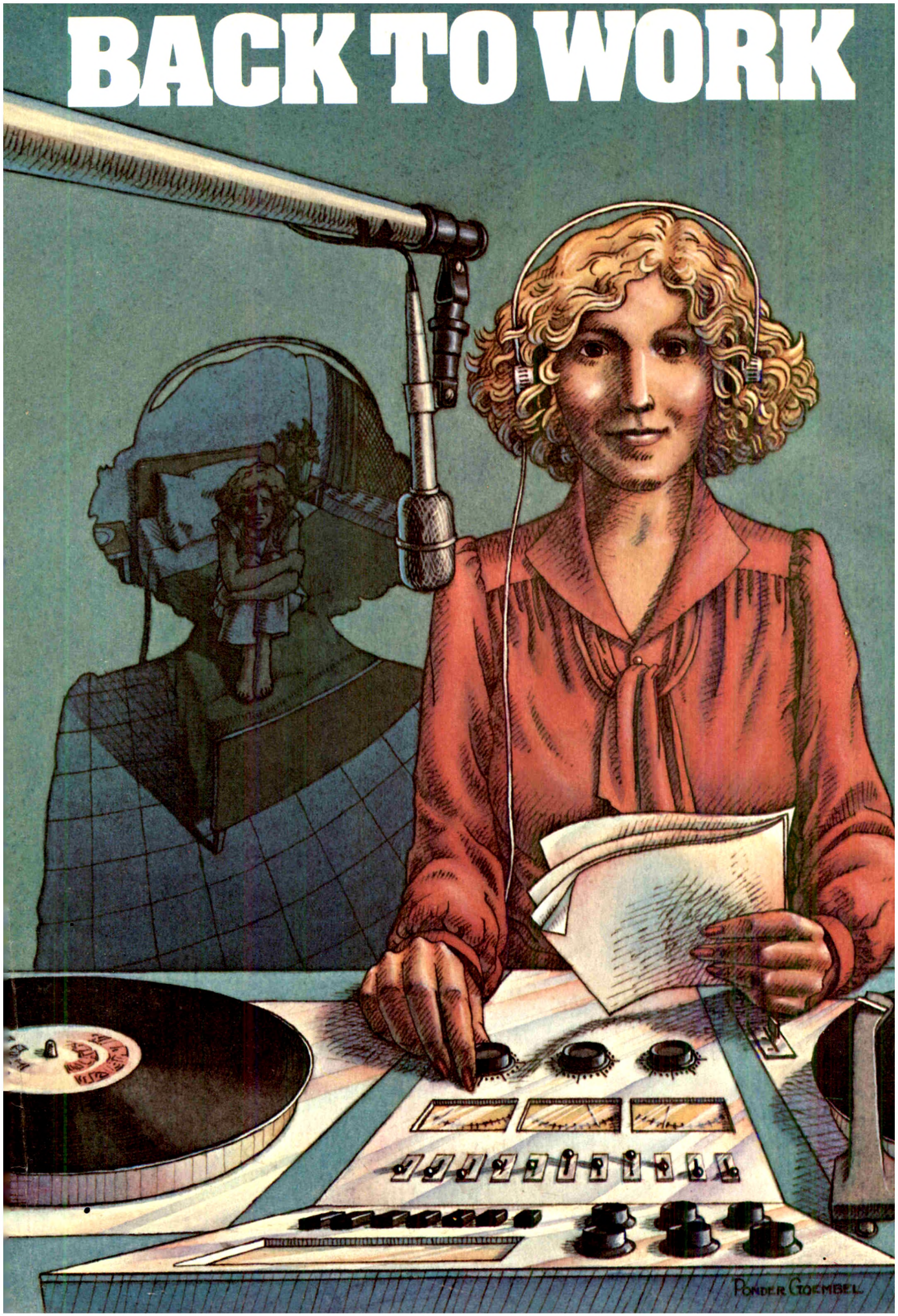
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Overview: Efficacy and Safety of the Rapid Neuroleptization Method with Injectable Haloperidol

By Patrick T. Donlon, John Hopkin, and Joe P. Tupin

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efore prescribing, see complete prescribing nformation in SK&F literature or PDR. The llowing is a brief summary.

Indications

Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

Effective: For the management of manifestations of psychotic disorders. For control of the manifestations of manic-depressive illness (manic phase).

Probably effective: For the control of moderate to severe agitation, hyperactivity or aggressiveness in disturbed children.

Possibly effective: For control of excessive anxiety, tension and agitation as seen in neuroses.

Final classification of the less-than-effective indications requires further investigation.

ontraindications: Comatose states, presence of rge amounts of C.N.S. depressants, or bone arrow depression.

Warnings: The possibility of extrapyramidal eactions from 'Thorazine' may confuse the agnosis of Reye's syndrome or other encephapathy. Therefore, avoid use in children or olescents with suspected Reye's syndrome.

oid using in patients hypersensitive (e.g., blood scrasia, jaundice) to any phenothiazine. Caution ients about activities requiring alertness (e.g., rating vehicles or machinery) especially during first few days' therapy. Avoid concomitant with alcohol. May counteract antihypertenseffect of guanethidine and related compounds.

n pregnancy only when essential. There are ted instances of jaundice or prolonged extraidal signs in newborn whose mothers had ed chlorpromazine.

tions: Use cautiously in persons with vascular, liver or chronic respiratory , or with acute respiratory infections. Due h reflex suppression, aspiration of vomitus ble. May prolong or intensify the action depressants, organophosphorus insecticat, atropine and related drugs. (Reduce f concomitant C.N.S. depressants.) Anti-

convulsant action of barbiturates is not intensified. Antiemetic effect may mask signs of toxic drug overdosage or obscure diagnosis of conditions such as intestinal obstruction, brain tumor, and Reye's syndrome (see Warnings). Discontinue high-dose, long-term therapy gradually.

Patients on long-term therapy, especially high doses, should be evaluated periodically for possible adjustment or discontinuance of drug therapy.

Adverse Reactions: Drowsiness, cholestatic jaundice, agranulocytosis, eosinophilia, leukopenia, hemolytic anemia, thrombocytopenic purpura and pancytopenia; postural hypotension, tachycardia, fainting, dizziness and, occasionally, a shock-like condition; reversal of epinephrine effects; EKG changes have been reported, but relationship to myocardial damage is not confirmed; neuromuscular (extrapyramidal) reactions; pseudoparkinsonism, motor restlessness, dystonias, persistent tardive dyskinesia, hyperreflexia in the newborn; psychotic symptoms, catatonic-like states, cerebral edema; convulsive seizures; abnormality of the cerebrospinal fluid proteins; urticarial reactions and photosensitivity, exfoliative dermatitis, contact dermatitis; lactation and breast engorgement (in females on large doses), false positive pregnancy tests, amenorrhea, gynecomastia; hyperglycemia, hypoglycemia, glycosuria; dry mouth, nasal congestion, constipation, adynamic ileus, urinary retention, miosis, mydriasis; after prolonged substantial doses, skin pigmentation, epithelial keratopathy, lenticular and corneal deposits and pigmentary retinopathy, visual impairment; mild fever (after large I.M. dosage); hyperpyrexia; increased appetite and weight; a systemic lupus erythematosus-like syndrome; peripheral edema.

NOTE: Sudden death in patients taking phenothiazines (apparently due to cardiac arrest or asphyxia due to failure of cough reflex) has been reported, but no causal relationship has been established.

Supplied: Tablets, 10 mg., 25 mg., 50 mg., 100 mg. and 200 mg., in bottles of 100; Single Unit Packages of 100 (intended for institutional use only).

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This program, supported by NIMH funding, has provided partial stipend assistance and support for participation in APA meetings. The objectives of the program are to increase the number of minority group psychiatrists, to provide educational enrichment and stimulate interest in pursuing training in those areas in which minority groups are especially underrepresented.

Applications** are being accepted through March 15, 1979, for residencies beginning in July 1979. Applicants must be US citizens and must have graduated from a U.S. medical school or possess the ECFMG certificate.

Information is available from:

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Office of Minority Affairs
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1700 18th Street, N.W.
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This section contains very brief articles (maximum of 1,000 words, 10 references, 1 table, and *no figures*) reporting new research findings, including preliminary data from pilot studies, and case reports that 1) describe new syndromes, 2) cast a new light on established ones, 3) indicate a new therapeutic procedure of potential value, or 4) describe adverse effects or previously unreported complications of drugs or therapeutic interventions. Because of the stringent criteria for this section, authors can expect more rapid publication than is possible in other sections. Submitted papers that substantially exceed the stated maximum length or contain figures will be returned to the author unreviewed.

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1. Berne E: Principles of Group Treatment. New York, Oxf University Press, 1966, p 26

2. Blackwell B, Marley E, Price J, et al: Hypertensive interactions between monoamine oxidase inhibitors and foodstuffs. Br J Psychiatry 113:349–365, 1967
3. Gold PW, Goodwin FK: Urinary free cortisol in depression and mania. Presented at the 130th annual meeting of the American Psychiatric Association, Toronto, Ont, Canada, May 2–6, 1977
4. Brosin H: Communication systems of the consultation process, in The Psychiatric Consultation. Edited by Mendel W, Solomon P. New York, Grune & Stratton, 1968

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The April 1979 issue of

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Navane (thiothixene) rapidly controls hallucinations as well as the agitation and hostility patterns they frequently generate.

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(thiothixene hydrochloride) Concentrate: 5 mg/ml,
Intramuscular: 2 mg/ml

Contraindications. Navane is contraindicated in patients with circulatory collapse, comatose states, central nervous system depression due to any cause, and blood dyscrasias. Navane is contraindicated in individuals who have shown hypersensitivity to the drug. It is not known whether there is a cross-sensitivity between the thioxanthenes and the phenothiazine derivatives, but this possibility should be considered.

Warnings. *Usage in Pregnancy*—Safe use of Navane during pregnancy has not been established. Therefore, this drug should be given to pregnant patients only when, in the judgment of the physician, the expected benefits from the treatment exceed the possible risks to mother and fetus. Animal reproduction studies and clinical experience to date have not demonstrated any teratogenic effects.

In the animal reproduction studies with Navane, there was some decrease in conception rate and litter size, and an increase in resorption rate in rats and rabbits, changes which have been similarly reported with other psychotropic agents. After repeated oral administration to rats (5 to 15 mg/kg/day), rabbits (3 to 50 mg/kg/day), and monkeys (1 to 3 mg/kg/day) before and during gestation, no teratogenic effects were seen. (See Precautions.)

Usage in Children—The use of Navane in children under 12 years of age is not recommended because safety and efficacy in the pediatric age group have not been established.

As is true with many CNS drugs, Navane may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery, especially during the first few days of therapy. Therefore, the patient should be cautioned accordingly.

As in the case of other CNS-acting drugs, patients receiving Navane should be cautioned about the possible additive effects (which may include hypotension) with CNS depressants and with alcohol.

Precautions. An antiemetic effect was observed in animal studies with Navane; since this effect may also occur in man, it is possible that Navane may mask signs of overdosage of toxic drugs and may obscure conditions such as intestinal obstruction and brain tumor.

In consideration of the known capability of Navane and certain other psychotropic drugs to precipitate convulsions, extreme caution should be used in patients with a history of convulsive disorders or those in a state of alcohol withdrawal since it may lower the convulsive threshold. Although Navane potentiates the actions of the barbiturates, the dosage of the anticonvulsant therapy should not be reduced when Navane is administered concurrently.

Caution as well as careful adjustment of the dosage is indicated when Navane is used in conjunction with other CNS depressants other than anticonvulsant drugs.

Though exhibiting rather weak anticholinergic properties, Navane should be used with caution in patients who are known or suspected to have glaucoma, or who might be exposed to extreme heat, or who are receiving atropine or related drugs.

Use with caution in patients with cardiovascular disease.

Also, careful observation should be made for pigmentary retinopathy, and lenticular pigmentation (fine lenticular pigmentation has been noted in a small number of patients treated with Navane for prolonged periods). Blood dyscrasias (agranulocytosis, pancytopenia, thrombocytopenic purpura), and liver damage (jaundice, biliary stasis) have been reported with related drugs.

Undue exposure to sunlight should be avoided. Photosensitive reactions have been reported in patients on Navane.

Intramuscular Administration—As with all intramuscular preparations, Navane Intramuscular should be injected well within the body of a relatively large muscle. The preferred sites are the upper outer quadrant of the buttock (i.e., gluteus maximus) and the mid-lateral thigh.

The deltoid area should be used only if well developed, such as in certain adults and older children, and then only with caution to avoid radial nerve injury. Intramuscular injections should not be made into the lower and mid-thirds of the upper arm. As with all intramuscular injections, aspiration is necessary to help avoid inadvertent injection into a blood vessel.

Adverse Reactions. *Note:* Not all of the following adverse reactions have been reported with Navane (thiothixene). However, since Navane has certain chemical and pharmacologic similarities to the phenothiazines, all of the known side effects and toxicity associated with phenothiazine therapy should be borne in mind when Navane is used.

Cardiovascular effects: Tachycardia, hypotension, lightheadedness, and syncope. In the event hypotension occurs, epinephrine should not be used as a pressor agent since a paradoxical further lowering of blood pressure may result. Nonspecific EKG changes have been observed in some patients receiving Navane. These changes are usually reversible and frequently disappear on continued Navane therapy. The incidence of these changes is lower than that observed with some phenothiazines. The clinical significance of these changes is not known.

CNS effects: Drowsiness, usually mild, may occur although it usually subsides with continuation of Navane therapy. The incidence of sedation appears similar to that of the piperazine group of phenothiazines, but less than that of certain aliphatic phenothiazines. Restlessness, agitation and insomnia have been noted with Navane (thiothixene). Seizures and paradoxical exacerbation of psychotic symptoms have occurred with Navane infrequently.

Hyperreflexia has been reported in infants delivered from mothers having received structurally related drugs.

In addition, phenothiazine derivatives have been associated with cerebral edema and cerebrospinal fluid abnormalities.

Extrapyramidal symptoms, such as pseudo-parkinsonism, akathisia, and dystonia have been reported. Management of these extrapyramidal symptoms depends upon the type and severity. Rapid relief of acute symptoms may require the use of an injectable antiparkinson agent. More slowly emerging symptoms may be managed by reducing the dosage of Navane and/or administering an oral antiparkinson agent.

Persistent Tardive Dyskinesia: As with all antipsychotic agents tardive dyskinesia may appear in some patients on long term therapy or may occur after drug therapy has been discontinued. The risk seems to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities.

There is no known effective treatment for tardive dyskinesia: antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear.

Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked.

It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time, the syndrome may not develop.

Hepatic effects: Elevations of serum transaminase and alkaline phosphatase, usually transient, have been infrequently observed in some patients. No clinically confirmed cases of jaundice attributable to Navane have been reported.

Hematologic effects: As is true with certain other psychotropic drugs, leukopenia and leukocytosis, which are usually transient, can occur occasionally with Navane. Other antipsychotic drugs have been associated with agranulocytosis, eosinophilia, hemolytic anemia, thrombocytopenia and pancytopenia.

Allergic reactions: Rash, pruritus, urticaria, photosensitivity and rare cases of anaphylaxis have been reported with Navane. Undue exposure to sunlight should be avoided. Although not experienced with Navane, exfoliative dermatitis and contact dermatitis (in nursing personnel) have been reported with certain phenothiazines.

Endocrine disorders: Lactation, moderate breast enlargement and amenorrhea have occurred in a small percentage of females receiving Navane. If persistent, this may necessitate a reduction in dosage or the discontinuation of therapy. Phenothiazines have been associated with false positive pregnancy tests, gynecomastia, hypoglycemia, hyperglycemia, and glycosuria.

Autonomic effects: Dry mouth, blurred vision, nasal congestion, constipation, increased sweating, increased salivation, and impotence have occurred infrequently with Navane therapy. Phenothiazines have been associated with miosis, mydriasis, and adynamic ileus.

Other adverse reactions: Hyperpyrexia, anorexia, nausea, vomiting, diarrhea, increase in appetite and weight, weakness or fatigue, polydipsia and peripheral edema.

Although not reported with Navane, evidence indicates there is a relationship between phenothiazine therapy and the occurrence of a systemic lupus erythematosus-like syndrome.

NOTE: Sudden deaths have occasionally been reported in patients who have received certain phenothiazine derivatives. In some cases the cause of death was apparently cardiac arrest or asphyxia due to failure of the cough reflex. In others, the cause could not be determined nor could it be established that death was due to phenothiazine administration.

Dosage and Administration. Dosage of Navane should be individually adjusted depending on the chronicity and severity of the condition. In general, small doses should be used initially and gradually increased to the optimal effective level, based on patient response.

Some patients have been successfully maintained on once-a-day Navane therapy.

Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

Navane Intramuscular Solution—For Intramuscular Use Only. Where more rapid control and treatment of acute behavior is desirable, the intramuscular form of Navane may be indicated. It is also of benefit where the very nature of the patient's symptomatology, whether acute or chronic, renders oral administration impractical or even impossible.

For treatment of acute symptomatology or in patients unable or unwilling to take oral medication, the usual dose is 4 mg of Navane Intramuscular administered 2 to 4 times daily. Dosage may be increased or decreased depending on response. Most patients are controlled on a total daily dosage of 16 to 20 mg. The maximum recommended dosage is 30 mg/day. An oral form should supplant the injectable form as soon as possible. It may be necessary to adjust the dosage when changing from the intramuscular to oral dosage. Dosage recommendations for Navane Capsules and Concentrate appear in the following paragraphs.

Navane Capsules; Navane Concentrate—In milder conditions, an initial dose of 2 mg three times daily. If indicated, subsequent increase to 15 mg/day total daily dose is usually effective.

In more severe conditions, an initial dose of 5 mg two to four times daily.

The usual optimal dose is 20 to 30 mg daily. If indicated, increase to 60 mg/day total daily dose is often effective. Exceeding a total daily dose of 60 mg rarely increases beneficial response.

Overdosage. Manifestations include muscular twitching, drowsiness, and dizziness. Symptoms of gross overdosage include CNS depression, rigidity, weakness, torticollis, tremor, salivation, dysphagia, hypotension, disturbances of gait, and coma.

Treatment: Essentially symptomatic and supportive. For Navane oral, early gastric lavage is helpful. For Navane oral or Intramuscular, keep patient under careful observation and maintain an open airway, since involvement of the extrapyramidal system may produce dysphagia and respiratory difficulty in severe overdosage. If hypotension occurs, standard measures for managing circulatory shock should be used (I.V. fluids and/or vasoconstrictors).

If a vasoconstrictor is needed, levarterenol and phenylephrine are the most suitable drugs. Other pressor agents, including epinephrine, are not recommended, since phenothiazine derivatives may reverse the usual pressor effect of these agents and cause further lowering of blood pressure.

If CNS depression is present, recommended stimulants include amphetamine, dextroamphetamine, or caffeine and sodium benzoate. Picrotoxin or pentylenetetrazol should be avoided. Extrapyramidal symptoms may be treated with antiparkinson drugs.

There are no data on the use of peritoneal or hemodialysis, but they are known to be of little value in phenothiazine intoxication.

How Supplied. Navane (thiothixene) is available as capsules containing 1 mg, 2 mg, 5 mg, and 10 mg of thiothixene in bottles of 100 and 1,000. Navane is also available as capsules containing 20 mg of thiothixene in bottles of 100 and 500.

Navane (thiothixene hydrochloride) Concentrate is available in 120 ml (4 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, 5 mg, 6 mg, 8 mg, and 10 mg, and in 30 ml (1 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, and 5 mg. Each ml contains thiothixene hydrochloride equivalent to 5 mg thiothixene. Contains alcohol, U.S.P. 7.0% v/v (small unavoidable).

Navane (thiothixene hydrochloride) Intramuscular solution is available in a 2 ml amber glass vial in packages of 10. Each ml contains thiothixene hydrochloride equivalent to 2 mg thiothixene, dextrose 5% w/v, benzyl alcohol 0.9% w/v, propyl gallate 0.02% w/v.

References. 1. Stotsky BA: Relative efficacy of parenteral haloperidol and thiothixene for the emergency treatment of acutely excited and agitated patients. *Dis Nerv Syst* 38:967-1977. 2. Bressler B, Friedel RO: A comparison between chlorpromazine and thiothixene in a Veterans Administration hospital population. *Psychosomatics* 12:275-277, 1971. 3. De HCB, Turns D: Double blind comparison of thiothixene, trifluoperazine in acute schizophrenia. *Psychosomatics* 13:104, 1972. 4. Sterlin C, Ban TA, Lehman HE, Saxena BM: place of thiothixene in the treatment of schizophrenic pat *Can Psychiatr Assoc J* 15:3-4, 1970. 5. Brauzer B, Goldstein Comparative effects of intramuscular thiothixene and fluoperazine in psychotic patients. *J Clin Pharmacol* 8:400-1968. 6. Engelhardt DM, Rudorfer L: The chronic schizophrenic outpatient in an urban community: Social and vocational adjustment. Presented as a Scientific Exhibit at the 1 Annual Meeting, American Psychiatric Association, Toronto, Canada, May 2-6, 1977. 7. Itil TM, Unverdi C, Wohlral Larsen V, Levitt J: Drug therapy of psychosis associated with organic brain syndrome, presented as a scientific exhibit at American Public Health Association Centennial, Atlantic New Jersey, November 12-16, 1972. 8. Dillenkoffer RL, Electrocardiographic evaluations of schizophrenic patients. Presented as a Scientific Exhibit at the 125th Annual Meeting the American Psychiatric Association, Dallas, Texas, May 1972. 9. Goldstein B, Weiner D, Banas F: Clinical evaluation thiothixene in chronic ambulatory schizophrenic patients. Lehmann HE, Ban TA (eds): *Modern Problems in Pharmacopsychiatry*. Basel, S Karger, 1969, vol 2, pp 45-52.

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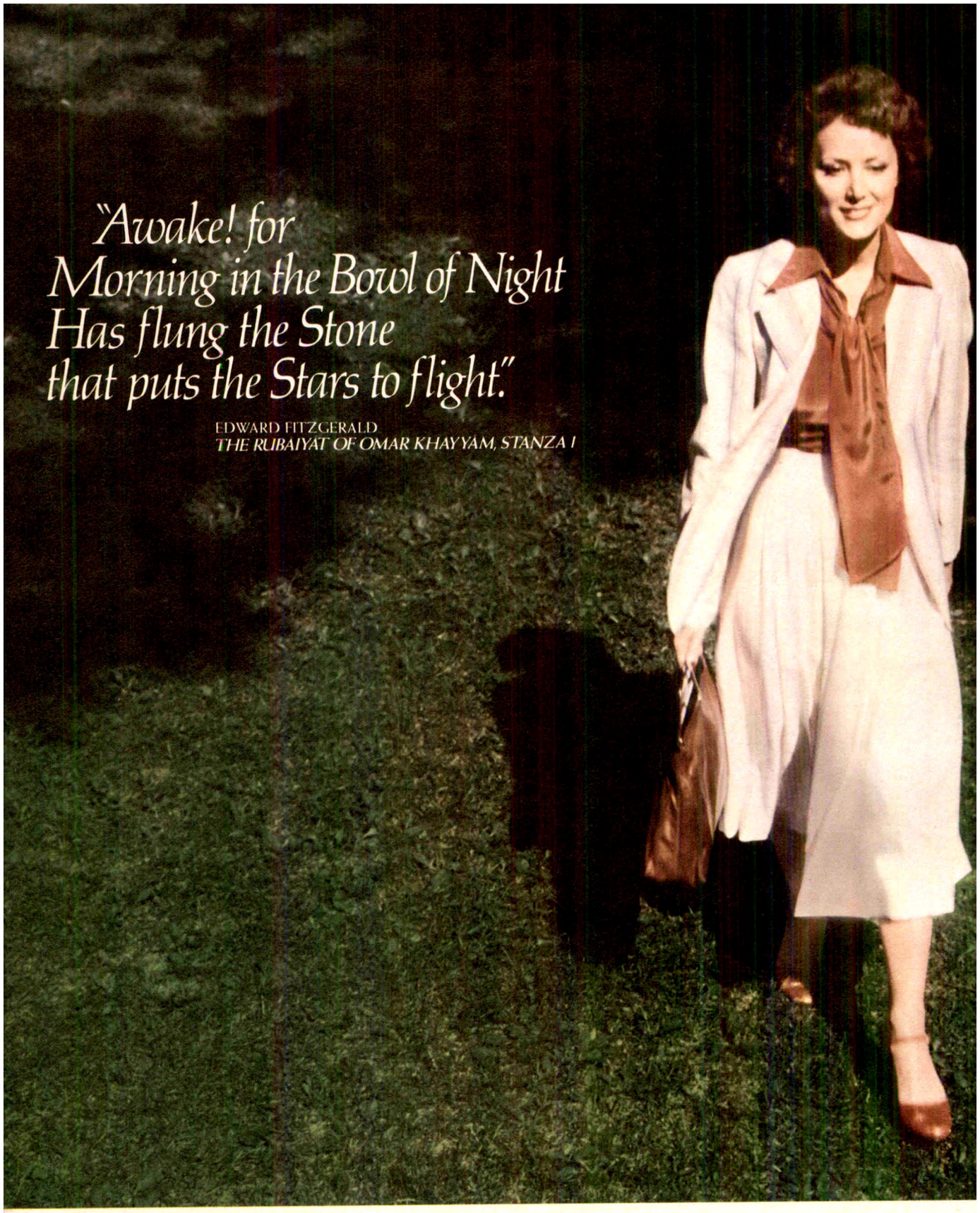
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Brief Summary of Prescribing Information

ADAPIN® (doxepin HCl) Capsules

Indications—Relief of symptoms of anxiety and depression.

Contraindications—Glaucoma, tendency toward urinary retention, or hypersensitivity to doxepin.

Warnings—Adapin has not been evaluated for safety in pregnancy. No evidence of harm to the animal fetus has been shown in reproductive studies. There are no data concerning secretion in human milk, nor on effect in nursing infants.

Usage in children under 12 years of age is not recommended. MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with this drug, as serious side-effects and death have been reported with the concomitant use of certain drugs and MAO inhibitors.

In patients who may use alcohol excessively potentiation may increase the danger inherent in any suicide attempt or overdosage.

Precautions—Drowsiness may occur and patients should be cautioned against driving a motor vehicle or operating hazardous machinery. Since suicide is an inherent risk in depressed patients they should be closely supervised while receiving treatment. Although Adapin has shown effective tranquilizing activity, the possibility of activating or unmasking latent psychotic symptoms should be kept in mind.

Adverse Reactions—Dry mouth, blurred vision and constipation have been reported. Drowsiness has also been observed.

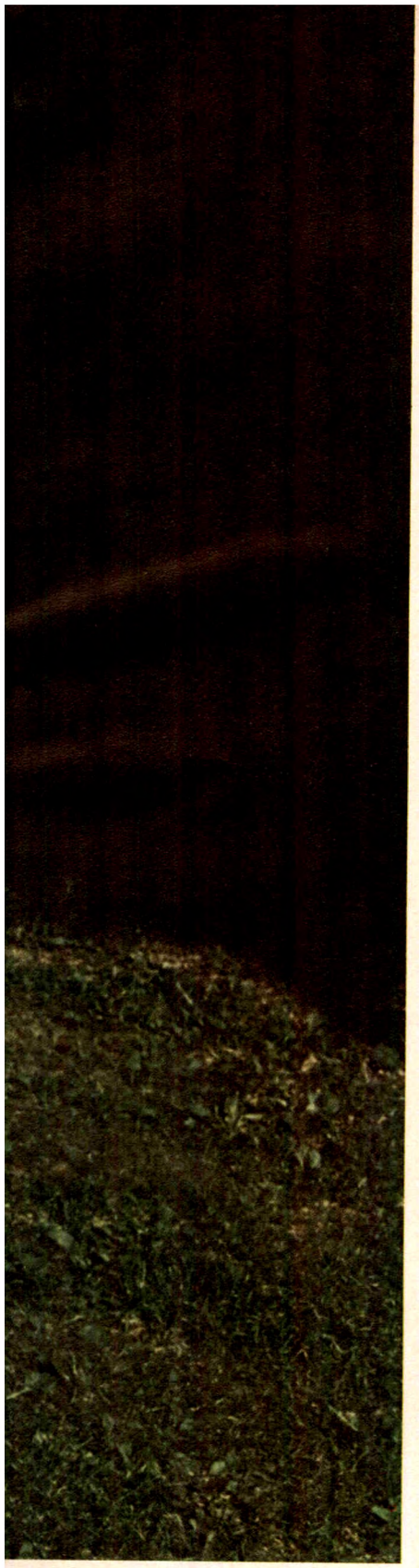
Adverse effects occurring infrequently include extrapyramidal symptoms, gastrointestinal reactions, secretory effects such as sweating, tachycardia and hypotension. Weakness, dizziness, fatigue, weight gain, edema, paresthesias, flushing, chills, tinnitus, photophobia, decreased libido, rash and pruritus may als occur.

Dosage and Administration—In mild to moderate anxiety and/ depression: 25 mg t.i.d. Increase or decrease the dosage accordi to individual response. Daily dosage, up to 150 mg may be taken bedtime without loss of effectiveness. Usual optimum daily dosa is 75 mg to 150 mg per day not to exceed 300 mg per day.

Antianxiety effect usually precedes the antidepressant effect two or three weeks.

How Supplied—Each capsule contains doxepin, as the h drochloride: **10 mg, 25 mg, 50 mg, and 100 mg** capsules in bottl of 100 and 1000.

For complete prescribing information please see package inse or PDR.



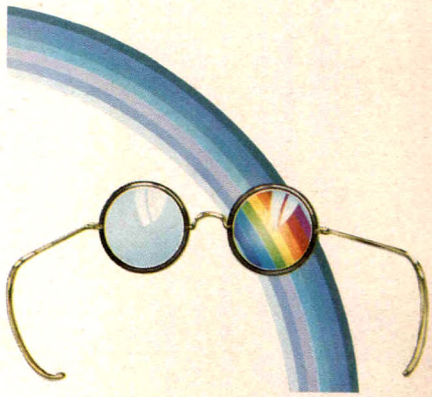
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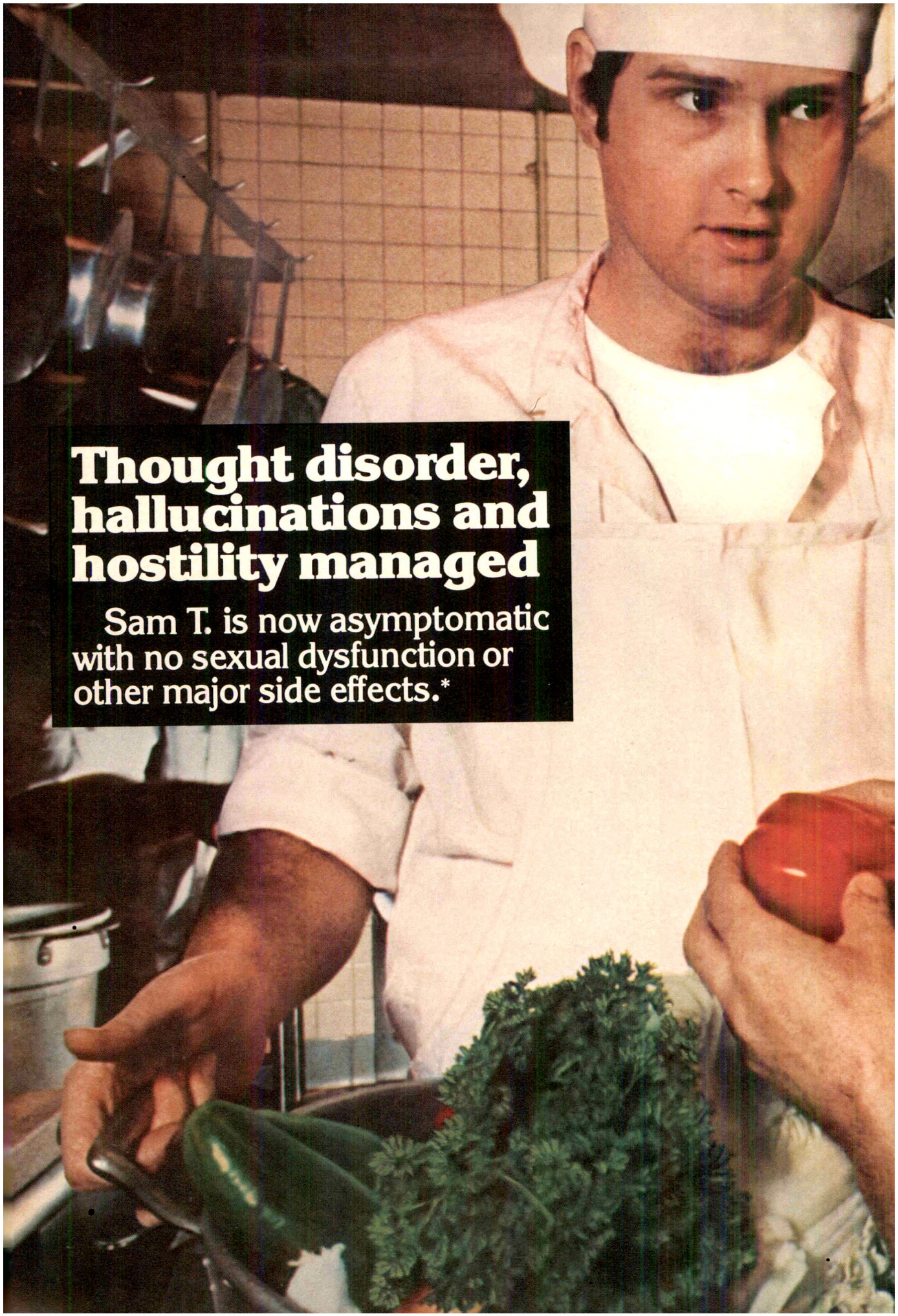
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Thought disorder, hallucinations and hostility managed
Sam T. is now asymptomatic with no sexual dysfunction or other major side effects.*



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Permits rapid management of Sam's hostility and excited behavior

Thirty-six year old salad maker, claiming to be the "Wild Man of the West" was brought to the hospital by his kitchen supervisor. History revealed prior psychiatric hospitalizations and therapies including psychotropic drugs. Hypertension was noted as a complication of earlier treatment with anti-psychotic drugs. Diagnosis: chronic schizophrenia, acute exacerbation. He responded promptly to LOXITANE® C Loxapine HCl Concentrate 50 mg stat, followed by LOXITANE® Loxapine Succinate capsules 30 mg t.i.d.

Permits aggressive titration for rapid establishment of optimal management of Sam's thought disorder and hallucinations

During first week, LOXITANE dosage increased to 50 mg q.i.d. Significant improvement in thought disorder noted by third week. Auditory hallucinations and delusions much less frequent. Patient calmer and more cooperative.

Permits sustained symptom management with minimal risk of serious adverse effects, such as sexual dysfunction

Patient revealed that while on outpatient status, he failed to take his previously prescribed antipsychotic for a ten-month period after he met and married his girlfriend. He stated the drug "didn't let me be a man." No sexual dysfunction reported with LOXITANE.

Side effects are usually mild, transitory and easily managed

Patient felt anxious because his "heart was beating so fast," and he "could not sit still." Increased pulse rate and restlessness subsided following decrease of LOXITANE to 20 mg q.i.d.

Consistence in schizophrenic symptom management and tolerance promotes patient compliance

All admitting symptomatology under control by the fourth week. By the sixth week, he was anxious to return home. Stated "this medicine doesn't make me feel drowsy like the other did." Discharged by the eighth week. He returned to work fully managed on LOXITANE 20 mg t.i.d., and free from recurrence of previously reported side effects.

*Painful ejaculation and urine containing sperm have not been reported. Endocrine disturbances such as galactorrhea and menstrual irregularities have only been rarely reported. Other sexual side effects infrequently reported. Although not reported to date, possibility of hepatic, renal, ocular, or phototoxicity cannot be ruled out at this time. Transient liver enzyme changes not definitely related to LOXITANE have been reported. Not an actual patient, but a simulation to illustrate the action of LOXITANE as reported in clinical studies.

See LOXITANE prescribing information on following page for indications, warnings and precautions and for more detailed information concerning side effects.

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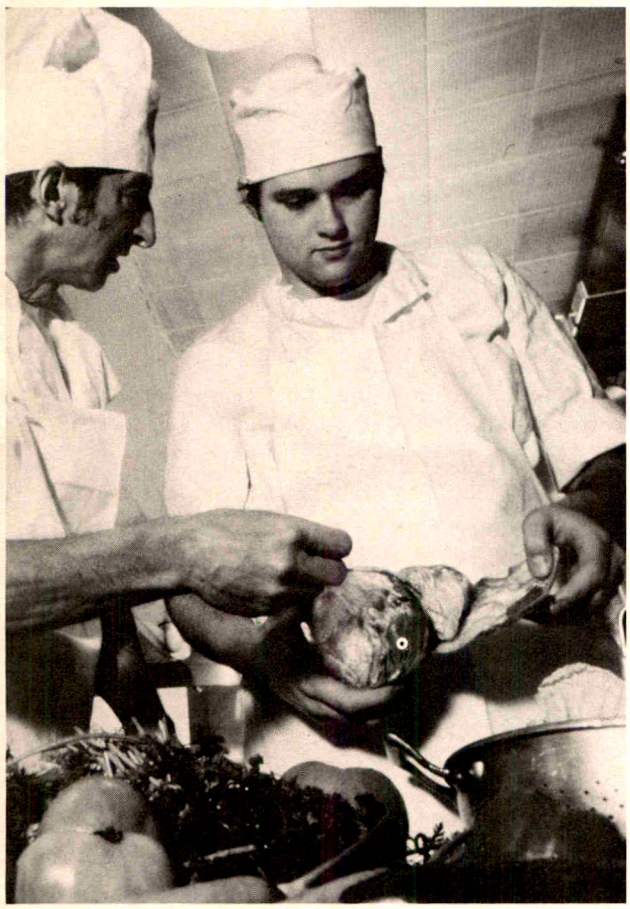
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Capsules:
5 mg, 10 mg,
25 mg, 50 mg

Maximum management... minimum risk

Maximum management... minimum risk



Loxitane®
LOXAPINE SUCCINATE
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Recommended Daily Dosage

	MILD	MODERATE	SEVERE
Initial Dosage	10 mg b.i.d.	10 mg t.i.d or q.i.d.	25 mg b.i.d.
First 7 to 10 days	Increase dosage until psychotic symptoms are controlled. Dosage should not exceed 250 mg/day. Usual dosage during titration: 50 to 150 mg/day.		
Maintenance Dosage	Adjust to lowest effective level. Usual maintenance dosage: 60 to 100 mg/day. Many patients are controlled with dosages as low as 20 to 60 mg/day.		

Brief Summary
LOXITANE® Loxapine Succinate Capsules
LOXITANE® C Loxapine Hydrochloride Oral Concentrate
INDICATIONS: Manifestations of schizophrenia.
CONTRAINDICATIONS: Comatose or severely depressed states; hypersensitivity to the drug.
WARNINGS: Safe use during pregnancy or lactation has not been established; weigh potential benefits to possible hazards. Not recommended for use in children under 16. May impair mental and/or physical abilities especially during early therapy; warn ambulatory patients about activities requiring alertness and concomitant use of alcohol or other CNS depressants. Not recommended for management of behavioral complications in mentally retarded patients.
PRECAUTIONS: Use with extreme caution in patients with a history of convulsive disorders; use with caution in patients with cardiovascular disease or in those with glaucoma or a tendency to urinary retention particularly when on concomitant anticholinergic medication. Loxapine has an antiemetic effect in animals which might occur in man masking signs of overdosage of toxic drugs and obscuring intestinal obstruction or brain tumor. Since possible ocular toxicity cannot be excluded, observe carefully for pigmentary retinopathy and lenticular pigmentation.
ADVERSE REACTIONS: CNS effects, other than extrapyramidal, infrequent. Mild drowsiness may occur at beginning of therapy or upon dosage increase, usually subsides with continued therapy. Sedation, dizziness, faintness, staggering gait, muscle twitching, weakness and confusional states have been reported. Extrapyramidal reactions often occur early in treatment manifested by Parkinson-like symptoms (tremor, rigidity, excessive salivation, masked facies, akathisia) controllable by dosage reduction or antiparkinson drugs at usual dosages. Dystonic and dyskinetic reactions, while less frequently occurring, may be more severe, requiring dosage reduction or temporary withdrawal plus appropriate counteractive drugs. Persistent Tardive Dyskinesia may appear during prolonged therapy or following discontinuance, the risk greater in the elderly, especially females, on high dosage. Symptoms, persistent and in some patients apparently irreversible, are characterized by rhythmical involuntary movement of the tongue, face, mouth and jaw sometimes accompanied by involuntary movement of extremities. Since there is no known effective treatment, discontinue all antipsychotic drugs if symptoms appear. Reinstitution of treatment, increased dosage, or switching to another agent may mask syndrome. The syndrome may not develop if medication is stopped when fine vermicular movements of the tongue first appear. Cardiovascular Effects: Tachycardia, hypotension, hypertension, lightheadedness and syncope. ECG changes, not known to be related to loxapine use, have been reported. Skin: Dermatitis, edema of face, pruritus, seborrhea. Possible photosensitivity and/or phototoxicity; skin rashes of unknown etiology seen in a few patients in hot summer months. Anticholinergic: Dry mouth, nasal congestion, constipation, blurred vision (more likely to occur with concomitant use of antiparkinson agents). Other: Nausea, vomiting, weight gain or loss, dyspnea, ptosis, hyperpyrexia, flushed facies, headache, paresthesia, polydipsia. Rarely, galactorrhea and menstrual irregularity of unknown etiology.

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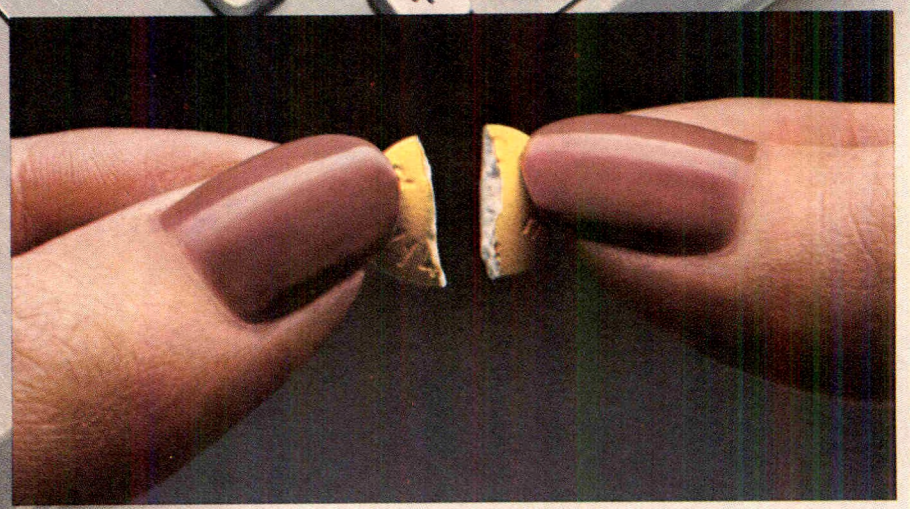
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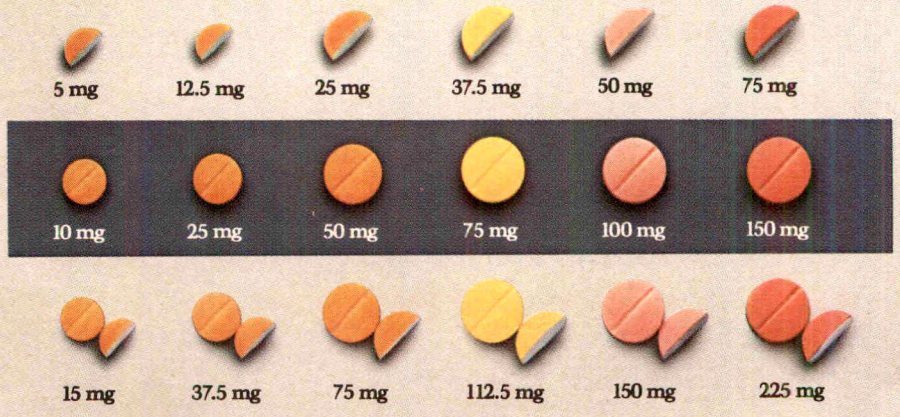
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Warnings: May block action of guanethidine or similar antihypertensives. Use with caution in patients with history of seizures, urinary retention, angle-closure glaucoma, increased intraocular pressure. Closely supervise cardiovascular patients, hyperthyroid patients and those receiving thyroid medications. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, including amitriptyline HCl, especially in high doses. Myocardial infarction and stroke reported with use of this class of drugs.) May impair alertness; warn against hazardous occupations or driving a motor vehicle during therapy. Weigh possible benefits against hazards during pregnancy, the nursing period and in women of childbearing potential. Not recommended in children under 12.

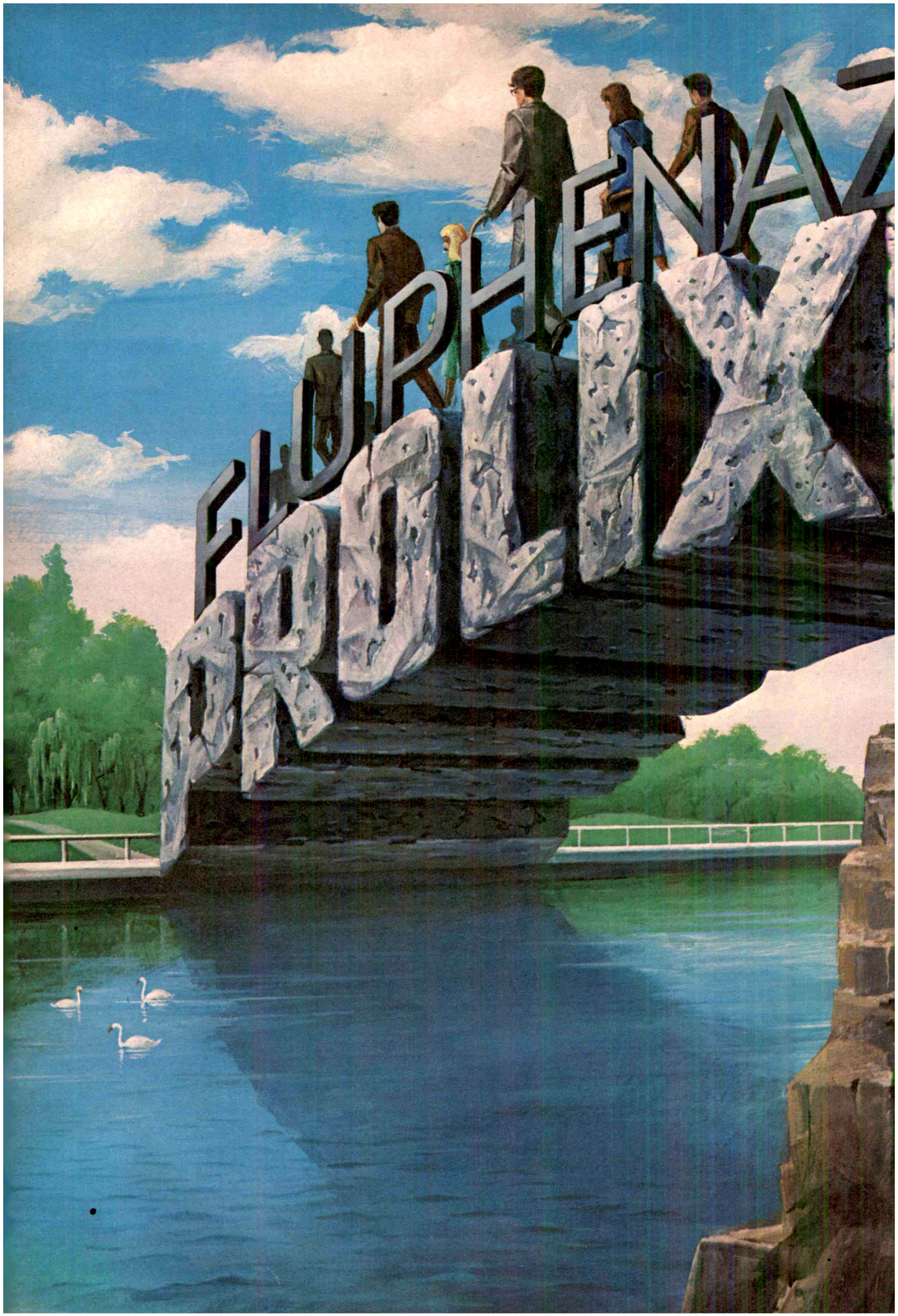
Precautions: May exaggerate symptoms in schizophrenic and paranoid patients, or shift manic-depressives to manic stage; reduce dose or administer major tranquilizer concomitantly. Close supervision and careful dose adjustments required when given with anticholinergic or sympathomimetic agents. Exercise care in patients receiving large doses of ethchlorvynol; transient delirium reported with concomitant administration. May enhance effects of alcohol, barbiturates and other CNS depressants. Because of the possibility of suicide in depressed patients, do not permit easy access to large drug quantities in these patients. Because it may increase hazards of electroshock therapy, limit concomitant use to essential treatment. If possible, discontinue drug several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported.

Adverse Reactions: Note: This list includes a few adverse reactions not reported with this specific drug but requiring consideration because of similarities of tricyclic antidepressants. *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, constipation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, black tongue. *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, jaundice, alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache and malaise. These are not indicative of addiction.

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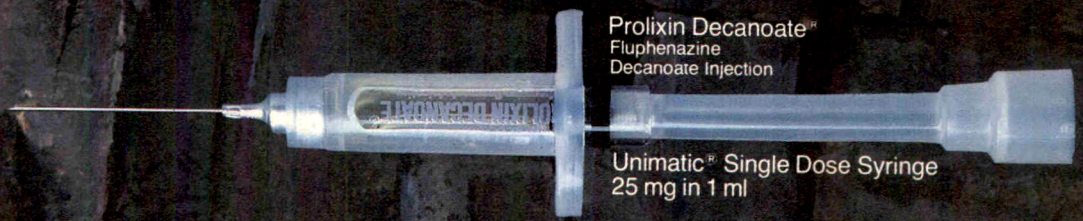
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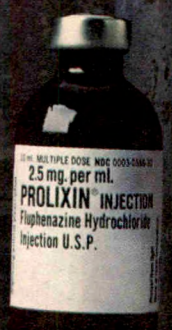
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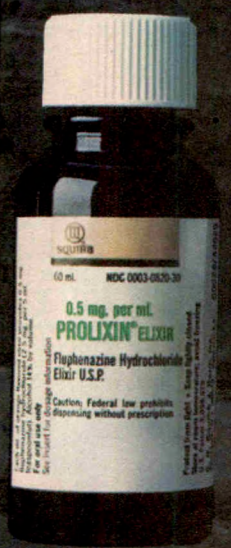
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See next page for brief summaries.

PROLIXIN® (Fluphenazine Hydrochloride)
TABLETS/ELIXIR/INJECTION

Prolixin Tablets (Fluphenazine Hydrochloride Tablets USP) provide 1, 2.5, 5, or 10 mg fluphenazine hydrochloride per tablet. Prolixin Elixir (Fluphenazine Hydrochloride Elixir USP) provides 0.5 mg fluphenazine hydrochloride per ml (2.5 mg per 5 ml teaspoonful) with 14% alcohol by volume. Prolixin Injection (Fluphenazine Hydrochloride Injection USP) provides 2.5 mg fluphenazine hydrochloride per ml; it contains 0.1% methylparaben and 0.01% propylparaben as preservatives.

CONTRAINDICATIONS: In presence of suspected or established subcortical brain damage. In patients who have a blood dyscrasia or liver damage, or who are receiving large doses of hypnotics, or who are comatose or severely depressed. In patients who have shown hypersensitivity to fluphenazine; cross-sensitivity to phenothiazine derivatives may occur.

WARNINGS: Mental and physical abilities required for driving a car or operating heavy machinery may be impaired by use of this drug. Potentiation of effects of alcohol may occur. Safety and efficacy in children have not been established because of inadequate experience in use in children.

Usage in Pregnancy: Safety for use during pregnancy has not been established; weigh possible hazards against potential benefits if administering this drug to pregnant patients.

PRECAUTIONS: Caution must be exercised if another phenothiazine compound caused cholestatic jaundice, dermatoses or other allergic reactions because of the possibility of cross-sensitivity. When psychotic patients on large doses of a phenothiazine drug are to undergo surgery, hypotensive phenomena should be watched for; less anesthetics or central nervous system depressants may be required. Because of added anticholinergic effects, fluphenazine may potentiate the effects of atropine.

Use fluphenazine cautiously in patients exposed to extreme heat or phosphorus insecticides; in patients with a history of convulsive disorders since grand mal convulsions have occurred; and in patients with special medical disorders such as mitral insufficiency or other cardiovascular diseases, and pheochromocytoma. Bear in mind that with prolonged therapy there is the possibility of liver damage, pigmentary retinopathy, lenticular and corneal deposits, and development of irreversible dyskinesia.

There is sufficient experimental evidence to conclude that chronic administration of antipsychotic drugs which increase prolactin secretion has the potential to induce mammary neoplasms in rodents under the appropriate conditions. There are recognized differences in the physiological role of prolactin between rodents and humans. Since there are, at present, no adequate epidemiological studies, the relevance to human mammary cancer risk from prolonged exposure to fluphenazine hydrochloride and other antipsychotic drugs is not known.

Periodic checking of hepatic and renal functions and blood picture should be done. Monitor renal function of patients on long-term therapy; if BUN becomes abnormal, discontinue fluphenazine. "Silent pneumonias" are possible.

Abrupt Withdrawal: In general, phenothiazines do not produce psychic dependence. However, gastritis, nausea and vomiting, dizziness, and tremulousness have been reported following abrupt cessation of high dose therapy; reports suggest that these symptoms can be reduced if concomitant antiparkinsonian agents are continued for several weeks after the phenothiazine is withdrawn.

ADVERSE REACTIONS: *Central Nervous System*—Extrapyramidal symptoms are most frequently reported. Most often these symptoms are reversible, but they may be persistent. They include pseudoparkinsonism, dystonia, dyskinesia, akathisia, oculogyric crises, opisthotonos, hyperreflexia. The incidence and severity of such reactions will depend more on individual patient sensitivity, but dosage level and patient age are also determinants. As these reactions may be alarming, the patient should be forewarned and reassured. These reactions can usually be controlled by administration of an anti-parkinsonian drug such as benztropine mesylate and by subsequent reduction in dosage.

Persistent Tardive Dyskinesia: As with all antipsychotic agents, persistent and sometimes irreversible tardive dyskinesia may appear in some patients on long-term therapy or may occur after discontinuation of drug. The risk seems greater in elderly patients, especially females, on high dosages. The syndrome is characterized by rhythmical involuntary movements of tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and may be accompanied by involuntary movements of extremities. There is no known effective therapy for tardive dyskinesia; usually the symptoms are not alleviated by antiparkinsonism agents. If the symptoms appear, discontinuation of all antipsychotic agents is suggested. The syndrome may be masked if treatment is reinstituted, or drug dosage increased, or a different antipsychotic agent used. Reports are that fine vermicular movements of the tongue may be an early sign of the syndrome which may not develop if medication is stopped at that time.

Phenothiazine derivatives have been known to cause restlessness, excitement, or bizarre dreams; reactivation or aggravation of psychotic processes may be encountered. If drowsiness or lethargy occur, the dosage may need to be reduced. Dosages, far in excess of the recommended amounts, may induce a catatonic-like state.

Autonomic Nervous System—Hypertension and fluctuations in blood pressure have been reported. Although hypotension is rarely a problem, patients with pheochromocytoma, cerebral vascular or renal insufficiency or severe cardiac reserve deficiency such as mitral insufficiency appear to be particularly prone to this reaction and should be observed carefully. Supportive measures including intravenous vasopressor drugs should be instituted immediately should severe hypotension occur; Levarterenol Bitartrate Injection is the most suitable drug; *epinephrine should not be used* since phenothiazine derivatives have been found to reverse its action. Nausea, loss of appetite, salivation, polyuria, perspiration, dry mouth, headache and constipation may occur. Reducing or temporarily discontinuing the dosage will usually control these effects. Blurred vision, glaucoma, bladder paralysis, fecal impaction, paralytic ileus, tachycardia, or nasal congestion have occurred in some patients on phenothiazine derivatives.

Metabolic and Endocrine—Weight change, peripheral edema, abnormal lactation, gynecomastia, menstrual irregularities, false results on pregnancy tests, impotency in men and increased libido in women have occurred in some patients on phenothiazine therapy.

Allergic Reactions—Itching, erythema, urticaria, seborrhea, photosensitivity, eczema and exfoliative dermatitis have been reported with phenothiazines. The possibility of anaphylactoid reactions should be borne in mind.

Hematologic—Blood dyscrasias including leukopenia, agranulocytosis, thrombocytopenic or nonthrombocytopenic purpura, eosinophilia, and pancytopenia have been observed with phenothiazines. If soreness of the mouth, gums or throat or any symptoms of upper respiratory infection occur and confirmatory leukocyte count indicates cellular depression, therapy should be discontinued and other appropriate measures instituted immediately.

Hepatic—Liver damage manifested by cholestatic jaundice, particularly during the first months of therapy, may occur; treatment should be discontinued. A cephalin flocculation increase, sometimes accompanied by alterations in other liver function tests, has been reported in patients who have had no clinical evidence of liver damage.

Others—Sudden deaths have been reported in hospitalized patients on phenothiazines. Previous brain damage or seizures may be predisposing factors. High doses should be avoided in known seizure patients. Shortly before death, several patients showed flare-ups of psychotic behavior patterns. Autopsy findings have usually revealed acute fulminating pneumonia or pneumonitis, aspiration of gastric contents, or intramyocardial lesions. Although not a general feature of fluphenazine, potentiation of central nervous system depressants such as opiates, analgesics, antihistamines, barbiturates, and alcohol may occur.

Systemic lupus erythematosus-like syndrome, hypotension severe enough to cause fatal cardiac arrest, altered electrocardiographic and electroencephalographic tracings, altered cerebrospinal fluid proteins, cerebral edema, asthma, laryngeal edema, and angioneurotic edema; with long-term use, skin pigmentation and lenticular and corneal opacities have occurred with phenothiazines.

For full prescribing information, consult package inserts.

HOW SUPPLIED: *Tablets*—1 mg in bottles of 50 and 500; 2.5 mg and 5 mg in bottles of 50 and 500 and in Unimatic® single-dose cartons of 100; 10 mg in bottles of 50 and 500. *Elixir*—in bottles of 473 ml (1 pint) and in 60 ml dropper-assembly bottles with dropper calibrated at 0.5 ml (0.25 mg), 1 ml (0.5 mg), 1.5 ml (0.75 mg), and 2 ml (1 mg). *Injection*—in multiple-dose vials of 10 ml.

PROLIXIN DECANOATE®
Fluphenazine Decanoate Injection

Prolixin Decanoate (Fluphenazine Decanoate Injection) provides 25 mg fluphenazine dec per ml in a sesame oil vehicle with 1.2% (w/v) benzyl alcohol as a preservative.

CONTRAINDICATIONS: In presence of suspected or established subcortical brain dam patients who have a blood dyscrasia or liver damage, or who are receiving large dc hypnotics, or who are comatose or severely depressed. In patients who have shown hyp sitivity to fluphenazine; cross-sensitivity to phenothiazine derivatives may occur.

Not intended for use in children under 12.

WARNINGS: Mental and physical abilities required for driving a car or operating hea chinery may be impaired by use of this drug. Physicians should be alert to the possibil severe adverse reactions may occur which require immediate medical attention. Potentia effects of alcohol may occur. Safety and efficacy in children have not been established b of inadequate experience in use in children.

Usage in Pregnancy: Safety for use during pregnancy has not been established; possible hazards against potential benefits if administering this drug to pregnant patie

PRECAUTIONS: Caution must be exercised if another phenothiazine compound caused tatic jaundice, dermatoses or other allergic reactions because of the possibility of sensitivity. When psychotic patients on large doses of a phenothiazine drug are to u surgery, hypotensive phenomena should be watched for; less anesthetics or central n system depressants may be required. Because of added anticholinergic effects, fluphe may potentiate the effects of atropine.

Use fluphenazine decanoate cautiously in patients exposed to extreme heat or phos insecticides; in patients with a history of convulsive disorders since grand mal convulsio occurred; and in patients with special medical disorders such as mitral insufficiency cardiovascular diseases, and pheochromocytoma. Bear in mind that with prolonged there is the possibility of liver damage, pigmentary retinopathy, lenticular and corneal de and development of irreversible dyskinesia.

Fluphenazine decanoate should be administered under the direction of a physician enced in the clinical use of psychotropic drugs. Periodic checking of hepatic and renal fu and blood picture should be done. Renal function of patients on long-term therapy sh monitored; if BUN becomes abnormal, treatment should be discontinued. "Silent pneu are possible.

There is sufficient experimental evidence to conclude that chronic administration of chotic drugs which increase prolactin secretion has the potential to induce mammary nec in rodents under the appropriate conditions. There are recognized differences in the ph ical role of prolactin between rodents and humans. Since there are, at present, no ac epidemiological studies, the relevance to human mammary cancer risk from prolonged e to fluphenazine decanoate and other antipsychotic drugs is not known.

ADVERSE REACTIONS: *Central Nervous System*—Extrapyramidal symptoms are m quently reported. Most often these symptoms are reversible, but they may be persister include pseudoparkinsonism, dystonia, dyskinesia, akathisia, oculogyric crises, opisth hyperreflexia. Muscle rigidity sometimes accompanied by hyperthermia has been following use of fluphenazine decanoate. One can expect a higher incidence of such re with fluphenazine decanoate than with less potent piperazine derivatives or straig phenothiazines. The incidence and severity will depend more on individual patient sensit dosage level and patient age are also determinants. As these reactions may be alarm patient should be forewarned and reassured. These reactions can usually be contr administration of an antiparkinsonian drug such as benztropine mesylate and by sub reduction in dosage.

Persistent Tardive Dyskinesia: As with all antipsychotic agents, persistent and sor irreversible tardive dyskinesia may appear in some patients on long-term therapy or ma after discontinuation of drug. The risk seems greater in elderly patients, especially fem high dosages. The syndrome is characterized by rhythmical involuntary movements of face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, movements) and may be accompanied by involuntary movements of extremities. The known effective therapy for tardive dyskinesia; usually the symptoms are not allevi antiparkinsonism agents. If the symptoms appear, discontinuation of all antipsychotic a suggested. The syndrome may be masked if treatment is reinstituted, or drug dosage inc or a different antipsychotic agent used. Reports are that fine vermicular movements of the may be an early sign of the syndrome which may not develop if medication is stopped at t

Phenothiazine derivatives have been known to cause restlessness, excitement, o dreams; reactivation or aggravation of psychotic processes may be encountered. If dro or lethargy occur, the dosage may need to be reduced. Dosages, far in excess of the mended amounts, may induce a catatonic-like state.

Autonomic Nervous System—Hypertension and fluctuations in blood pressure ha reported. Although hypotension is rarely a problem, patients with pheochromocytoma, vascular or renal insufficiency or severe cardiac reserve deficiency such as mitral insu appear to be particularly prone to this reaction and should be observed carefully. Su measures including intravenous vasopressor drugs should be instituted immediatel severe hypotension occur; Levarterenol Bitartrate Injection is the most suitable drug; *epi should not be used* since phenothiazine derivatives have been found to reverse it Nausea, loss of appetite, salivation, polyuria, perspiration, dry mouth, headache and c tion may occur. Reducing or temporarily discontinuing the dosage will usually cont effects. Blurred vision, glaucoma, bladder paralysis, fecal impaction, paralytic ileus, tach or nasal congestion have occurred in some patients on phenothiazine derivatives.

Metabolic and Endocrine—Weight change, peripheral edema, abnormal la gynecomastia, menstrual irregularities, false results on pregnancy tests, impotency in increased libido in women have occurred in some patients on phenothiazine therap

Allergic Reactions—Itching, erythema, urticaria, seborrhea, photosensitivity, ecze exfoliative dermatitis have been reported with phenothiazines. The possibility of anapl reactions should be borne in mind.

Hematologic—Blood dyscrasias including leukopenia, agranulocytosis, thrombocyt nonthrombocytopenic purpura, eosinophilia, and pancytopenia have been obser phenothiazines. If soreness of the mouth, gums or throat or any symptoms of upper re infection occur and confirmatory leukocyte count indicates cellular depression, therap be discontinued and other appropriate measures instituted immediately.

Hepatic—Liver damage manifested by cholestatic jaundice, particularly during months of therapy, may occur; treatment should be discontinued. A cephalin floccu crease, sometimes accompanied by alterations in other liver function tests, has been re patients who have had no clinical evidence of liver damage.

Others—Sudden deaths have been reported in hospitalized patients on phenot Previous brain damage or seizures may be predisposing factors. High doses should be in known seizure patients. Shortly before death, several patients showed flare-ups of p behavior patterns. Autopsy findings have usually revealed acute fulminating pneu pneumonitis, aspiration of gastric contents, or intramyocardial lesions. Although not feature of fluphenazine, potentiation of central nervous system depressants such as analgesics, antihistamines, barbiturates, and alcohol may occur.

Systemic lupus erythematosus-like syndrome, hypotension severe enough to ca cardiac arrest, altered electrocardiographic and electroencephalographic tracings cerebrospinal fluid proteins, cerebral edema, asthma, laryngeal edema, and angi edema; with long-term use, skin pigmentation and lenticular and corneal opacities have with phenothiazines. Local tissue reactions occur only rarely with injections of flup decanoate.

For full prescribing information, consult package insert.

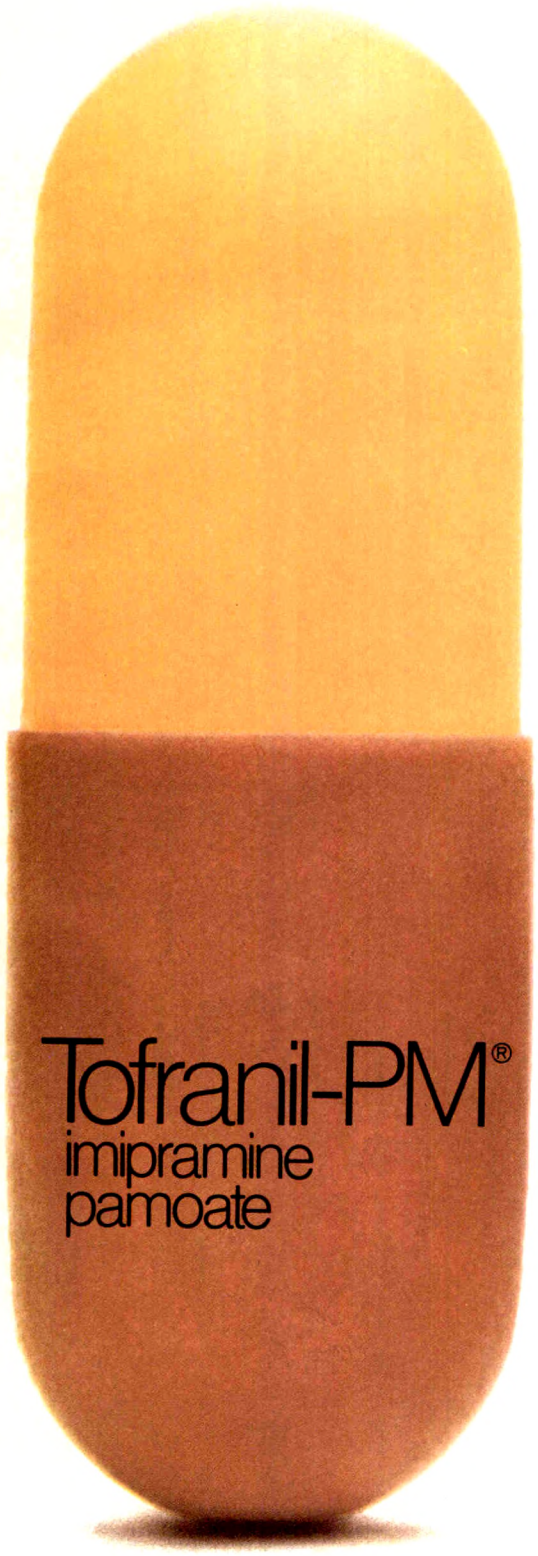
HOW SUPPLIED: 1 ml Unimatic® single-dose preassembled syringes and cartridg units and 5 ml vials.

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ny patients,

HE RIGHT ANTIDEPRESSANT



No tricyclic antidepressant works more effectively to brighten the mood, relieve sleep disturbances, and ameliorate mild anxiety.[1,2] And none appears to be better balanced[3] in terms of the biogenic amine theory of depression, which relates depression to functional deficiencies of the catecholamines.

For Tofranil-PM is almost equally effective in inhibiting the postsynaptic uptake of both norepinephrine and serotonin, the two neurotransmitters most often implicated in depression, while other tricyclic antidepressants tested are heavily skewed towards one or the other.*

*Nortriptyline, a chemical derivative of amitriptyline, also exhibits balanced action, but does not appear as effective in treating serotonin-mediated depression as imipramine.

Please see last page for a brief summary of the prescribing information.

Geigy

for many patients
with mixed
anxiety-depression,

THE RIGH

Geigy

COMBINATION*...



Although Tofranil-PM provides a sedative effect, when you decide to add a tranquilizer for more control of the anxiety, you can confidently prescribe diazepam with Tofranil-PM.

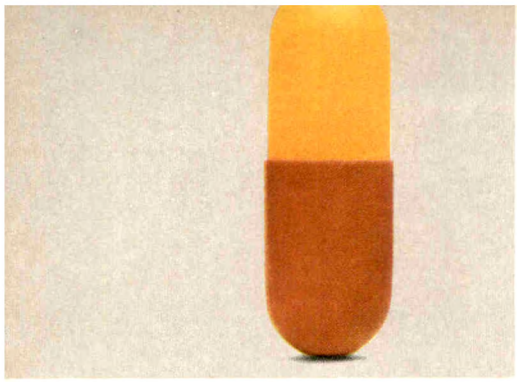
Tofranil-PM is effective when used alone or with virtually any benzodiazepine selected—a flexibility difficult to achieve with fixed-combination antidepressant regimens.

The decision of which drugs to combine, and at what dosages, remains entirely in your hands . . . where it belongs.

* The actions of benzodiazepines may be potentiated by concomitant use with Tofranil-PM. Careful consideration should be given to the pharmacology of the agents in any such combination before prescribing.

Please see last page for a brief summary of the prescribing information.

† Valium, a product of Roche Laboratories



for many patients, the right antidepressant, alone... or the basis of the right combination

Tofranil-PM® imipramine pamoate ...unsurpassed antidepressant action

For the majority of patients with neurotic depression, Tofranil-PM may be the only drug needed to produce a satisfactory brightening of mood.[1, 2] A recent report[4] notes that the two leading single-entity tricyclic antidepressants—imipramine pamoate (Tofranil-PM) and amitriptyline—provide equally effective antidepressant activity.

Better quality sleep—usually without morning "hangover"

Recent evaluation confirms that "...patients treated with imipramine pamoate tended ($P < .09$) to have a better quality of sleep than those treated with amitriptyline."[4]



75 mg 100 mg 125 mg 150 mg

Each capsule contains imipramine pamoate equivalent to 75, 100, 125 or 150 mg of imipramine hydrochloride.

Many patients show good results on Tofranil-PM 75 mg h.s.; however, the most effective daily dose may be the 150-mg capsule.

References

1. Lippman S: Depression—A good approach for the non-psychiatrist. II—Treatment of choice. Res and Staff Phys *24* (7):100-104, 1978.
2. Data on file, Geigy Pharmaceuticals, Ardsley, New York 10502.
3. Maas JW: Biogenic amines and depression. Arch Gen Psychiatry *32*:1357-1361, 1975.
4. Goldberg HL, Finnerty RJ: Which tricyclic for depressed outpatients, imipramine pamoate or amitriptyline? Dis Nerv Syst *38*:785-789, 1977.

Tofranil-PM®
imipramine pamoate

Tofranil-PM®
imipramine pamoate

Capsules of 75 mg
Capsules of 100 mg
Capsules of 125 mg
Capsules of 150 mg
For oral administration

Each 75-mg capsule contains imipramine pamoate equivalent to 75 mg of imipramine hydrochloride.
Each 100-mg capsule contains imipramine pamoate equivalent to 100 mg of imipramine hydrochloride.
Each 125-mg capsule contains imipramine pamoate equivalent to 125 mg of imipramine hydrochloride.
Each 150-mg capsule contains imipramine pamoate equivalent to 150 mg of imipramine hydrochloride.

Brief Summary of Prescribing Information

Indications For the relief of symptoms of depression. Endogenous depression is more likely to be alleviated than other depressive states. One to three weeks of treatment may be needed before optimal therapeutic effects are evident.

Contraindications The concomitant use of monoamine oxidase inhibiting compounds is contraindicated. Hyperpyretic crises or severe convulsive seizures may occur in patients receiving such combinations. The potentiation of adverse effects can be serious, or even fatal. When it is desired to substitute Tofranil-PM in patients receiving a monoamine oxidase inhibitor, as long an interval should elapse as the clinical situation will allow, with a minimum of 14 days. Initial dosage should be low and increases should be gradual and cautiously prescribed.

The drug is contraindicated during the acute recovery period after a myocardial infarction. Patients with a known hypersensitivity to this compound should not be given the drug. The possibility of cross-sensitivity to other dibenzazepine compounds should be kept in mind.

Warnings Extreme caution should be used when this drug is given to:

—patients with cardiovascular disease because of the possibility of conduction defects, arrhythmias, congestive heart failure, myocardial infarction, strokes and tachycardia. These patients require cardiac surveillance at all dosage levels of the drug;
—patients with increased intraocular pressure, history of urinary retention, or history of narrow-angle glaucoma because of the drug's anticholinergic properties;
—hyperthyroid patients or those on thyroid medication because of the possibility of cardiovascular toxicity;
—patients with a history of seizure disorder because this drug has been shown to lower the seizure threshold;
—patients receiving guanethidine, clonidine, or similar agents, since imipramine pamoate may block the pharmacologic effects of these drugs;
—patients receiving methylphenidate hydrochloride. Since methylphenidate hydrochloride may inhibit the metabolism of imipramine pamoate, downward dosage adjustment of imipramine pamoate may be required when given concomitantly with methylphenidate hydrochloride.

Since imipramine pamoate may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks, such as operating an automobile or machinery, the patient should be cautioned accordingly.

Tofranil-PM may enhance the CNS depressant effects of alcohol. Therefore, it should be borne in mind that the dangers inherent in a suicide attempt or accidental overdosage with the drug may be increased for the patient who uses excessive amounts of alcohol. (See **Precautions**.)

Usage in Children: Tofranil-PM should not be used in children of any age because of the increased potential for acute overdosage due to the high unit potency (75 mg, 100 mg, 125 mg and 150 mg). Each capsule contains imipramine pamoate equivalent to 75 mg, 100 mg, 125 mg or 150 mg imipramine hydrochloride.

Precautions An ECG recording should be taken prior to the initiation of larger-than-usual doses of imipramine pamoate and at appropriate intervals thereafter until steady state is achieved. (Patients with any evidence of cardiovascular disease require cardiac surveillance at all dosage levels of the drug. See **Warnings**.) Elderly patients and patients with cardiac disease or a prior history of cardiac disease are at special risk of developing the cardiac abnormalities associated with the use of imipramine pamoate. It should be kept in mind that the possibility of suicide in seriously depressed patients is inherent in the illness and may persist until significant remission occurs. Such patients should be carefully supervised during the early phase of treatment with imipramine pamoate and may require hospitalization. Prescriptions should be written for the smallest amount feasible.

Hypomanic or manic episodes may occur, particularly in patients with cyclic disorders. Such reactions may necessitate discontinuation of the drug. If needed, imipramine pamoate may be resumed in lower dosage when these episodes are relieved. Administration of a tranquilizer may be useful in controlling such episodes. An activation of the psychosis may occasionally be observed in schizophrenic patients and may require reduction of dosage and the addition of a phenothiazine. Concurrent administration of imipramine pamoate with electroshock therapy may increase the hazards; such treatment should be limited to those patients for whom it is essential, since there is limited clinical experience.

Usage During Pregnancy and Lactation:
Animal reproduction studies have yielded inconclusive results.

There have been no well-controlled studies conducted with pregnant women to determine the effect of imipramine on the fetus. However, there have been clinical reports of congenital malformations associated with the use of the drug. Although a causal relationship these effects and the drug could not be establi possibility of fetal risk from the maternal ingest imipramine cannot be excluded. Therefore, im should be used in women who are or might be pregnant only if the clinical condition clearly ju potential risk to the fetus.

Limited data suggest that imipramine is likely t creted in human breast milk. As a general rule, woman taking a drug should not nurse since th possibility exists that the drug may be excrete breast milk and be harmful to the child.

Patients should be warned that imipramine pa may enhance the CNS depressant effects of a (See **Warnings.**)

Imipramine pamoate should be used with caut patients with significantly impaired renal or hep function.

Patients who develop a fever and a sore throat therapy with imipramine pamoate should have cyte and differential blood counts performed. I ramine pamoate should be discontinued if ther dence of pathological neutrophil depression.

Prior to elective surgery, imipramine pamoate s be discontinued for as long as the clinical situa allow.

In occasional susceptible patients or in those r anticholinergic drugs (including antiparkinson agents) in addition, the atropine-like effects ma come more pronounced (e.g., paralytic ileus). supervision and careful adjustment of dosage quired when imipramine pamoate is administe comitantly with anticholinergic drugs.

Avoid the use of preparations, such as decong and local anesthetics, which contain any symp mimetic amine (e.g., epinephrine, norepinephr since it has been reported that tricyclic antidep can potentiate the effects of catecholamines.

Caution should be exercised when imipramine oate is used with agents that lower blood pres Imipramine pamoate may potentiate the effect depressant drugs.

Patients taking imipramine pamoate should av cessive exposure to sunlight since there have l ports of photosensitization.

Both elevation and lowering of blood sugar lev been reported with imipramine pamoate use.

Adverse Reactions Note: Although the listing follows includes a few adverse reactions which not been reported with this specific drug, the p macological similarities among the tricyclic ant sant drugs require that each of the reactions b ered when imipramine is administered.

Cardiovascular: Orthostatic hypotension, hype tachycardia, palpitation, myocardial infarction, mias, heart block, ECG changes, precipitation gestive heart failure, stroke.

Psychiatric: Confusional states (especially in th derly) with hallucinations, disorientation, delusi iety, restlessness, agitation; insomnia and nigh hypomania; exacerbation of psychosis.

Neurological: Numbness, tingling, paresthesia tremities; incoordination, ataxia, tremors; perip neuropathy; extrapyramidal symptoms; seizure tions in EEG patterns; tinnitus.

Anticholinergic: Dry mouth, and, rarely, associa lingual adenitis; blurred vision, disturbances of commodation, mydriasis; constipation, paralyt urinary retention, delayed micturition, dilation c nary tract.

Allergic: Skin rash, petechiae, urticaria, itching sensitization; edema (general or of face and tc drug fever; cross-sensitivity with desipramine.

Hematologic: Bone marrow depression includi agranulocytosis; eosinophilia; purpura; thromb penia.

Gastrointestinal: Nausea and vomiting, anorex epigastric distress, diarrhea; peculiar taste, sti abdominal cramps, black tongue.

Endocrine: Gynecomastia in the male; breast e ment and galactorrhea in the female; increase creased libido, impotence; testicular swelling; or depression of blood sugar levels.

Other: Jaundice (simulating obstructive); alter function; weight gain or loss; perspiration; flusi nary frequency; drowsiness, dizziness, weakn fatigue; headache; parotid swelling; alopecia; ness to falling.

Withdrawal Symptoms: Though not indicative tion, abrupt cessation of treatment after prolon therapy may produce nausea, headache and

How Supplied Tofranil-PM is available as follo
75-mg, coral-colored capsules equivalent to 7 imipramine hydrochloride in bottles of 30, 100 1,000 capsules, and unit strip packages of 100
100-mg, dark yellow/coral-colored capsules e to 100 mg imipramine hydrochloride in bottles and 1,000 capsules.
125-mg, light yellow/coral-colored capsules e to 125 mg imipramine hydrochloride in bottles and 1,000 capsules.
150-mg, coral-colored capsules equivalent to imipramine hydrochloride in bottles of 30, 100 capsules, and unit strip packages of 100.

For complete details, including description, ac dosage and administration, and overdosage, see full prescribing information.

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A Comprehensive, Multimedia Program Using Structured Learning Therapy

By ARNOLD P. GOLDSTEIN, Syracuse University, and ROBERT P. SPRAFKIN and N. JANE GERSHAW, Syracuse VA Hospital and SUNY Upstate Medical Center

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Edited by H.J. EYSENCK and S. RACHMAN, Institute of Psychiatry, London

Analyses are provided by Borkovec, Eysenck, Kazdin, Lang, Poser, Rosenthal, Teasdale, Wilson, and Wolpe.

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Brief Summary

ACTIONS — Pharmacologically, clorazepate dipotassium has the characteristics of the benzodiazepines. It has depressant effects on the central nervous system. The primary metabolite, nordiazepam, quickly appears in the blood stream. The serum half-life is about 2 days. The drug is metabolized in the liver and excreted primarily in the urine. (See CLINICAL PHARMACOLOGY section.)

INDICATIONS — TRANXENE is indicated for the symptomatic relief of anxiety associated with anxiety neurosis, in other psychoneuroses in which anxiety symptoms are prominent features, and as an adjunct in disease states in which anxiety is manifested.

TRANXENE is indicated for the symptomatic relief of acute alcohol withdrawal.

The effectiveness of TRANXENE in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should reassess periodically the usefulness of the drug for the individual patient.

CONTRAINDICATIONS — TRANXENE is contraindicated in patients with a known hypersensitivity to the drug, and in those with acute narrow angle glaucoma.

WARNINGS — TRANXENE is not recommended for use in depressive neuroses or in psychotic reactions.

Patients on TRANXENE should be cautioned against engaging in hazardous occupations requiring mental alertness, such as operating dangerous machinery including motor vehicles.

Since TRANXENE has a central nervous system depressant effect, patients should be advised against the simultaneous use of other CNS-depressant drugs, and cautioned that the effects of alcohol may be increased.

Because of the lack of sufficient clinical experience, TRANXENE is not recommended for use in patients less than 18 years of age.

Physical and Psychological Dependence:

Withdrawal symptoms (similar in character to those noted with barbiturates and alcohol) have occurred following abrupt discontinuance of clorazepate. Symptoms of nervousness, insomnia, irritability, diarrhea, muscle aches and memory impairment have followed abrupt withdrawal after long-term use of high dosage.

Caution should be observed in patients who are considered to have a psychological potential for drug dependence.

Evidence of drug dependence has been observed in dogs and rabbits which was characterized by convulsive seizures when the drug was abruptly withdrawn or the dose was reduced; the syndrome in dogs could be abolished by administration of clorazepate.

Usage in Pregnancy:

An increased risk of congenital malformations associated with the use of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) during the first trimester of pregnancy has been suggested in several studies. TRANXENE, a benzodiazepine derivative, has not been studied adequately to determine whether it, too, may be associated with an increased risk of fetal abnormality. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physician about the desirability of discontinuing the drug.

Usage during Lactation:

TRANXENE should not be given to nursing mothers since it has been reported that nordiazepam is excreted in human breast milk.

PRECAUTIONS — In those patients in which a degree of depression accompanies the anxiety, suicidal tendencies may be present and protective measures may be required. The least amount of drug that is feasible should be available to the patient.

Patients on TRANXENE for prolonged periods should have blood counts and liver function tests periodically. The usual precautions in treating patients with impaired renal or hepatic function should also be observed.

In elderly or debilitated patients, the initial dose should be small, and increments should be made gradually, in accordance with the response of the patient, to preclude ataxia or excessive sedation.

ADVERSE REACTIONS — The side effect most frequently reported was drowsiness. Less commonly reported (in descending order of occurrence) were: dizziness, various gastrointestinal complaints, nervousness, blurred vision, dry mouth, headache, and mental confusion. Other side effects included insomnia, transient skin rashes, fatigue, ataxia, genitourinary complaints, irritability, diplopia, depression and slurred speech.

There have been reports of abnormal liver and kidney function tests and of decrease in hematocrit.

Decrease in systolic blood pressure has been observed.

DOSAGE AND ADMINISTRATION

For the symptomatic relief of anxiety:

TRANXENE is administered orally. The capsules may be given in divided doses. The usual daily dose is 30 mg. The dose should be adjusted gradually within the range of 15 to 60 mg. daily in accordance with the response of the patient. In elderly or debilitated patients it is advisable to initiate treatment at a daily dose of 7.5 to 15 mg.

TRANXENE capsules may also be administered as a single dose dail bedtime; the recommended initial dose is 15 mg. After the initial dose, response of the patient may require adjustment of subsequent dos Lower doses may be indicated in the elderly patient. Drowsiness may o at the initiation of treatment and with dosage increment.

TRANXENE-SD tablets (22.5 mg.) may be administered as a single every 24 hours. This tablet is intended as an alternate dosage form fo convenience of patients stabilized on a dose of 7.5 mg. capsules three t a day. TRANXENE-SD tablets should not be used to initiate therapy.

TRANXENE-SD HALF STRENGTH tablets (11.25 mg.) may be ministered as a single dose every 24 hours.

For the symptomatic relief of acute alcohol withdrawal:

Recommended schedule: 1st 24 hours, 30 mg. TRANXENE initially, follo by 30 to 60 mg. in divided doses; 2nd 24 hours, 45 to 90 mg. in div doses; 3rd 24 hours, 22.5 to 45 mg. in divided doses; 4th day, 15 to 30 in divided doses. Thereafter gradually reduce to 7.5 to 15 mg. daily, discontinue as soon as condition is stable. Maximum daily dose is 90 Avoid excessive reductions in total drug on successive days.

DRUG INTERACTIONS — If TRANXENE is to be combined with other d acting on the central nervous system, careful consideration should be g to the pharmacology of the agents to be employed. Animal experience cates that TRANXENE prolongs the sleeping time after hexobarbital or ethyl alcohol, increases the inhibitory effects of chlorpromazine, but not exhibit monoamine oxidase inhibition. Clinical studies have show creased sedation with concurrent hypnotic medications. The actions o benzodiazepines may be potentiated by barbiturates, narco phenothiazines, monoamine oxidase inhibitors or other antidepressan

If TRANXENE is used to treat anxiety associated with somatic dis states, careful attention must be paid to possible drug interaction with comitant medication.

MANAGEMENT OF OVERDOSAGE — Overdosage is usually manifeste varying degrees of CNS depression ranging from slight sedation to com in the management of overdosage with any drug, it should be borne in that multiple agents may have been taken.

There are no specific antidotes for the benzodiazepines. The treatme overdosage should consist of the general measures employed in the agement of overdosage of any CNS depressant. Gastric evacuation eith the induction of emesis, lavage, or both, should be performed immedi General supportive care, including frequent monitoring of the vital and close observation of the patient, is indicated. Hypotension, th rarely reported, may occur with large overdoses. In such cases the u agents such as Levophed® (levarterenol) or Aramine® (metaraminol) s be considered.

While reports indicate that individuals have survived overdos TRANXENE (clorazepate dipotassium) as high as 450 to 675 mg., doses are not necessarily an accurate indication of the amount of dru sorbed since the time interval between ingestion and the institution of ment was not always known. Sedation in varying degrees was the common physiological manifestation of TRANXENE overdosage. Deep when it occurred was usually associated with the ingestion of other dr addition to TRANXENE.

CLINICAL PHARMACOLOGY — Studies in healthy men have shown TRANXENE has depressant effects on the central nervous sy Prolonged administration of single daily doses as high as 120 mg without toxic effects. Abrupt cessation of high doses was followed in patients by nervousness, insomnia, irritability, diarrhea, muscle ach memory impairment.

Absorption — Excretion:

After oral administration of TRANXENE, there is essentially no circu parent drug. Nordiazepam, its primary metabolite, quickly appears blood stream. In 2 volunteers given 15 mg. (50 μC) of ^{14}C-TRAN about 80% was recovered in the urine and feces within 10 days. Exc was primarily in the urine with about 1% excreted per day on day 1

HOW SUPPLIED — TRANXENE (clorazepate dipotassium) is suppli capsules in three dosage strengths:

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15 mg. capsules (all gray) in bottles of 100 (NDC 0074-3419-13) ar (NDC 0074-3419-53). Also available in ABBO-PAC unit dose packa 100 capsules (NDC 0074-3419-11).

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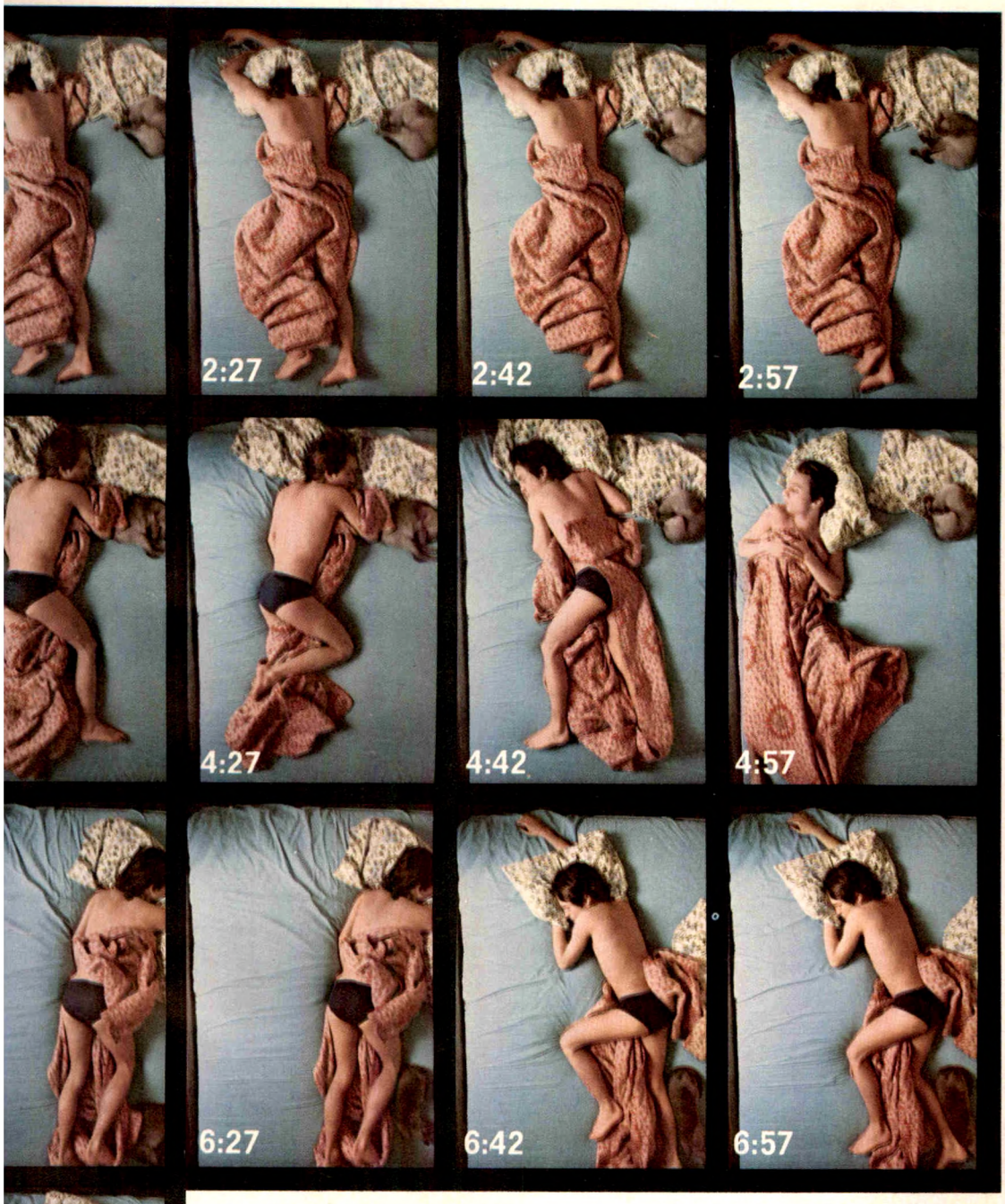
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Actual case photographs by Theodore Spagna.

Photographs of a normal subject experiencing a night of sleep.

ght of sleep



The photographs above reflect 15-minute intervals in a night of sleep at home. This "sleep portrait" is extraordinary because time-lapse photography, available since the 1880's, has never before been used to objectify human sleep. This innovative technique is less expensive than sleep laboratory evaluation, and paves the way for future field research. In the home, the intervalometer-controlled camera captures many naturalistic features of sleep behavior available only to direct observation; the rich behavioral data it records serve to complement those of the sleep laboratory, and may prove to have important significance in sleep research. This is a first step toward an ethology of sleep; it represents the joint efforts of Allan Hobson, M.D., and Theodore Spagna, photographer, sleep researchers at Harvard University.*

*Hobson A, Spagna T: Time-lapse photography and the ethology of sleep. *Sleep Res* 5:210, 1976.

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...as proved by nightlong electrophysiologic evidence of increased total sleep time in insomniac subjects in eight sleep laboratory studies.[1]



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...of relief from chronic insomnia, as proved in one study using an original 47-night protocol[2] and confirm in another.[3] (Prolonged adm istration, though seldom necessary, should be moni-tored by periodic blood cou and liver and kidney functio tests.)

Before prescribing Dalmane (flurazepam HCl), please consult complete product information, a summary of which follows:
Indications: Effective in all types of insomnia characterized by difficulty in falling asleep, frequent nocturnal awakenings and/or early morning awakening; in patients with recurring insomnia or poor sleeping habits; in acute or chronic medical situations requiring restful sleep. Since insomnia is often transient and intermittent, prolonged administration is generally not necessary or recommended.
Contraindications: Known hypersensitivity to flurazepam HCl.
Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).
Usage in Pregnancy: **Several studies of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) suggest increased risk of congenital malformations during the first trimester of pregnancy. Dalmane, a benzodiazepine, has not been studied adequately to determine whether it may be associated with such an increased risk. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**
Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage.
Precautions: In elderly and debilitated, limit initial dosage to 15 mg to preclude oversedation, dizziness and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in patients who are severely depressed, or with latent depression or suicidal tendencies. Periodic blood counts and liver and kidney function tests are advised during repeated therapy. Observe usual precautions in presence of impaired renal or hepatic function.
Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity, and elevated SGOT, SGPT, total and direct bilirubins and alkaline phosphatase.
Dosage: Individualize for maximum beneficial effect.
Adults: 30 mg usual dosage; 15 mg may suffice in some patients. *Elderly or debilitated patients:* 15 mg initially until respons is determined.
Supplied: Capsules containing 15 mg or 30 mg flurazepam HCl.

REFERENCES: 1. Data on file, Medical Department, Hoffmann-La Roche Inc., Nut NJ. 2. Kales A, *et al: Clin Pharmacol Ther* 18:356-363, Sep 1975. 3. Dement WC, Guilleminault C, Zarcone V: Progress in cli sleep research. Scientific exhibit at the American Medical Association, Atlantic City Jun 14-18, 1975. 4. Kales A, *et al: Arch Ger Psychiatry* 23:226-232, Sep 1970. 5. Kales Kales J: Recent advances in the diagnosis treatment of sleep disorders, in *Sleep Res and Clinical Practice,* edited by Usdin G. New York, Brunner/Mazel Inc., 1973, p. 61 6. Kales A, Kales JD, Martin ED: *Drug Ther* 5:31-39, Jun 1975. 7. Frost JD Jr: Data on Medical Department, Hoffmann-La Roche Nutley NJ



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proved more effective than oral hydrate,[4,5] glutemide,[4,5] pentobarbital[2] d secobarbital[5,6] — over 14 hts of administration in the ep laboratory.



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...at 15 mg: this dosage (recommended to *initiate* therapy in the elderly or debilitated to help preclude oversedation, dizziness or ataxia) provided elderly insomniacs with 83 additional minutes of sleep, on average, in the sleep laboratory.[7]

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can increase the intervals between episodes

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efore prescribing, see complete prescribing information in SK&F terature or PDR. The following is a brief summary.

WARNING
Lithium toxicity is closely related to serum lithium levels, and can occur at doses close to therapeutic levels. Facilities for prompt and accurate serum lithium determinations should be available before initiating therapy.

ndications: Treatment of manic episodes of manic-depressive illness. Maintenance therapy prevents or diminishes the intensity of subsequent pisodes in manic-depressive patients with a history of mania.

Warnings: Lithium should generally not be given to patients with significant renal or cardiovascular disease, severe debilitation or dehydration, odium depletion, or to patients receiving diuretics.

ithium therapy has been reported in some cases to be associated with orphologic changes in the kidneys.

aution patient and family to watch for diarrhea, vomiting, tremor, mild axia, drowsiness, or muscular weakness as signs of lithium toxicity, nd to discontinue therapy and contact a physician should they occur. atients receiving combined therapy with lithium and an antipsychotic ould be monitored closely for early evidence of neurologic toxicity nd treatment discontinued promptly if such signs appear. Caution atients about activities requiring alertness (e.g., operating vehicles or achinery).

thium should not be used in pregnancy, especially during the first mester, unless potential benefits outweigh possible hazards.

ot recommended for children under 12.

recautions: Lithium tolerance is greater during the acute manic phase nd decreases when manic symptoms subside.

thium therapy may lead to sodium depletion. Normal diet (including lt) and adequate fluid intake (2500-3000 ml.) must be maintained, at ast during initial stabilization period. Protracted sweating or diarrhea n decrease tolerance; in such cases, administer supplemental fluid d salt.

veating, diarrhea, and concomitant infection with elevated temperares may require temporary reduction or cessation of dosage.

dverse Reactions: Mild to moderate toxic reactions may occur at rum lithium levels from 1.5 to 2.5 mEq./l., and moderate to severe actions at levels from 2.0 to 2.5 mEq./l. Fine hand tremor, polyuria, d mild thirst may occur during initial therapy and persist. Transient d mild nausea and general discomfort also appear during initial erapy. These effects usually subside with continued treatment or mporary reduction or cessation of dosage. If persistent, discontinue sage.

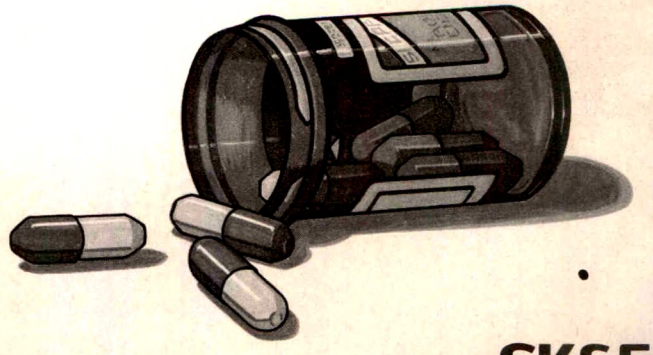
arrhea, vomiting, drowsiness, muscular weakness, and lack of coordition may be early signs of toxicity and may occur at levels below) mEq./l. At higher levels, ataxia, giddiness, tinnitus, blurred vision, d a large output of dilute urine may be seen. Serum levels above) mEq./l. may produce a complex clinical picture, involving multiple gans and systems. Serum levels should not exceed 2.0 mEq./l. during ute phase.

The following reactions appear to be related to serum lithium levels, including levels within the therapeutic range: **Neuromuscular**—tremor, muscle hyperirritability (fasciculations, twitching, clonic movements of whole limbs), ataxia, choreo-athetotic movements, hyperactive deep tendon reflex; **Central Nervous System**—blackout spells, epileptiform seizures, slurred speech, dizziness, vertigo, incontinence of urine or feces, somnolence, psychomotor retardation, restlessness, confusion, stupor, coma; **Cardiovascular**—cardiac arrhythmia, hypotension, peripheral circulatory collapse; **Gastrointestinal**—anorexia, nausea, vomiting, diarrhea; **Genitourinary**—albuminuria, oliguria, polyuria, glycosuria; **Dermatologic**—drying and thinning of hair, alopecia, anesthesia of skin, chronic folliculitis, exacerbation of psoriasis, xerosis cutis; **Autonomic**—blurred vision, dry mouth; **Thyroid Abnormalities**—euthyroid goiter and/or hypothyroidism (including myxedema) with lower T_3 and T_4. I^{131} uptake may be elevated; **EEG Changes**—diffuse slowing, widening of the frequency spectrum, potentiation and disorganization of background rhythm; **EKG Changes**—reversible flattening, isoelectricity or inversion of T-waves; **Miscellaneous**—fatigue, lethargy, transient scotomata, dehydration, weight loss, tendency to sleep.

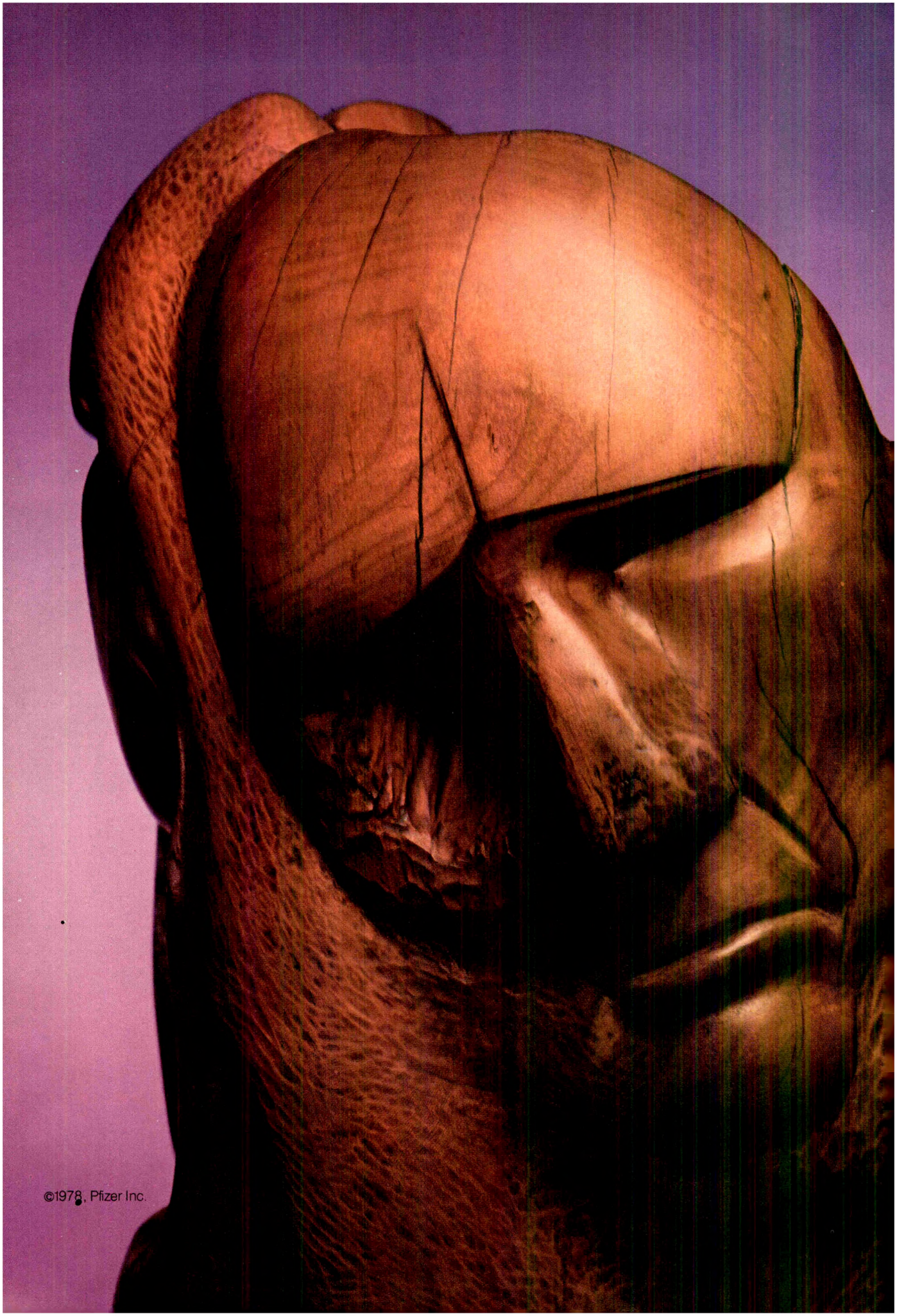
Reactions unrelated to dosage include: transient EEG and EKG changes, leukocytosis, headache, diffuse nontoxic goiter with or without hypothyroidism, transient hyperglycemia, generalized pruritus with or without rash, cutaneous ulcers, albuminuria, worsening of organic brain syndromes, excessive weight gain, edematous swelling of ankles or wrists, thirst or polyuria, sometimes resembling diabetes insipidus, and metallic taste. A single case of a syndrome resembling Raynaud's has been reported.

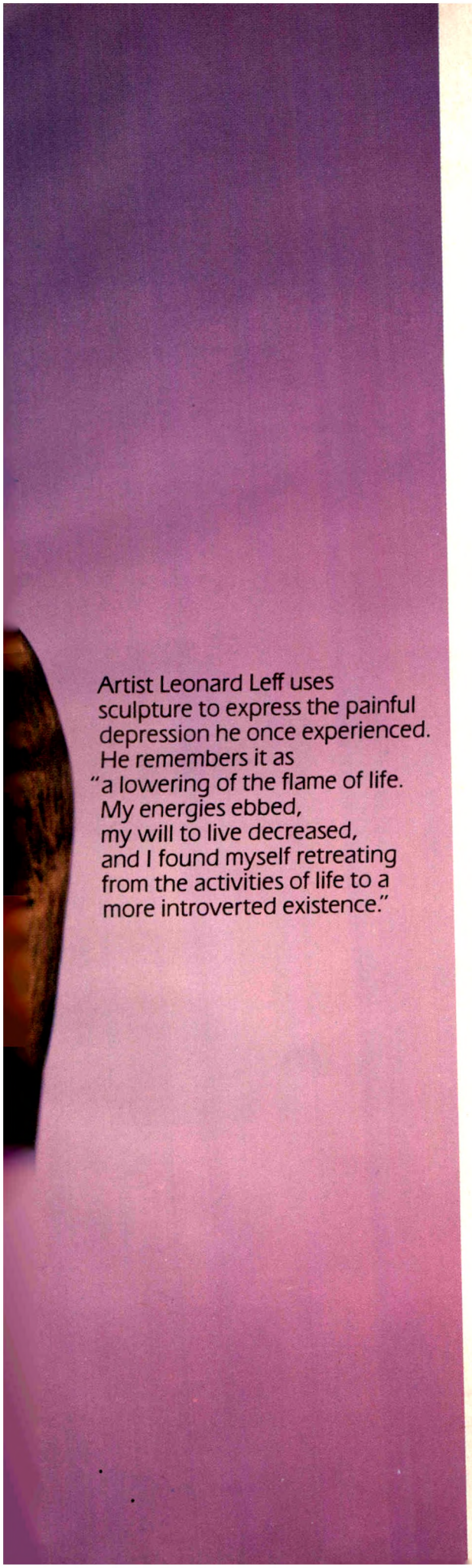
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Artist Leonard Leff uses sculpture to express the painful depression he once experienced. He remembers it as "a lowering of the flame of life. My energies ebbed, my will to live decreased, and I found myself retreating from the activities of life to a more introverted existence."

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See Brief Summary on next page for information on contraindications, warnings, precautions and adverse reactions.

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which may improve patient compliance. The to daily dosage, up to 150 mg per day, may be giv on a once-a-day schedule without loss of effec- tiveness. Sinequan may also be given on a divided dosage schedule, up to 300 mg per da

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which may help to relieve the difficulty in falling and staying asleep, and the early-morning awakening often associated with depression.

ESTABLISHED ANTIANXIETY ACTIVITY

to help alleviate the anxiety which often accom panies clinical depression.

USUALLY WELL TOLERATED

At doses up to 150 mg per day, Sinequan does n generally affect the antihypertensive activity of guanethidine and related compounds. Tachy- cardia and hypotension have been reported occasionally. Drowsiness is the most commonly served side effect. Dry mouth, blurred vision, con pation and urinary retention have been reporte

EXTENDED RANGE OF DOSAGE STRENGTHS

for flexibility in individualizing therapy.

BRIEF SUMMARY
SINEQUAN® (doxepin HCl) Capsules/Oral Concentrate

Contraindications. Contraindicated in individuals who have shown hypersensitivity to the drug, and in patients with glaucoma or a tendency to urinary retention. These disorders should be ruled out, particularly in older patients. Possibility of cross sensitivity with other dibenzoxepines should be kept in mind.

Warnings. The once-a-day dosage regimen of SINEQUAN (doxepin HCl) in patients with intercurrent illness or patients taking other medications should be carefully adjusted. This is especially important in patients receiving other medications with anticholinergic effects.

Usage in Geriatrics: The use of SINEQUAN on a once-a-day dosage regimen in geriatric patients should be adjusted carefully based on the patient's condition.

Usage in Pregnancy: Reproduction studies performed in animals have shown no evidence of harm to the animal fetus. Since there is no experience in pregnant women receiving this drug, safety in pregnancy has not been established. There are no data with respect to the secretion of the drug in human milk and its effect on the nursing infant.

Usage in Children: Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

MAO Inhibitors: Serious side effects and even death have been reported following the concomitant use of certain drugs with MAO inhibitors. Therefore, MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with this drug. The exact length of time may vary and is dependent upon the particular MAO inhibitor being used, the length of time it has been administered and the dosage involved.

Usage with Alcohol: It should be borne in mind that alcohol ingestion may increase the danger inherent in any intentional or unintentional SINEQUAN overdosage. This is especially important in patients who may use alcohol excessively.

Precautions. Since drowsiness may occur with the use of this drug, patients should be warned of that possibility and cautioned against driving a car or operating dangerous machinery while taking this drug.

Patients should also be cautioned that their response to alcohol may be potentiated.

Since suicide is an inherent risk in any depressed patient, and may remain so until significant improvement has occurred, patients should be closely supervised during the early course of therapy. Prescriptions should be written for the smallest feasible amount.

Should increased symptoms of psychosis or shift to manic symptomatology occur, it may be necessary to reduce dosage or add a major tranquilizer to the dosage regimen.

Adverse Reactions. NOTE: Some of the adverse reactions noted below have not been specifically reported with SINEQUAN use. However, due to the close pharmacological similarities among the tricyclics, the reactions should be considered when prescribing SINEQUAN.

Anticholinergic Effects: Dry mouth, blurred vision, constipation, and urinary retention have been reported. If they do not subside with continued therapy, or become severe, it may be necessary to reduce the dosage.

Central Nervous System Effects: Drowsiness is the most commonly noticed side effect. This tends to disappear as therapy is continued. Other infrequently reported CNS side effects are confusion, disorientation, hallucinations, numbness, paresthesias, ataxia, and extrapyramidal symptoms and seizures.

Cardiovascular: Cardiovascular effects including hypotension and tachycardia have been reported occasionally.

Allergic: Skin rash, edema, photosensitization, and pruritus have occasionally occurred.

Hematologic: Eosinophilia has been reported in a few patients. There have been occasional reports of bone marrow depression manifesting as agranulocytosis, leukopenia, thrombocytopenia, and purpura.

Gastrointestinal: Nausea, vomiting, indigestion, taste disturbances, diarrhea, anorexia, and aphthous stomatitis have been reported. (See anticholinergic effects.)

Endocrine: Raised or lowered libido, testicular swelling, gynecomastia in males, enlargement of breasts and galactorrhea in the female, raising or lowering of blood sugar levels have been reported with tricyclic administration.

Other: Dizziness, tinnitus, weight gain, sweating, chills, fatigue, weakness, flushing, jaundice, alopecia, and headache have been occasionally observed as adverse effects.

Dosage and Administration. For most patients with illness of mild to moderate sev starting daily dose of 75 mg is recommended. Dosage may subsequently be increa decreased at appropriate intervals and according to individual response. The usual o dose range is 75 mg/day to 150 mg/day.

In more severely ill patients higher doses may be required with subsequent gradual incr 300 mg/day if necessary. Additional therapeutic effect is rarely to be obtained by exce dose of 300 mg/day.

In patients with very mild symptomatology or emotional symptoms accompanying o disease, lower doses may suffice. Some of these patients have been controlled on doses as 25-50 mg/day.

The total daily dosage of SINEQUAN (doxepin HCl) may be given on a divided or once dosage schedule. If the once-a-day schedule is employed the maximum recommended 150 mg/day. This dose may be given at bedtime. **The 150 mg capsule strength is intend maintenance therapy only and is not recommended for initiation of treatment.**

Antianxiety effect is apparent before the antidepressant effect. Optimal antidepressant may not be evident for two to three weeks.

Overdosage.

A. Signs and Symptoms

1. Mild: Drowsiness, stupor, blurred vision, excessive dryness of mouth.
2. Severe: Respiratory depression, hypotension, coma, convulsions, cardiac arrhythmia tachycardias.

Also: urinary retention (bladder atony), decreased gastrointestinal motility (paralytic hyperthermia (or hypothermia), hypertension, dilated pupils, hyperactive reflexes.

B. Management and Treatment

1. Mild: Observation and supportive therapy is all that is usually necessary.
2. Severe: Medical management of severe SINEQUAN overdosage consists of aggre supportive therapy. If the patient is conscious, gastric lavage, with appropriate precauti prevent pulmonary aspiration, should be performed even though SINEQUAN is rapidly abs The use of activated charcoal has been recommended, as has been continuous gastric l with saline for 24 hours or more. An adequate airway should be established in comatose pa and assisted ventilation used if necessary. EKG monitoring may be required for several since relapse after apparent recovery has been reported. Arrhythmias should be treated wi appropriate antiarrhythmic agent. It has been reported that many of the cardiovascular and symptoms of tricyclic antidepressant poisoning in adults may be reversed by the slow venous administration of 1 mg to 3 mg of physostigmine salicylate. Because physostigm rapidly metabolized, the dosage should be repeated as required. Convulsions may respo standard anticonvulsant therapy; however, barbiturates may potentiate any respiratory de sion. Dialysis and forced diuresis generally are not of value in the management of overdo due to high tissue and protein binding of SINEQUAN.

Supply. SINEQUAN is available as capsules containing doxepin HCl equivalent to: 10 mg, 7 and 100 mg doxepin: bottles of 100, 1000, and unit-dose packages of 100 (10 x 10's). 25 m 50 mg doxepin: bottles of 100, 1000, 5000, and unit-dose packages of 100 (10 x 10's). 15 doxepin: bottles of 50, 500, and unit-dose packages of 100 (10 x 10's). SINEQUAN Concentrate (10 mg/ml) is available in 120 ml bottles with an accompanying dropper calib at 5 mg, 10 mg, 15 mg, 20 mg, and 25 mg. Each ml contains doxepin HCl equivalent to 1 doxepin. Just prior to administration, SINEQUAN Oral Concentrate should be diluted approximately 120 ml of water, whole or skimmed milk, or orange, grapefruit, tomato, pru pineapple juice. SINEQUAN Oral Concentrate is not physically compatible with a numb carbonated beverages. For those patients requiring antidepressant therapy who ar methadone maintenance, SINEQUAN Oral Concentrate and methadone syrup can be m together with Gatorade®, lemonade, orange juice, sugar water, Tang®, or water; but not grape juice. Preparation and storage of bulk dilutions is not recommended.

More detailed professional information available on request.

Pfizer **LABORATORIES DIVISION**
PFIZER INC.

a consideration beyond efficacy alone...

Infrequent akathisia and other disabling extrapyramidal effects...

The rapidly rising readmission rate among discharged psychotic patients is mainly due to noncompliance with antipsychotic drug therapy. And this, in turn, may be largely attributed to disabling extrapyramidal side effects, notably akathisia.[1]

Although extrapyramidal effects are characteristic of antipsychotic agents in general, with Mellaril (thioridazine) such effects are infrequent. Adding an antiparkinsonian agent—which can cause its own side effects—can usually be avoided. Mellaril (thioridazine) is contraindicated in patients with severe hypotensive or hypertensive heart disease.

Extrapyramidal Effects of Selected Antipsychotic Agents*[2]

DRUG	EXTRAPYRAMIDAL EFFECTS
Chlorpromazine	Moderate
Perphenazine	High
Prochlorperazine	High
Fluphenazine	High
Acetophenazine	Moderate
Trifluoperazine	High
Chlorprothixene	Moderate
Thiothixene	Moderate
Haloperidol	High
MELLARIL® (thioridazine)	Low

*Based on antipsychotic dosage ranges

References

1. Van Putten T: The rising rehospitalization rate of psychiatric patients. Scientific Exhibit, American Psychiatric Association, 130th Annual Meeting, Toronto, Canada, May 2-6, 1977.

2. Byck R: Drugs and the treatment of psychiatric disorders, in Goodman LS, Gilman A (eds): *The Pharmacological Basis of Therapeutics*, ed. 5. New York, Macmillan Publishing Co, Inc, 1975, pp 170-171.

Mellaril® (thioridazine)

TABLETS: 50 mg, 100 mg, 150 mg, and 200 mg thioridazine HCl, USP

MELLARIL-S™ (thioridazine) SUSPENSION, per 5 ml: thioridazine base equivalent to 100 mg thioridazine HCl, USP

Before prescribing or administering, see Sandoz literature for full product information. The following is a brief summary.

Contraindications: Severe central nervous system depression, comatose states from any cause, hypertensive or hypotensive heart disease of extreme degree.

Warnings: Administer cautiously to patients who have previously exhibited a hypersensitivity reaction (e.g., blood dyscrasias, jaundice) to phenothiazines. Phenothiazines are capable of potentiating central nervous system depressants (e.g., anesthetics, opiates, alcohol, etc.) as well as atropine and phosphorus insecticides; carefully consider benefit versus risk in less severe disorders. During pregnancy, administer only when the potential benefits exceed the possible risks to mother and fetus.

Precautions: There have been infrequent reports of leukopenia and/or agranulocytosis and convulsive seizures. In epileptic patients, anticonvulsant medication should also be maintained. Pigmentary retinopathy, observed primarily in patients receiving larger than recommended doses, is characterized by diminution of visual acuity, brownish coloring of vision, and impairment of night vision; the possibility of its occurrence may be reduced by remaining within recommended dosage limits. Administer cautiously to patients participating in activities requiring complete mental alertness (e.g., driving), and increase dosage gradually. Orthostatic hypotension is more common in females than in males. Do not use epinephrine in treating drug-induced hypotension since phenothiazines may induce a reversed epinephrine effect on occasion. Daily doses in excess of 300 mg should be used only in severe neuropsychiatric conditions.

Adverse Reactions: *Central Nervous System*—Drowsiness, especially with large doses, early in treatment; infrequently, pseudoparkinsonism and other extrapyramidal symptoms; rarely, nocturnal confusion, hyperactivity, lethargy, psychotic reactions, restlessness, and headache. *Autonomic Nervous System*—Dryness of mouth, blurred vision, constipation, nausea, vomiting, diarrhea, nasal stuffiness, and pallor. *Endocrine System*—Galactorrhea, breast engorgement, amenorrhea, inhibition of ejaculation, and peripheral edema. *Skin*—Dermatitis and skin eruptions of the urticarial type, photosensitivity. *Cardiovascular System*—ECG changes (see *Cardiovascular Effects* below). *Other*—Rare cases described as parotid swelling.

It should be noted that efficacy, indications and untoward effects have varied with the different phenothiazines. It has been reported that old age lowers the tolerance for phenothiazines; the most common neurologic side effects are parkinsonism and akathisia, and the risk of agranulocytosis and leukopenia increases. The following reactions have occurred with phenothiazines and should be considered whenever one of these drugs is used. *Autonomic Reactions*—Miosis, obstipation, anorexia, paralytic ileus. *Cutaneous Reactions*—Erythema, exfoliative dermatitis, contact dermatitis. *Blood Dyscrasias*—Agranulocytosis, leukopenia, eosinophilia, thrombocytopenia, anemia, aplastic anemia, pancytopenia. *Allergic Reactions*—Fever, laryngeal edema, angioneurotic edema, asthma. *Hepatotoxicity*—Jaundice, biliary stasis. *Cardiovascular Effects*—Changes in terminal portion of electrocardiogram, including prolongation of Q-T interval, lowering and inversion of T-wave, and appearance of a wave tentatively identified as a bifid T or a U wave have been observed with phenothiazines, including Mellaril (thioridazine); these appear to be reversible and due to altered repolarization, not myocardial damage. While there is no evidence of a causal relationship between these changes and significant disturbance of cardiac rhythm, several sudden and unexpected deaths apparently due to cardiac arrest have occurred in patients showing characteristic electrocardiographic changes while taking the drug. While proposed, periodic electrocardiograms are not regarded as predictive. Hypotension, rarely resulting in cardiac arrest. *Extrapyramidal Symptoms*—Akathisia, agitation, motor restlessness, dystonic reactions, trismus, torticollis, opisthotonus, oculogyric crises, tremor, muscular rigidity, and akinesia. *Persistent Tardive Dyskinesia*—Persistent and sometimes irreversible tardive dyskinesia, characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and sometimes of extremities may occur on long-term therapy or after discontinuation of therapy, the risk being greater in elderly patients on high-dose therapy, especially females; if symptoms appear, discontinue all antipsychotic agents. Syndrome may be masked if treatment is reinstituted, dosage is increased, or antipsychotic agent is switched. Fine vermicular movements of tongue may be an early sign, and syndrome may not develop if medication is stopped at that time. *Endocrine Disturbances*—Menstrual irregularities, altered libido, gynecomastia, lactation, weight gain, edema, false positive pregnancy tests. *Urinary Disturbances*—Retention, incontinence. *Others*—Hyperpyrexia; behavioral effects suggestive of a paradoxical reaction, including excitement, bizarre dreams, aggravation of psychoses, and toxic confusional states; following long-term treatment, a peculiar skin-eye syndrome marked by progressive pigmentation of skin or conjunctiva and/or accompanied by discoloration of exposed sclera and cornea; stellate or irregular opacities of anterior lens and cornea; systemic lupus erythematosus-like syndrome.

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SANDOZ

Infrequent akathisia and other disabling extrapyramidal effects...



a consideration beyond efficacy alone for the discharged patient who needs an antipsychotic agent

Mellaril® [thioridazine]

TABLETS: 50 mg, 100 mg, 150 mg, and 200 mg thioridazine HCl, USP

MELLARIL-S™ (thioridazine) SUSPENSION, per 5 ml:
thioridazine base equivalent to 100 mg thioridazine HCl, USP

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For brief summary, please see preceding page.

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Overview: Efficacy and Safety of the Rapid Neuroleptization Method with Injectable Haloperidol

BY PATRICK T. DONLON, M.D., JOHN HOPKIN, M.D., AND JOE P. TUPIN, M.D.

The authors review the literature on the rapid neuroleptization (titration) method with I.M. haloperidol. Most of the approximately 650 predominantly schizophrenic and manic patients represented in the studies calmed down rapidly on medication, and some demonstrated an early reduction in core psychotic symptoms. The initial doses varied widely, ranging from 1 to 30 mg, with a maximum total daily dosage of 100 mg. The medication seemed to have been well tolerated in all cases, with no reported major complications. The authors conclude that the method shows definite merit with agitated and belligerent patients. However, they make a number of specific recommendations for further research to clearly establish the effectiveness and safety of this method of neuroleptic administration.

THE RAPID NEUROLEPTIZATION (titration) technique is a method of administering repeat doses of neuroleptic medication under close clinical supervision that provides rapid control of acute functional psychotic symptoms in most patients. The technique is an important landmark because agitated and belligerent patients are often seen clinically. Rapid symptom control not only relieves distressing symptoms but also greatly reduces the risk of injury to self and others.

The advocates of the technique recommend administering I.M. medication every 30–60 minutes until symptoms are controlled (1, 2). Parenteral medication is preferred over oral medication because the former works faster and the more potent neuroleptics are preferred over chlorpromazine because of their greater cardiovascular safety, reduced sedation, and absence of pain at the injection site. Although many potent agents have been used, haloperidol has been the most extensively evaluated and reported. Clinically, 1–4 injections of 2.5–10.0 mg each are normally required to produce rapid control. The patient is then switched to oral medication.

In this paper we will review the published reports on injectable haloperidol in acute psychotic states and critically evaluate the clinical findings of the rapid neuroleptization technique. More specifically, we will focus on both medical and nonmedical factors that must be carefully evaluated before unequivocally establishing the effectiveness of the rapid titration method in the treatment of schizophrenia.

LITERATURE REVIEW

Open Studies

Danik and Goverdham (3) reported the successful use of parenteral haloperidol administered in 10-mg doses to initiate treatment of psychotic patients. They had a favorable experience treating acute manic and schizophrenic patients with both intravenous and intramuscular haloperidol in total daily dosages up to 25 mg. Nearly all of their patients showed symptom remission within 24 hours. Medication was well tolerated, and adverse effects were predominantly extrapyramidal symptoms.

Oldham and Bott (4) administered I.M. haloperidol to 124 patients, using initial doses of 10–30 mg to control excitement. With total daily doses of 20–40 mg, 73% of Oldham and Bott's patients (91 of 124) were stabilized, usually within 72 hours. Of the remainder, 27 patients (22%) were partly controlled and only 6 (5%) did not respond at all. The patients whose symp-

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toms were controlled by parenteral haloperidol were then given oral haloperidol and orphenadrine hydrochloride in diminishing doses to maintenance levels. The treatment regimen had to be discontinued and replaced by another in only 6 of the 124 patients. Four of these 6 had not responded; 1 had severe dystonia, and 1 had transient hypotension (90/60 mm/Hg). Oldham and Bott recommended using haloperidol in doses of 20–30 mg initially to control psychotic excitement because its margin of safety permitted use in all but the frailest patients.

Feldman and associates (5) administered 5 mg of I.M. haloperidol 3 times daily, generally for a maximum of 3 days, to 25 inpatients suffering from acute psychotic episodes or exacerbations. Clinical evaluation after 3 days of treatment showed a significant improvement in the areas of affect, social behavior, motor behavior, perception, ideation, and sensorium. The main side effects were extrapyramidal symptoms, but these were easily controlled.

Sangiovanni and associates (6) administered I.M. injections of haloperidol (10–30 mg) 1–3 times daily as part of the initial treatment of 40 acutely disturbed psychotic inpatients. A maximum of 60 mg was administered daily to each patient. A wide range of therapeutic doses titrated to response in individual patients resulted in marked or moderate control of symptoms in 36 of the 40 patients (90%) in 72 hours. Fourteen patients (35%) were controlled by parenteral haloperidol in doses totaling less than 30 mg; 14 patients (35%) were controlled by 30–60 mg; and 8 patients (20%) required more than 60 mg. Four patients (10%) responded only minimally and required other treatment.

The adverse reactions of the patients in this study were unexceptional. Lethargy lasting several hours was noted in approximately a third of the patients on the first day of treatment. At low doses (less than 20 mg), parkinsonism developed in 6 patients and coarse tremor of the hand developed in 5 others. Dystonic reactions in 4 other patients were rapidly relieved by treatment with antiparkinsonian agents.

Carter (7) retrospectively tabulated and analyzed the outcome of 48 acutely disturbed psychotic patients receiving parenteral haloperidol. Except for a massively obese, excited, manic man who received an initial dose of 20 mg, all of these patients received 5–10 mg at 30–60-minute intervals. Thirty-six of the 48 patients received a total of 40 mg or less of parenteral haloperidol over 24 hours. The remaining 12 patients received a total of 45–100 mg I.M. over 24 hours.

The therapeutic response was determined by comparing the severity of psychomotor agitation, hostility and belligerence, auditory hallucinations, and flight of ideas at pretreatment and posttreatment. Within 2 hours of treatment initiation all ratings were significantly reduced. Only 18 patients had residual symptoms at 4 hours, and only 4 of the 48 patients (8%) responded minimally or not at all to the treatment. Extrapyramidal symptoms, predominantly dystonia or cogwheel rigidity, occurred in 14 subjects at total doses between 5 and 30 mg. Transient hypotension occurred in 3 cases. Sedation was not characteristic, but 16 subjects slept following symptom relief.

Firling (8) administered parenteral haloperidol to 169 chronic psychotic patients who were experiencing symptom exacerbation, usually associated with the discontinuation of neuroleptic medication. All were acutely disturbed and often belligerent. These patients, 124 men and 45 women, were diagnosed as follows: 130 had schizophrenia, 20 had manic-depressive psychosis, 10 had drug-abuse-induced psychosis, and 9 had other diagnoses. One hundred twenty-five of the subjects required a single 10-mg injection, and 23 required a single 5-mg injection. The remaining 21 patients required 2–4 injections of 10 mg of haloperidol given at 30-minute intervals.

The outcome was determined by a retrospective review of case records. Within 20 minutes after the first injection, psychomotor agitation was significantly reduced in 157 patients and hostility and belligerence were reduced in 136 patients. The related signs and symptoms of psychosis were also reduced.

The medication was tolerated in all cases without undue sedation or hypotensive reactions. Sixteen subjects developed extrapyramidal symptoms (12 with dystonia) that remitted either spontaneously or after administration of 2 mg of contra-active trihexyphenidyl orally.

In a study of injectable haloperidol that is being prepared by Hopkin, Donlon, and Schaffer, 2.5–10 mg of I.M. haloperidol was administered every 30 minutes as needed over a 6-hour evaluative period with 25 newly admitted, hospitalized, acute schizophrenic patients. The sample consisted of 8 women and 17 men with a mean age of 26 and a mean dosage of 23 mg.

Most of the patients responded rapidly to treatment. The patients demonstrated the following improvement at the 6-hour clinical global improvement rating: marked, 6; moderate, 11; slight, 5; and no change, 3. For the group as a whole, significant reduction occurred in excitement and belligerence as well as in core pathological signs and symptoms (thought disorganization, delusions, hallucinations, and suspiciousness). One patient developed dystonia, 1 patient developed mild transient and asymptomatic hypotension, and 5 patients experienced sedation. The cardiovascular safety of the technique was further documented by monitoring vital signs every 30 minutes on all subjects and obtaining Holter monitors on 6 subjects. No significant changes were identified.

Double-Blind Studies

Man and Chen (9) administered I.M. injections of 5 mg of haloperidol to 15 acutely disturbed psychotic adults and 50 mg of chlorpromazine to 15 others. Up to 7 injections were administered at 20-minute intervals until the patients were no longer assaultive or manic or until it was evident that further injections would be of no therapeutic benefit. Two or 3 injections were re-

quired in most of the cases (N=16). The results were evaluated by the Rating Scale for Target Symptoms (mania, agitation, hostility) (9) and the Brief Psychiatric Rating Scale (BPRS). In patients treated with haloperidol, the values for the severity of target symptoms were reduced by approximately half in an average of 2 hours. The total BPRS mean score was reduced by 45% from a pretreatment mean value of 56 to an adjusted final mean of 31. The results with chlorpromazine were similar, although an average of 2½ hours was required for equivalent reductions.

Two patients treated with chlorpromazine were removed from the study after severe hypotensive reactions. One of these reactions occurred after 1 injection of 50 mg of chlorpromazine. The other occurred after 2 injections of 50 mg. In both cases, blood pressure was not obtainable and was restored only after intensive emergency measures. In another patient who received chlorpromazine, the development of status epilepticus necessitated treatment with anticonvulsants. There were no adverse reactions among the patients given haloperidol.

Follow-up evaluation on day 3 showed that patients whose symptoms were controlled continued to improve after switchover to oral medication. These patients were transferred to a day hospital or discharged to an outpatient clinic for maintenance therapy. Man and Chen concluded that haloperidol was the safer agent for injection every 30 minutes and preferable to chlorpromazine for the treatment of acutely disturbed psychotic patients.

Reschke (10) compared parenteral haloperidol (1, 2 or 5 mg) with chlorpromazine (25 mg) and with a placebo control in the management of disruptive behavior in adults with acute schizophrenia. A total of 50 patients were treated in five groups of 8–11 patients each. The drugs and placebo were administered I.M. every 30 minutes until agitation, excitement, and assaultiveness were controlled or until a maximum of 4 injections were given. An analysis of global evaluations and BPRS scores indicated that control was significantly ($p<.05$) greater in patients who received haloperidol in doses of 2 or 5 mg than in those who received doses of 1 mg of haloperidol, 25 mg of chlorpromazine, or placebo. The largest reduction in BPRS signs and symptoms occurred in the 10 patients who received 5 mg of haloperidol every 30 minutes; an average of 2.8 injections of this dose reduced pretreatment BPRS values by 42%.

The side effects of the drugs were minimal in this study. Systolic blood pressure was reduced transiently from normal to less than 100 mm/Hg in 2 patients, 1 of whom received a total of 10 mg of haloperidol and the other a total of 50 mg of chlorpromazine. Drowsiness was evident in 13 of the 29 patients who received haloperidol (45%), but only 1 fell asleep during the 2-hour test period. However, 6 of the 10 patients who received chlorpromazine and none of the 11 who received placebo fell asleep. Reschke concluded that 2 or 5 mg of I.M. haloperidol was superior to the other regimens tested in the control of disruptive behavior in acute schizophrenic patients.

Anderson and associates (11) treated 14 acutely psychotic patients with 10 mg of I.M. haloperidol every 30 minutes and another 10 patients with 5 mg of haloperidol every 60 minutes. Both regimens rapidly controlled disruptive behavior within 3 hours after the first injection (7 of the 14 patients receiving 10-mg doses and 4 of the 10 receiving 5-mg doses). Another 5 patients improved markedly within 72 hours. Of the remaining 8 patients, 5 were dropped from the study and 3 others improved after more than 72 hours. In addition to calming the patients, haloperidol significantly reduced the severity of the signs of psychosis, specifically hallucinations, unusual thought content, suspiciousness, conceptual disorganization, and grandiosity. In both test groups, mean scores on the BPRS were reduced from pretreatment values of 57–66 to less than 30. In this study marked remission was not evident at total doses under 15 mg, and additional improvement was not evident in patients who received total doses higher than 45 mg.

The side effects of haloperidol were mild and occurred equally in patients in either regimen; none was unusual or required discontinuation of the regimen. Extrapyramidal signs in 8 patients were controlled with 2 mg of I.M. benztropine mesylate followed by oral medication if necessary. Eight patients were drowsy but in no case sedated to the point that they could not be interviewed.

The study demonstrated the feasibility of bringing about remission in patients with acute psychosis within a few hours and also the need for flexible dosage: some moderately disturbed patients required high doses for relief of symptoms and many severely disturbed patients did not. This has been Anderson and Keuhnle's clinical experience and the basis of their recommendation (12) to use 5–10 mg of parenteral haloperidol every 30 to 60 minutes for the rapid control of sudden onset psychosis.

Gerstenzang and Krulisky (13) compared parenteral haloperidol and chlorpromazine in 58 patients brought to an emergency room for disruptive behavior. The symptom pictures of these patients ranged from marked agitated behavior to marked negativism and withdrawal. Approximately half of the patients were given a provisional diagnosis of schizophrenia. The subjects were evaluated 30 to 60 minutes after a single injection of 5 mg of haloperidol or 50 mg of chlorpromazine.

Half of the 30 patients receiving haloperidol became calm, cooperative, and alert, and another 8 of these patients improved. In contrast, only 3 of the 28 chlorpromazine patients were controlled successfully, and 11 were partly controlled. Covariant analysis of BPRS scores significantly favored haloperidol for conceptual disorganization, tension, hostility, uncooperativeness, and excitement. None of the statistical comparisons favored chlorpromazine. No adverse reactions, including hypotension and extrapyramidal symptoms,

occurred during the brief evaluation period.

Levenson and associates (14) administered single daily doses of parenteral fluphenazine HCl, thiothixene, or haloperidol to newly hospitalized acute schizophrenic patients to compare efficacy and speed of recovery. The doses were predetermined and equipotent and were given I.M. each day at 9:00 a.m. Initial doses were 2.5 mg for fluphenazine HCl and haloperidol and 5 mg for thiothixene. By day 4 the doses were gradually elevated and fixed at 7.5 mg and 15 mg, respectively. The study period lasted 21 days, but patients who responded optimally were terminated early.

Seventeen patients started the investigation, but 5 received administrative discharge before symptom remission and were not in the data analysis. With this small sample size no significant differences were found using the Kruskal-Wallis test, although a definite trend favored haloperidol for speed of recovery. The overall rate of remission for all groups combined was 83%. Side effects were similar for all three groups and were limited to extrapyramidal symptoms.

Paprock and Versiani (15) compared parenteral loxapine with haloperidol in 35 acutely and severely ill newly hospitalized schizophrenic patients. Seventeen patients were treated for 4 days with loxapine in a mean daily dosage of 115.4 mg (range, 87.5–125 mg), and 18 patients were treated with haloperidol in a mean daily dosage of 11.5 mg (range, 8.8–12.5 mg). Progress was monitored using daily BPRS, NOSIE, and clinical global impression rating scales. Laboratory determinations included complete blood count, urine analysis, and chemistries. Vital signs were monitored, and adverse effects were assessed and recorded.

A few significant differences were found using analysis of covariance. Loxapine demonstrated some superiority in the reduction of agitated/excited and aggressive behaviors. According to global assessment 10 of the 17 loxapine patients and 15 of the 18 haloperidol patients improved. Adverse effects were similar for both groups and were predominantly extrapyramidal symptoms and drowsiness.

The 25 patients who responded favorably were switched to oral medication for a 4-week assessment period; the same rating scales were used. The switchover was made on an equal mg-for-mg basis. After 3 days, dosage was left open and dependent on clinical response up to maximum daily dosages of 150 mg of loxapine and 15 mg of haloperidol. Improvement was considerable and comparable for the two groups given oral medication.

Stotsky (16) treated 30 acutely psychotic, agitated outpatients with either I.M. haloperidol or I.M. thiothixene. Patients received 4 mg or 8 mg of thiothixene or haloperidol every hour as needed during a 4-hour treatment program. An additional 2-hour period was allowed for further observation and ratings for a total 6-hour evaluative period.

The total mean dosage for the 15 haloperidol patients was 15 mg (range, 8–32 mg); for the 15 thiothixene patients it was 10.1 mg (range, 4–16). All 30 patients achieved rapid tranquilization and significant improvement on the total BPRS score, the 4 BPRS factors, and the 17 symptoms of the Psychiatric Target Symptom Profile (16). Side effects were few and similar for both groups, the most frequent being drowsiness. Analysis of covariance demonstrated no significant differences between the two groups for efficacy or time required to control symptoms.

DISCUSSION

Striking in this review is the consistency of the reports of favorable and rapid therapeutic outcome for parenteral haloperidol in reducing acute excitement and agitation in psychotic patients. Some authors also reported a reduction in the core symptoms of schizophrenia and mania. Although most of the patients were schizophrenic, the method appears effective for other acute psychotic states as well. Relative safety was reported in all subjects; the most common adverse effects were extrapyramidal symptoms, sedation, and transient hypotension. No major treatment complications were reported for the approximately 650 subjects taking haloperidol who were receiving initial doses in the range of 1–30 mg I.M. and total daily dosages up to 100 mg I.M.

However, many methodological shortcomings in these studies make the data difficult to discuss. Most of the study periods lasted hours or a few days, making it difficult to precisely establish outcome and safety. Of note also, some of the studies lacked a placebo control to monitor nonmedical effects and spontaneous recoveries. The double-blind comparison studies should have allowed for differences in potency before claiming that haloperidol is efficaciously superior.

In spite of these shortcomings, the following guidelines may be summarized from the review:

1. Acute psychotic states may be associated with agitated and belligerent behavior that requires immediate medical attention to reduce the risk of injury to self or others and to provide greater comfort.

2. Injectable haloperidol is effective in rapidly controlling the signs and symptoms of acute psychotic excitement. Other neuroleptics when controlled for potency are probably equally effective.

3. Medication dosages must be highly individualized because dosage requirements cannot be predetermined by body weight, past requirements for neuroleptic dosage, presence and severity of particular target symptoms, or degree of psychotic impairment.

4. Individual doses normally range from 2.5–10.0 mg I.M., and patients typically require 1–4 injections given at 30–60-minute intervals.

5. The response to medication normally can be observed within minutes, minimizing the possibility of overdosing or cumulative medical effects.

6. In some schizophrenic and manic patients the reduction of excitement may also be associated with a

reduction in core psychotic symptoms (e.g., cognitive disorganization, hallucinations, and delusions).

7. The method is relatively safe, and the most common adverse effects are extrapyramidal symptoms, sedation, and hypotension.

8. Oral neuroleptics when indicated may be substituted following response to parenteral medication.

Ayd (2) has outlined guidelines for rapid I.M. neuroleptization (2). He stressed the importance of gathering historical data, evaluating general health, and establishing a preliminary diagnosis before initiating treatment. He further reported that I.V. neuroleptic therapy is preferable over I.M. in some patients. Our experience has been exclusively with I.M. neuroleptic therapy, so we cannot recommend I.V. therapy until its safety is further documented. In psychotic subjects who are toxic from psychedelic drugs or organic conditions with or without delirium, we have found that neuroleptics may potentiate the psychosis or mask developing medically important signs or symptoms. Thus the method should be reserved for physically healthy, excited schizophrenic or affectively disordered patients who require immediate symptom relief. Further, the least amount of medication should be given that will provide such relief.

Dosages and dosage schedules varied widely in the studies reviewed. The patient groups treated seemed to have been similar. Thus, the dosages administered might have reflected a bias toward a particular treatment regime among investigators rather than individual dosage requirements. That the outcomes were similar in all of these studies suggests that the medication is well tolerated in a wide range of dosages and without behavioral toxicity in higher dosages. The fact that most patients responded to the more conservative treatment suggests that the more aggressive use of medication should be withheld and used only as a backup treatment.

SUGGESTIONS FOR FURTHER RESEARCH

Further research is needed to answer the question of which patients are most likely to benefit from rapid I.M. neuroleptization with haloperidol. Most of the subjects selected for the studies reviewed were excited psychotic patients. The typical clinical response was a calming effect; some subjects demonstrated a reduction in core psychotic symptoms. It remains unclear whether the chronic schizophrenic patient and the acutely schizophrenic subject without dysphoric affect would benefit similarly. Furthermore, in patients who respond, do I.M. preparations act faster than oral neuroleptics in reducing core psychotic symptoms? Is the initial treatment response attributed to I.M. medication sustained after switching to oral neuroleptics? If so, are particular subgroups of patients involved? These questions may be more fully answered in well-controlled double-blind oral versus parenteral investigation that lasts for several weeks and that uses a heterogeneous clinical population of schizophrenic patients.

Another question requiring further research is, why is the I.M. rapid neuroleptization method effective? It has long been recognized that neuroleptics are effective in controlling psychotic states. However, some of the authors of the studies reviewed here strongly suggested that I.M. medication is more effective than oral preparations with acute patients. Are we dealing simply with patient selection factors? For example, it is well known that acute schizophrenic patients with dysphoric affect have a more favorable prognosis when given neuroleptics than more insidiously decompensated patients with a limited show of emotion. Certainly, the samples of patients in these studies consisted almost exclusively of those who were most likely to respond favorably to neuroleptics.

What about the effect of hospital and other emergency room influences (e.g., being removed from possible stress and placed in a protective environment) on the patient's overall response? Reschke's study (10), the only one that used a placebo control, demonstrated that an adequate dosage of a neuroleptic is significantly superior to a placebo injection, indicating that nonmedical influences are minor. Anderson and associates (11) also concluded that nonmedical influences are of minor importance. However, further studies like Reschke's, which compared an I.M. placebo versus an I.M. neuroleptic, are needed.

A third question for future research is, if the I.M. method is superior, why? Assuming that the pharmacological properties of parenteral and oral preparations of an agent are identical, then either the increased bioavailability or the rapid saturation of tissue at the site of action in the brain must account for the superiority of the I.M. method. Both would seem to favor I.M. preparation because I.M. absorption is faster and bioavailability is increased two- to threefold by eliminating the initial passage through the gut and liver and avoiding early metabolic breakdown. Thus, the excited patient who receives 15 mg of I.M. haloperidol over a 2-hour period may be receiving the equivalent of 40–50 mg orally. In fact, according to a personal communication from McNeil Laboratories in May 1976, if a 1-mg dose of haloperidol is administered orally and another parenterally, the patient's serum level in the first 20–30 minutes will favor the parenteral drug 10 to 1. After 24 hours these levels plateau to a 2 to 1 parenteral advantage. Thus, the early availability of higher amounts of the drug at the receptor sites provides for the rapid clinical response.

It remains unclear whether the dysphoric acute schizophrenic patient would respond equally to a comparably large dosage of oral medication. We hypothesized that this was precisely the case when we reported that treatment response could be accelerated by rapidly increasing the oral dosage of neuroleptic with acute schizophrenic patients (17). However, our preliminary research did not support this hypothesis (18). It is still of clinical interest to know if a particular

serum level is necessary for clinical response and if this response can be more readily obtained via oral or parenteral preparations. Although serum levels were not obtained in the studies reviewed, one can certainly conclude that dosage requirements vary widely among patients. Also, prescribing habits varied so widely in these studies that it is difficult to comment on dose-response relationships. Most patients seemed to demonstrate clinical response, and this often occurred only after repeat injections. However, some subjects were treatment failures in spite of large dosages. Thus, total dosage for optimal response varied widely and must be highly individualized. As Carter emphasized (7), high dosages should be administered only if there is a clear indication that they will be of therapeutic value.

Finally, haloperidol has long been recognized as a neuroleptic that rapidly controls excitement. Perhaps this effect is greater than that of other neuroleptics when controlled for potency. Clinically this is often assumed, i.e., haloperidol is often considered the agent of choice for acute mania. However, in the comparisons cited above with I.M. loxapine (15), fluphenazine (14), and thiothixene (14, 16), outcome was similar, and in the comparison studies with chlorpromazine outcomes may have been similar for efficacy if dosages had been better controlled for potency (9, 10, 13).

The problem with switchover to oral agents is another subject requiring further research. According to our clinical experience switchover to oral agents may be problematic. However, this topic received little attention in the studies reviewed. Ayd (2) recommended doubling the total I.M. dose required in the first 24 hours to achieve symptomatic control. Mason and Granacher (1) recommended increasing the oral dosage up to half that required for I.M. control. These guidelines are helpful, but they should be considered rough approximations. Clinically, if one follows these guidelines some patients will become overmedicated and others will show signs of relapse. Thus, dosage requirements remain highly individualized and some patients require frequent dosage changes.

Most certainly some patients who respond favorably to I.M. agents may not require additional neuroleptic medication. Others may be found to be suffering from affective disorders, and their treatment of choice will be antidepressive agents or lithium. Additional research should help document which patients should be continued on neuroleptics and to better establish guidelines for switchover to oral agents.

Another question requiring research is that of safety. The relative safety of haloperidol is well established (19). In general, adverse effects are related to dosage. The risks of treatment should be outweighed by the potential merits. According to the studies reviewed, I.M. haloperidol is well tolerated and does not involve serious complications. However, the longer term adverse effects and toxicity of the drug were not evaluated in these studies because of the rapid switchover to oral medication. Such data could be obtained only by maintaining patients for several days on injectable medication, a practice that is not normally clinically practical. Therefore, since adverse effects are related to dosage, the physician should use the least amount of medication necessary to provide therapeutic response.

Finally, the investigation of pharmacological effects has provided important leads into the biology of schizophrenia (20). Rapid neuroleptization may be of particular value here due to higher initial serum levels and rapid patient response.

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Coercive Persuasion (Brainwashing), Religious Cults, and Deprogramming

BY J. THOMAS UNGERLEIDER, M.D., AND DAVID K. WELLISCH, PH.D.

Psychiatric interviews and psychological testing were conducted with 50 members or former members of a variety of religious cults who contacted the authors about the issue of deprogramming. The subjects were divided into four groups: cult members who feared deprogramming, those who had returned to the cult after deprogramming, ex-cult members who had left after deprogramming, and those who had left without deprogramming. There were significant differences between these groups on length of time in the cult, perception of and resistance toward the deprogramming experience, status of parental marriage, and who became a deprogrammer. No evidence of insanity or mental illness in the legal sense was found.

THE INFLUENCE of religious cults, or new religions, on young people and the recent spate of efforts to "deprogram" them have raised intriguing and disturbing questions for the medical, legal, and mental health professions, as well as for religious leaders and government authorities. Religious freedom and parental control, so central to American society, are crucial related issues. Deprogramming has recently received wide coverage in the mass media. The procedure is performed at the instigation of and financed by the parents. The deprogrammer forcibly removes the person who has reached the age of majority) from his cult or group and puts him through a series of procedures called deprogramming. These procedures vary widely and may range from gentle rap sessions to sleep deprivation and sensory overload, with marathon-type encounters that feature shouting, repetitious derogations of the cult, isolation of the person from former associates, moving the deprogramee from place to place within a period of days, and, occasionally, use of physical force in the event that he or she tries to leave or escape.

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The issue of group control and group influence over an individual's ideologic, political, or religious beliefs is not new. Concern over group domination and manipulation of individual belief systems peaked in the behavioral sciences after the Korean War, during which prisoners of war were purportedly brainwashed and influenced to accept Communist doctrine. Lifton (1), after intensive study of the Communist Chinese methods for mass ideological influence, suggested eight psychological themes as basic ingredients in "thought reform": 1) milieu control, 2) mystical manipulation, 3) demands for political and/or ideologic purity, 4) personal confession, 5) acceptance of basic group dogma as sacred, 6) constriction or "loading" of language into polarizing terms, 7) subordination of person to doctrine, and 8) dispensation of existence. Lifton theorized that a firm control over these eight variables would result in a process of "ideologic totalism."

Our purposes in this paper are 1) to report on certain psychosocial characteristics of 50 individuals who are or were members of several new religions, 2) to describe their ability to make sound legal decisions about their choice of lifestyle, and 3) to call attention to some of the problematic issues woven into the complicated patchwork of religious cults.

METHOD

As word spread of our interest, we were contacted by a variety of people on both sides of the issue of cults and deprogramming who asked us to evaluate members who had left cults, who had been taken from but later escaped and returned to cults, or who feared being taken from the cults for purposes of deprogramming. We examined these people without charge. We did indicate, however, that we would be willing, if asked, to give our findings in a court proceeding. However, this was never required. This became the motivation of many cult members to cooperate with us. Those who were no longer cult members cooperated mainly out of a desire to further knowledge in this area. It is important to note that we did not promise cult members that our findings would be positive or helpful to them should they be presented in court. We promised only to deliver the data, if asked, exactly as obtained, whether they indicated soundness of personality and intellect or deficits in these spheres.

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Subjects

We examined subjects' status in relation to the groups rather than focusing on which cults they belonged to or had belonged to. The first group consisted of 22 subjects who feared deprogramming and remained in cults (concerned members). The second group consisted of 11 subjects who had been deprogrammed and had returned to their cults (returnees). The third group included 9 subjects who had been deprogrammed and had not returned to the cult (the nonreturnees); 6 of these 9 subjects had subsequently become deprogrammers. The fourth group consisted of 8 subjects who had left the cult of their own volition (voluntary ex-members). All subjects were voluntary and self-referred, and they received no monetary or other form of compensation. The subjects were tested in various locales in California and New York during their participation in the study.

Procedures

Both psychological and psychiatric data were gathered during one visit. Psychiatric data consisted of 1) a structured interview, which probed areas such as mental and physical health history, drug use history, family variables, experience in the group, and general motivational characteristics, and 2) a traditional mental status examination, which assessed affect, cognition, general psychological orientation, degree of insight, and social judgment.

Psychological data consisted of 1) the short form of the Weschler Adult Intelligence Scale, which correlates highly (r=.97) with the full-length form, 2) the Minnesota Multiphasic Personality Inventory (MMPI), 3) the Draw-a-Person test (not discussed in this paper), and 4) the Interpersonal Check List (ICL), developed by La Forge and Suczek (2). On the checklist all subjects were asked to rate the way they viewed themselves before and after they joined the group. The two main dimensions of dominance/submission and love/hate (affiliativeness/disaffiliativeness) were the ones considered in the study.

Statistical Analysis

The psychiatric interview data were sorted into categories, coded, and analyzed by the chi-square test with unequal cells. The psychological test data were analyzed by multivariate or one-way analysis of variance (ANOVA).

RESULTS

Structured Interview Data

Data analysis revealed that most of the interview variables did not significantly differentiate the groups. Nonsignificant variables included age, sex, marital status, religion of nuclear family, birth order, drug history, mental health history, educational level, and father's occupation.

However, some significant differences did emerge. The length of time in the cult was highly significant ($p \leq .0003$); 68% of the concerned members group and 91% of the returnees had been in the cult 1 year or more, while all of the nonreturnees had been in for less than 1 year. The voluntary ex-member group was more evenly spread across time intervals—38% had been in for less than 1 year and 62% for 1 year or more.

Perception of and resistance toward deprogramming significantly differentiated the groups ($p \leq .0001$). All subjects who had returned to the cult viewed their deprogramming experience as "not gentle" while 88% of those who did not return viewed it as "gentle." This is associated with physical resistance or nonresistance to the procedure; 63% of those in the returnee group physically resisted deprogramming, while only 11% of nonreturnees did so ($p \leq .0001$).

Status of parental marriage significantly differentiated the groups ($p \leq .03$). The rate of intact parental marriages was significantly higher for the concerned member (91%), returnee (82%), and nonreturnee groups (78%) than for the voluntary ex-member group (50%).

The issue of becoming a deprogrammer differed significantly between the groups ($p \leq .0001$): 88% of the nonreturnees but none of the subjects in the other groups became involved in deprogramming activities.

Mental Status Exam Data and WAIS

All persons tested were normal on all aspects of the mental status examination. The groups did not differ significantly on the verbal, performance, or overall IQ scores. The ranges of group means on the IQ were verbal, 103–114 (overall verbal mean=111); performance, 114–120 (overall performance mean=116); and overall, 115–119 (overall mean=117).

Interpersonal Check List Data

The group data on the ICL dominance/submission variable were analyzed by one-way ANOVA, and significant main effects of group status ($p \leq .02$) and subject's self-image (before versus in cult) ($p \leq .03$) were found. There was a strong but nonsignificant trend toward interaction of these main effects ($p \leq .08$) (see figure 1). The returnees and concerned members were closely paired, viewing themselves as having become highly dominant while in the cult, whereas the voluntary ex-members saw themselves as much less dominant while in the cult, and the nonreturnees indicated they had become frankly submissive during their cult membership.

The group data on the ICL love/hate variable (see figure 2), also analyzed by one-way ANOVA, revealed a significant main effect of subject's self-image ($p \leq .001$) and a Self-Image × Group Status interaction ($p \leq .04$). The nonreturnee, returnee, and concerned member groups viewed themselves as having become significantly more emotionally affiliative while in the cult, whereas the voluntary ex-members saw themselves as almost unchanged on this variable.

Scoring for these two variables ranges from 10 to 90

FIGURE 1
Pregroup and In-Group Mean Scores on ICL Dominance Dimension

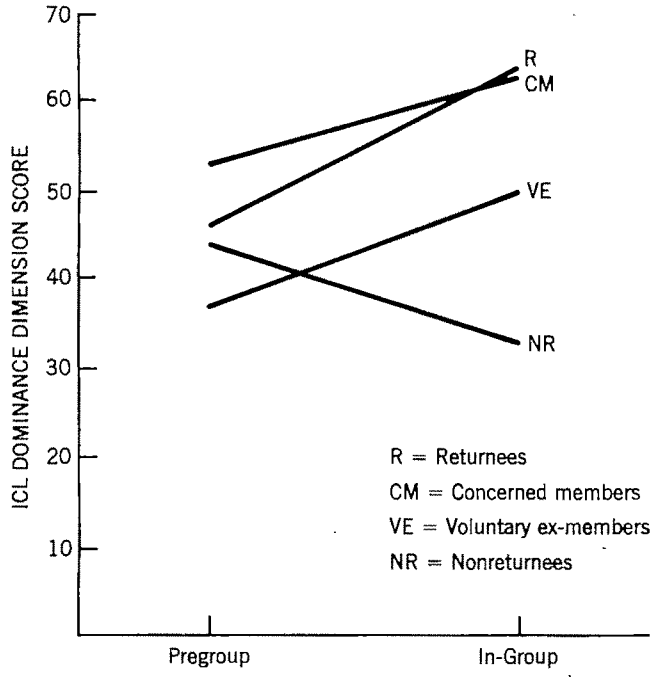
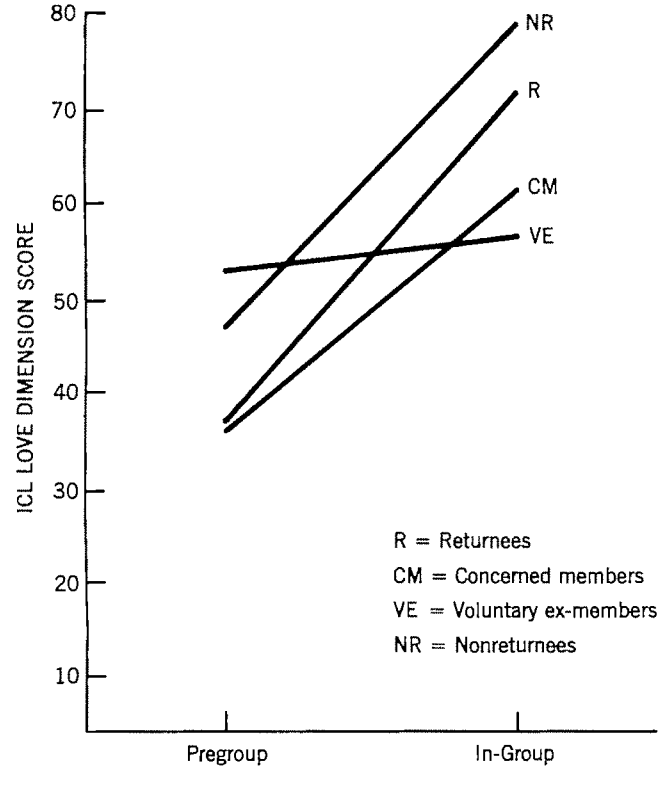


FIGURE 2
Pregroup and In-Group Mean Scores on the ICL Love Dimension



on the dominance/submission variable. Scores of 10–40 indicate a submissive interpersonal style, while scores of 60–90 indicate a dominant interpersonal style. Scores between 40 and 60 situationally reflect elements of both dominance and submissiveness. On the love/hate dimension, scores of 10–40 indicate a disaffiliative interpersonal style, while scores of 60–90 indicate an affiliative interpersonal style. Scores between 40 and 60 would situationally reflect elements of both affiliativeness and disaffiliativeness.

MMPI Data

The MMPI data were dealt with by collapsing the four groups into two: the concerned member and returnee groups were labeled "in" and the nonreturnee and voluntary ex-members were labeled "out." Both groups fell within the grossly normal range: the out group had peaks on scales 4 and 3 (Psychopathic-Deviancy and Hysteria), while the in group had peaks on scales 8 and 6 (Schizophrenia and Paranoia). It should be noted that the in group had an elevation on the Lie scale, which indicates 1) an intentional attempt to make a good impression and to deny faults ("fake good"), and 2) neurotic characteristics, including the prominent use of the defenses of repression and denial, coupled with a generalized lack of insight. Thus, it is likely that many of the in group's subscales would have been elevated had they not consciously skewed their responses.

The in group scored significantly higher on the Lie scale ($p \leq .02$). The out group scored significantly higher on the Paranoia scale ($p \leq .05$) and the Schizophrenia scale, with a strong but nonsignificant trend ($p \leq .09$) on the Psychasthenia scale.

Five research subscales were assessed between the two groups. The in group was significantly higher on the Psychopathic-Deviancy subscale 1 (anger at family members) ($p \leq .007$), and on Overcontrolled Hostility ($p \leq .05$), a subscale developed by Megargee (3, 4). A strong but nonsignificant trend ($p \leq .08$) emerged for the in group to score higher on the Alcoholism Vulnerability subscale developed by MacAndrew (5).

DISCUSSION

No data emerged from intellectual, personality, or mental status testing to suggest that any of these subjects are unable or even limited in their ability to make sound judgments and legal decisions as related to their persons and property. Most clear from our data was the finding that forcible removal of individuals from any group to which they have belonged for more than 12 months is exceedingly difficult.

Data from the MMPI delineate substantial intrapsychic and interpersonal differences between current cult members and those who have left. Subjects who were still cult members indicated difficulty with impulses in several areas and attendant superego deficits, such that the cults appear to serve as externalized superego substitutes. These difficulties were particularly strong in the area of hostility management. The trend toward concern over impulsive and dependent

use of alcohol and other drugs might be viewed as a reflection of frustrated oral dependency needs.

Test results indicate these impulses were dealt with by overconventionalism, repression, denial, and lack of insight. All of these can easily be seen to be incorporated into a lifestyle based within the cults. Those subjects who had left the cults were not free of psychological difficulties, and for this group social alienation, emotional alienation, and lack of ego mastery were dominant themes. We did not focus extensively on the nuclear family system of the cult member. This variable is a logical and critical next step in research on the core motivational aspects of the young religious cult member.

COMMENT

The groups all emerged as intellectually capable on testing. They revealed clinically an intellectual and philosophical bent that resembles what Lifton has termed "strong ideological hunger," regardless of their status in relation to the cult. These cults appear to provide, at least for a time, nourishment for these ideologic hungers as well as relief from the internal turmoil of ambivalence.

The ICL data indicated that those who tended to drop out without outside intervention appeared to have different motivations for joining the cults than did those who left the cults via outside intervention (deprogramming). It appeared that for those who left on their own, the needs were less intense for a safe, structured, predictable environment which would permit relatively conflict-free emotional affiliation with others. The other three groups appeared to join these religious cults, in part, out of intense needs for such structured opportunities to make emotional connections with others, which they had viewed as highly difficult before they joined the groups. The data did show, however, that the provision of a safe milieu for affiliation with others is not enough to hold all those who join cults. For those who left after deprogramming, the attraction of affiliation appeared to be counterbalanced by a rejection of the group based at least in part on a negative reaction to a sense of being dominated and forced into an unendurably submissive role. The fact that those who remained in the cults did not feel dominated and actually felt dominant may be partially explained by a cognitive-dissonance model. Their perceptions appear to differ from the reality that all of the cults function on a hierarchical model of power. This structure, by definition, excludes most members from major decisions or from positions of dominance.

The basis or etiology of the difficulties with hostility present an enigma not readily answerable by this study. There is some evidence, however, that points to the existence of hostility prior to joining the cults. The finding that those in the cults experienced hostile feelings toward their families far in excess of those who had left the cults is noteworthy. It is highly probable that the threat or actual experience of being deprogrammed increased the hostility. It would seem unlikely that the hostility stems entirely from the deprogramming issue and is totally unrelated to the pre-cult family process. It also seems equally unlikely that the overcontrolled, internalized hostility would exist only in regard to the family. The data suggest that the cult members might also feel hostile toward other authority figures, including cult leaders. The conscious realization or experience of hostility toward cult leaders would very likely be heavily repressed, or denied. Alternatively, this denied or repressed hostility might be projected onto figures outside the cults.

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Antipsychotic Effects of Lithium in Schizophrenia

BY PAUL E. ALEXANDER, M.D., DANIEL P. VAN KAMMEN, M.D.,
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The lithium carbonate therapy of 13 psychotic schizophrenic patients was evaluated in a placebo-controlled three-week study that was double-blind. Seven of the 13 patients were less psychotic while receiving lithium; 4 of these 7 patients relapsed after lithium withdrawal. Patients who improved during the third week on lithium could be differentiated from nonresponders on the basis of their improvement during the first week. Clinical factors such as diagnosis, prognosis, and symptoms failed to predict responders from nonresponders. To the authors' knowledge, this is the first controlled study to yield positive results with schizophrenic patients treated with lithium alone.

LITHIUM HAS proven efficacy in the treatment (1-4) and prophylaxis (5-9) of mania and depression, but it is generally believed to be ineffective in the treatment of schizophrenia (10-14). The history of lithium trials in schizophrenia began with Cade's classic paper (15) introducing lithium to psychiatry. He found that 3 of 6 patients with chronic dementia praecox who were treated with lithium showed a reduction in excitement; this work led to further trials, first with excited schizophrenic patients (16, 17) and later with excited schizoaffective patients (18-20). In all of these anecdotal reports some patients were found to improve on lithium.

Two controlled studies (14, 21) with excited schizoaffective patients yielded conflicting results. The first report (14) indicated that 6 of 11 patients worsened on lithium, with 5 showing organic brain syndromes; 4 other patients remitted or showed some improvement. High lithium blood levels, which ranged from 1.0 to 2.5 mEq/liter, probably had a role in the organic side effects. In a larger collaborative study with excited schizo-affective patients (21), lithium produced as marked an improvement in affective and psychotic behavior as chlorpromazine did in mildly active patients. Equivalent results with lithium and chlorpromazine were also found in the group of highly active patients who completed the lithium trial, but 35% of these patients could not complete the trial because of behavior problems. One patient terminated due to toxic confusion.

To our knowledge, lithium prophylaxis with schizoaffective schizophrenic patients has been reported in only one study, which was large, collaborative, and uncontrolled (7). The frequency of psychotic episodes was significantly reduced in the lithium treatment group.

Clinical trials of lithium in patients with schizophrenic subtypes other than schizo-affective have yielded good results (22, 23). One placebo-controlled report (22) found that lithium combined with phenothiazines was more effective than phenothiazines alone in chronic schizophrenic patients. In an anecdotal report, atypical psychoses with paranoid, confusional, and periodic features were responsive to lithium (23).

We have found only one previous double-blind controlled study of lithium therapy with schizophrenic patients (13). As a group, the 11 patients treated with lithium in that study did not respond favorably, and 6 of them worsened, with central nervous system toxicity, despite blood levels of less than 1.3 mEq/liter.

One of the difficulties in reviewing the literature on lithium involves the diagnostic criteria for schizophrenia. Spitzer and associates' Research Diagnostic Criteria (RDC) for typical schizophrenia and schizo-affective schizophrenia have been developed recently (24) but have not been used in controlled lithium trials. Similarly, "good prognosis schizophrenia" (25) is a clearly defined subgroup that is indistinguishable in several prognostic and genetic characteristics from manic-depression; lithium has not been studied in a group of patients defined in this manner.

In this study the antipsychotic properties of lithium were examined in a double-blind placebo-controlled study with psychotic schizophrenic patients.

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At the time this work was done all the authors were with the Biological Psychiatry Branch, National Institute of Mental Health, Bethesda, Md., where Dr. Alexander was Clinical Associate; Dr. van Kammen is Chief, Unit on Neuropsychopharmacology; and Dr. Bunney is Chief of the Branch. Dr. Alexander is now Assistant Professor, Section of Psychiatry and Human Behavior, Division of Biology and Medicine, Brown University, Providence, R.I. Address reprint requests to Dr. Alexander, Chief, Intensive Treatment Unit, Butler Hospital, 345 Blackstone Blvd., Providence, R.I. 02906.

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TABLE 1
Characteristics of 13 Schizophrenic Subjects in Lithium Trial

Patient	Age (years)	Sex	Diagnosis	
			DSM-II	RDC[a]
1[b]	19	F	Acute	S/A-M
2[b]	24	F	S/A	S/A-M
3[c]	53	F	Chronic	S/A-M
4	31	F	Catatonic	S
5	32	F	Chronic	S/A-D
6	19	M	Chronic	S
7[b]	19	M	Acute	S/A-D
8	23	M	Catatonic	S
9[c]	34	F	Paranoid	S
10	21	M	S/A	S/A-D
11	21	M	S/A	S/A-D
12[b]	19	M	Catatonic	S
13[c]	28	F	Acute	S/A-D

[a]S=Schizophrenic; S/A-M=Schizo-affective, manic type; S/A-D=Schizo-affective, depressed type.
[b]Clinical improvement of more than 1.5 points during the third week on lithium compared with the drug-free week, with relapse after lithium withdrawal.
[c]Clinical improvement of more than 1.5 points during the third week on lithium compared with the drug-free week, without relapse after lithium withdrawal.

METHOD

Thirteen schizophrenic patients (7 women and 6 men) participated in the study. They ranged in age from 19 to 53 years old, with a mean age of 26. They were voluntarily hospitalized on a clinical research unit of the National Institute of Mental Health and gave written informed consent for this study.

The clinical diagnosis of schizophrenia, including *DSM-II* subtypes (26), was made by consensus of 3 psychiatrists familiar with the patient. Each patient was also evaluated by his or her therapist according to the 1975 edition of the RDC (24) (patients' therapists were not directly involved in the study). Furthermore, patients had to have four or more differential symptoms from the International Pilot Study of Schizophrenia (27).

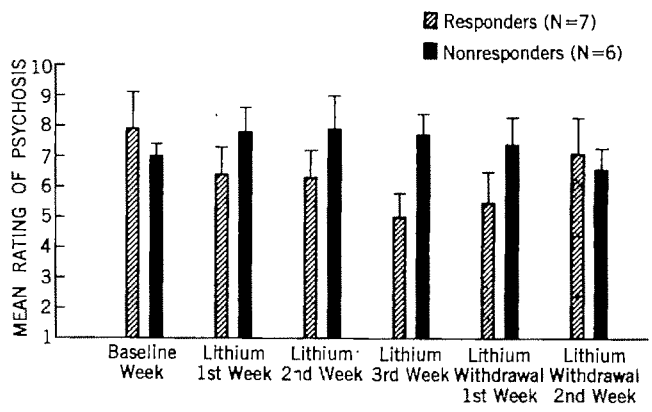
Patients were drug-free prior to the lithium trial; they took placebo capsules for at least 1 week before and 2 weeks after the 3-week trial of lithium.

Nursing staff, blind to drug, rated patients daily for psychosis using the modified 15-point Bunney-Hamburg Global Rating Scale (28).

Lithium carbonate was administered orally in divided doses, starting with a loading dose of 1800 mg for the first 3 days, followed by a lowering of the dose based on serum levels, side effects, and the clinical judgment of the physician responsible for medication. The dose during the third week averaged 1600 mg/day (range=1200 to 2800 mg/day). Serum levels during the third week averaged 0.9 mEq/liter (range=0.7 to 1.2 mEq/liter).

The mean ratings for the third week of lithium therapy were compared with those of the week prior to lithium as well as with those of the second week after withdrawal. Baseline ratings for psychosis ranged from 4.1 to 11.5 (mean=7.5). Responders showed a mean rating improvement of at least 1.5 points during the third week on lithium.

FIGURE 1
Mean Group Ratings of Psychosis for Lithium Responders and Nonresponders



The *DSM-II* schizophrenic subtypes of the patients were as follows: schizo-affective schizophrenia (N=3), acute (N=3), paranoid (N=1), catatonic (N=3), and chronic undifferentiated schizophrenia (N=3). Based on the RDC system, 5 patients were schizophrenic; 3 were schizo-affective, manic type; and 5 were schizo-affective, depressed type.

Patients were also evaluated by the following: age of onset, Phillips scale (29), Vaillant criteria (30), Schneiderian first-rank symptoms (31), the Modified Prognostic Scale (32), and the Taylor-Abrams prognostic criteria (25). The Modified Prognostic Scale consists of the following 4 items that Strauss and Carpenter found to be predictive of outcome: previous hospitalization, unemployment, poor social relations, and flattened affect. The Taylor-Abrams modification of the Robins and Guze criteria (33) was used with the original definition of sudden onset as less than 6 months.

RESULTS

Of the 13 patients in the trial, 7 showed clinical improvement during the third week on lithium (table 1). Four of these 7 responders relapsed within 2 weeks after lithium withdrawal.

The mean psychosis ratings of responders are compared with the ratings of nonresponders in figure 1. The means of the 2 groups were not significantly different during the drug-free baseline week. On lithium the mean psychosis ratings of responders decreased during the first week and decreased further during the third week. The rating during the third week on lithium improved from a baseline mean of 7.9 to a mean of 5.0. After lithium withdrawal the responders showed a progressive deterioration to a mean rating of 7.1. Nonresponders showed minimal changes during lithium treatment and withdrawal.

Figure 1 presents the individual data for both responders and nonresponders. Responders included pa-

FIGURE 2
Mean Individual Ratings of Psychosis for Lithium Responders and Nonresponders

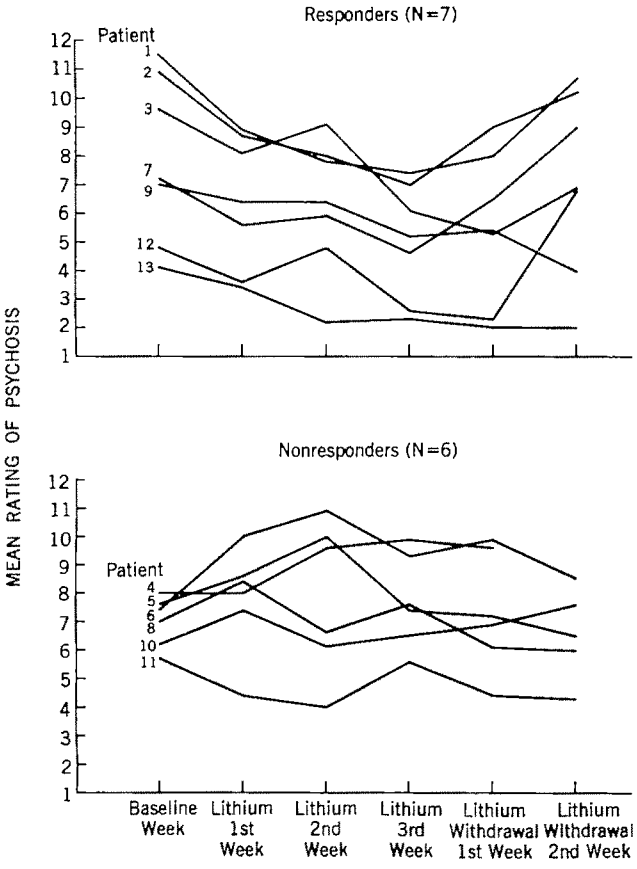
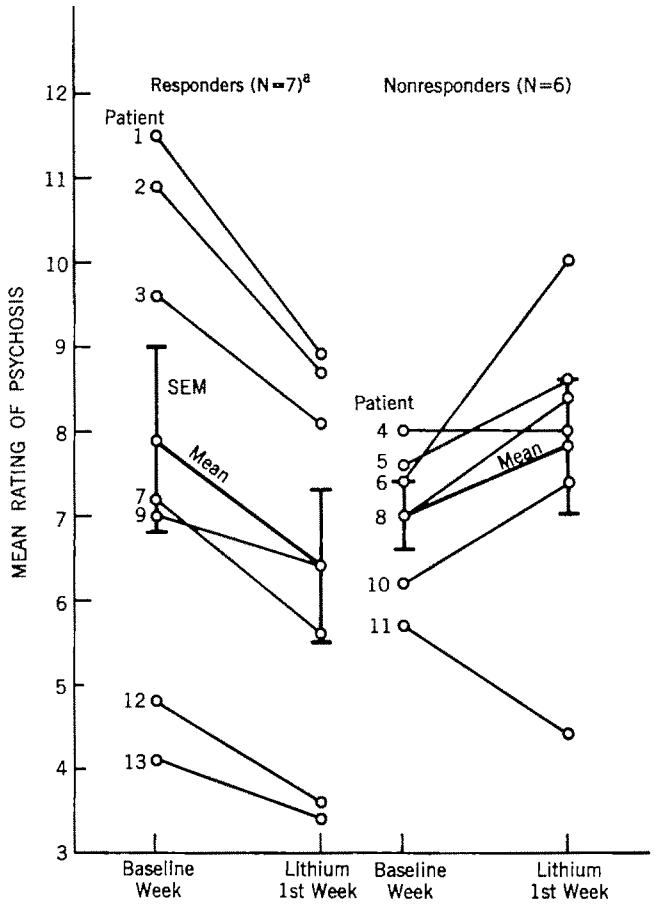


FIGURE 3
Mean Ratings of Psychosis During the First Week for Lithium Responders and Nonresponders



[a]Lithium responders improved significantly more during the first week ($p<.005$).

tients with the following *DSM-II* diagnoses: schizophrenia, schizo-affective, chronic undifferentiated, paranoid, catatonic, and acute schizophrenic episode. The RDC diagnoses of the responders included schizophrenia, schizo-affective, manic type, and schizo-affective, depressed type (table 1).

The data in figure 2 suggest that improvement occurred in responders within the first week. This effect is shown in more detail in figure 3. All responders showed some improvement during the first week from a baseline mean rating of 7.9 to 6.4. Only 1 of 6 nonresponders showed improvement during the first week. Differences between the two groups in improvement during the first week were found to be highly significant ($p<.005$).

Responders to lithium were compared with nonresponders on the basis of several criteria (see table 2). Both groups were similar in all factors examined, including age of onset, prognostic scales, and symptoms of schizophrenia.

DISCUSSION

Psychotic patients of diverse schizophrenic subtypes participated in a double-blind placebo-controlled trial of lithium carbonate. Slightly more than half of the patients showed clinical improvement in psychosis during lithium treatment. A majority of these responders relapsed within two weeks after lithium withdrawal. The improvement was modest (mean rating change of 2.9 on the Bunney-Hamburg scale), and no patients showed a complete remission of psychotic symptoms. However, the study provides evidence that lithium has antipsychotic as well as antimanic and antidepressant properties.

To our knowledge, this is the first controlled study to yield positive results with schizophrenic patients treated with lithium alone. The findings confirm anecdotal reports of lithium efficacy in schizophrenia (23, 34) but are at variance with the only other controlled lithium study of acutely psychotic patients. In that study Shopsin and associates (13) reported no improvement in any patients and deterioration in about half of the sample. In contrast, in the present study lithium did not appear to aggravate psychotic symptoms. The only patients whose psychosis ratings were higher during the third week on lithium (patients 5 and

TABLE 2
Similarities Between Responders and Nonresponders to Lithium

Item	Responders (N=7) Mean	Responders (N=7) Range	Nonresponders (N=6) Mean	Nonresponders (N=6) Range
Age of onset	20	12–28	20	16–26
Phillips scale score	14.1	6–22	15.2	10–23
Vaillant prognostic scale score	3.7	0–6	3.8	2–6
Modified Prognostic Scale score[a]	8.1	2–12	9.0	5–15
Number of patients with good prognosis schizophrenia[b]	4		2	
Number of patients with Schneiderian first-rank symptoms	6		5	
Number of IPSS[c] differential symptoms	5.7	4–7	7.2	6–9

[a]Modified Prognostic Scale of Strauss and Carpenter (MODPROG) (32).
[b]Modification of Taylor-Abrams criteria (25).
[c]International Pilot Study of Schizophrenia (27).

6) were not affected by lithium withdrawal, suggesting that their clinical changes were independent of lithium. Neurotoxicity, which was frequent in the previous study, was not observed in any patients in our study. The most likely explanation for the difference in the two studies is the patient population. Quitkin and associates (34) have suggested that patients in the earlier study may have been "asocial schizophrenics" who had central nervous system dysfunction and were prone to toxicity to any psychotic agent. Other lithium studies with schizophrenic patients have not reported an unusual sensitivity to lithium toxicity (3, 22, 23). Our data thus suggest that schizophrenic patients are neither worsened by lithium nor more sensitive to neurotoxicity.

The findings suggest that the clinical efficacy of lithium is not disease-specific for manic-depressive illness, as others have suggested (12, 14, 35). The patients in the study were diagnosed as schizophrenic by 3 psychiatrists and met several criteria prior to the lithium trial. Schizophrenic features predominated over affective symptoms in patients who met the RDC criteria for schizo-affective illness, and 5 of 8 patients diagnosed as schizo-affective by RDC criteria carried diagnoses other than schizo-affective based on *DSM-II* criteria.

In contrast to the generally slow improvement with neuroleptic drugs, the data suggest that lithium responders show early signs of improvement in the first week (figure 3). All 7 responders showed improvement in ratings in the first week on lithium; 5 of 6 nonresponders showed a worsening or no change during the first week. This rapid improvement suggests the feasibility of performing relatively short clinical trials of lithium.

The magnitude of the responders' improvement in the first week was also examined in relation to the magnitude of the improvement during the third week on lithium. Linear regression analysis of the improvements during these two periods was found to be directly proportional ($r=0.94$, $p<.01$). That is, the patients with the greatest improvement during the first week showed the greatest improvement during the third week relative to the baseline week. This preliminary finding suggests that the response within the first week may predict later outcome on lithium.

The response during the first week was the only predictive factor found in the study. The responders and nonresponders showed no prospective clinical differences that could be detected in our study. In fact, the similarities between the two groups were striking (see table 2).

A previous study of lithium efficacy in patients with primary affective illness (36) suggested that serum calcium (Ca) and magnesium (Mg) were of predictive value. In that study, a high ratio of drug-free Ca/Mg was predictive of positive outcome, as were increases in Ca and Mg during the first 5 days of treatment. These measures were also used in our study. Serum Ca and Mg, drawn during the baseline drug-free week at 8:00 a.m., were measured by atomic absorption spectroscopy (37) at the clinical chemistry laboratory of the NIH clinical center. Our data showed a trend level of significance ($p=.08$, Fisher's exact test) for the drug-free ratio Ca/Mg as a predictor of responsiveness to lithium in schizophrenic patients. Responders had a higher ratio than nonresponders during the drug-free periods ($2.70\pm.05$ mEq/liter versus $2.60\pm.05$ mEq/liter). The increase in Ca and Mg during the first week was found in the large majority of patients, regardless of responsiveness (10 of 11 patients had an increase in Mg and 11 of 12 patients had an increase in Ca). The increase during the first week for all patients on lithium compared with the drug-free week was significant for both Ca ($p<.005$) and Mg ($p<.025$) by paired t tests. The findings thus support prediction by drug-free Ca/Mg but not by the increase in electrolyte values during the first week.

We urge caution in applying our results to clinical practice. The possible clinical usefulness of lithium in schizophrenia needs to be evaluated in the context of the preliminary nature of our findings and the well-established efficacy of neuroleptic drugs. The current study involved a small number of patients treated with lithium for 3 weeks. It is important to emphasize that, although lithium had antipsychotic effects, it did not lead to a complete remission of symptoms in any patients in a manner similar to optimal neuroleptic response. Lithium has never been shown to be more effective than neuroleptics in treating schizophrenic patients as a group. Clearly, neuroleptics remain the medication of choice for schizophrenia. However, lithium may serve as an alternative medication for 3 groups of patients: those who do not respond to neuroleptics, those who cannot tolerate neuroleptic side effects, and those with medical conditions that contraindicate the use of neuroleptics.

Further controlled clinical trials of lithium in the treatment of schizophrenia are indicated.

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Parental Criminality and Medical Histories of Delinquent Children

BY DOROTHY OTNOW LEWIS, M.D., SHELLEY S. SHANOK, M.P.H., AND DAVID A. BALLA, PH.D.

In clinical practice the authors observed that many of the delinquent children of psychiatrically impaired and/or criminal parents had remarkably adverse health histories. To test their clinical impression they systematically compared the medical histories of 20 delinquent children with criminal parents and 85 delinquent children with less obviously maladapted parents. They found a strong association between paternal criminality and serious medical problems in these delinquent children. On the basis of this finding the authors suggest that the behavioral manifestations resulting from such physical illness or neglect, although they appear genetic in nature, may reflect the physical and psychological consequences of parental neglect and/or battering.

THE PURPOSE of this paper is threefold: 1) to report the differences in medical histories between delinquent children with criminal parents and delinquent children with noncriminal parents, 2) to document the especially strong association between paternal criminality and serious medical problems in delinquent children, and 3) to explore some of the psychological, behavioral, and physical implications of these findings. In a previous study (1) we reported more serious and more numerous medical problems in a sample of delinquent children compared with a matched sample of nondelinquent children. We have also reported an association between criminality and serious psychopathology in the parents of delinquent children (2, 3). We wondered whether differences existed within the delinquent population itself between the medical histories of children with criminal parents and children with less overtly maladapted parents.

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THE LITERATURE

We consider parental criminality not only a measure of social maladaptation but also an index of parental psychopathology that is especially hazardous to the development of offspring. There is strong evidence in the literature that criminality and psychiatric disorder are often closely related.

Penrose (4) noted that before World War II European countries with large prison populations had small mental hospital populations and vice versa. Gunn and associates (5, 6) reported that approximately one-third of the prisoners they interviewed had abnormalities in their mental state at the time of the interview. Our own studies of the parents of delinquent children (2, 3) indicated an association between parental criminality and parental psychopathology.

Most studies of parents and children have focused on the relationships between the psychiatric states of children and parental psychiatric or physical illness rather than parental criminality. Rutter (7) found psychiatric illness to be three times as common in the parents of children in a psychiatric clinic as in the parents of children in a pediatric or dental clinic. However, Sterne (8) reported that parental psychiatric illness was less closely associated than parental physical illness with children's serious antisocial behaviors. Waring and Ricks (9) reported an association between paternal and maternal psychopathology and schizophrenia in children.

Some investigators have focused on differences between the effects on children of paternal compared with maternal psychopathology. Wolff and Acton (10) reported maternal neurosis to be more closely related than paternal neurosis to children's emotional disorders. West (11) reported an association between maternal instability and serious behavior problems in male children. Mednick and Schulsinger (12) found significantly more mental illness requiring hospitalization in fathers of the psychiatrically disturbed offspring of schizophrenic mothers than in fathers of the well-functioning offspring of schizophrenic mothers.

Landau and her colleagues (13) conducted one of the few studies focusing on the physical as well as the mental states of offspring of seriously disturbed parents. They reported not only more undisciplined behavior at home and at school, more physical aggression against peers, and more delinquency in the off-

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spring of psychotic parents but also poorer health histories in the children of psychotic parents compared with those of nonpsychotic parents. Although behavioral problems in the children of psychiatrically disturbed parents have been noted in many offspring studies, rarely has there been a focus specifically on documented delinquency. Furthermore, to the best of our knowledge, the question of parental criminality and the medical status of delinquent offspring has not been addressed.

Studies of the offspring of antisocial parents have, for the most part, focused on the transmission of deviant behaviors rather than on medical histories. They have been predominantly psychodynamic (14), genetic (12, 15), or sociological (16–18). Health problems of the offspring of antisocial parents have generally been ignored. The few studies concerned with the physical health of delinquent children have been inconclusive (19, 20), and those indicating an association between poor physical health and delinquency have not usually included studies of the parents of the delinquent children.

CLINICAL OBSERVATIONS

Our own investigation of the possible association of severe parental maladaptation and serious medical problems in delinquent children sprang from the clinical observation that many of the delinquent children of psychiatrically impaired and/or criminal parents had remarkably adverse health histories. For example, one psychotic mother of a seriously delinquent boy had broken all of his fingers at different times. This mother was known to have been psychiatrically hospitalized. Another mother of a depressed epileptic girl had frequently thrown the child around by the hair and banged the child's head against the wall. One psychotic father, on discharge from a psychiatric hospital, threw his infant son across the room like a basketball. At other times he burned his children with cigarette butts.

Many children of criminal parents have suffered serious injury not directly inflicted by the criminal parent. One child who fell from a second-story window had a father who had been jailed for shooting off guns in the house. Another child who had fallen from a third-floor window had a violent father who had been incarcerated as a youngster.

We also found that the delinquent children of seriously disturbed parents often made many visits to hospitals for a variety of apparently minor illnesses as well as for accidents and injuries. Clinically, it looked as though the delinquent children of disturbed, violent parents had especially adverse medical histories.

These kinds of clinical observations led us to undertake a more systematic comparative study of the medical histories of delinquent children with criminal parents and the medical histories of delinquent children with less obviously maladaptive parents.

METHOD

Subjects

We selected a random sample of 109 delinquent children from the total number of referrals to a juvenile court in the early 1970s. A list of the name, birthdate, race, and place of residence of the child and the child's parents was compiled. The medical records of 4 of the 109 children were unavailable.

Measures of Parental Criminality

The list of parents was cross-checked with the central files of the state police to determine documented parental criminality. Issues of case loss arising in such research strategy have been discussed extensively elsewhere (2, 21). Twenty of the 105 children whose medical records were available had criminal parents; 85 did not.

Measures of Medical Status

We evaluated the medical history of each child in the study on the basis of an extensive review of his or her hospital record in the major general hospital serving the area. Approximately 95% of the children had used this hospital. Few of the children in the sample (about 25%) had used the one other smaller hospital serving the area. Furthermore, data from that hospital could not be retrieved with accuracy at the time of the study.

The decision to limit our study to one main large general hospital raises issues of case loss. Although it would have been desirable to interview parents and children regarding medical events, such personal contact was prohibited by the court. There is evidence that data based on parental recall are often inaccurate (22). Data from other public and private medical facilities were unavailable because of issues of confidentiality. However, the hospital selected tended to be used by the less affluent in the community as a source of primary care. In fact, the hospital subsequently developed a primary care service to meet the needs of the many patients who used the clinics and emergency room in this way. Furthermore, serious accidents and illnesses would most likely have been referred to this hospital, making hospital use for such problems an especially accurate reflection of incidence.

The date and the reason for each hospital visit were recorded. These data were later categorized as follows: total number of visits through age 16, total number of visits before age 12, total number of visits before age 4, total number of accidents and injuries through age 16 and before ages 12 and 4, head or face injuries through age 16 and before ages 12 and 4; and respiratory infections through age 16 and before ages 12 and 4. We also took note of any reference to child abuse in the medical record.

The number of and reasons for hospital visits through age 16 provided an overall picture of a child's medical status. Our interest in hospital contacts before

TABLE 1
Number of Hospital Contacts and Nature of Hospital Visits for Delinquent Children of Criminal and Noncriminal Parents

Item	Mean Number of Visits: Children with Criminal Parents (N=20)	Mean Number of Visits: Children with Noncriminal Parents (N=85)	t[a]	Significance
Accidents or injuries				
Before age 4	0.750	0.247	2.363	p=.021
Before age 12	1.850	0.976	1.974	p=.052
Through age 16	2.300	1.812	0.800	n.s.
Head or face injury				
Before age 4	0.300	0.106	1.610	n.s.
Before age 12	0.750	0.424	1.433	n.s.
Through age 16	0.800	0.553	0.972	n.s.
Respiratory illness				
Before age 4	0.950	0.471	1.087	n.s.
Before age 12	1.850	0.718	1.405	n.s.
Through age 16	1.950	0.976	1.182	n.s.
Total hospital visits				
Before age 4	6.800	2.400	2.682	p=.009
Before age 12	12.600	5.918	2.373	p=.020
Through age 16	15.900	8.965	1.943	p=.055

[a]Two-tailed t test.

TABLE 2
Number of Hospital Contacts and Nature of Hospital Visits for Delinquent Children of Criminal and Noncriminal Fathers

Item	Mean Number of Visits: Children with Criminal Father (N=15)	Mean Number of Visits: Children with Noncriminal Father (N=90)	t[a]	Significance
Accidents or injuries				
Before age 4	0.933	0.244	2.923	p=.005
Before age 12	2.067	0.989	2.179	p=.030
Through age 16	2.667	1.778	1.304	p=.195
Head or face injuries				
Before age 4	0.400	0.100	2.243	p=.028
Before age 12	0.933	0.411	2.065	p=.042
Through age 16	1.000	0.533	1.662	p=.10
Respiratory illness				
Before age 4	1.267	0.444	1.675	p=.097
Before age 12	2.467	0.678	1.996	p=.049
Through age 16	2.600	0.922	1.833	p=.070
Total hospital visits				
Before age 4	7.600	2.511	2.770	p=.007
Before age 12	14.000	6.056	2.523	p=.014
Through age 16	18.267	8.956	2.343	p=.022

[a]Two-tailed t test.

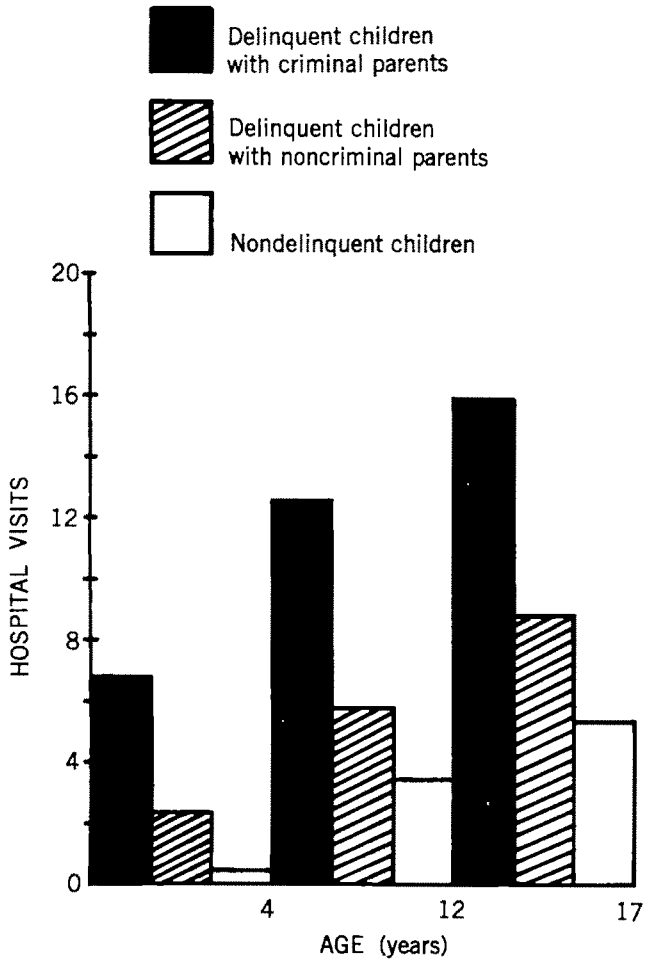
age 4 reflected the consensus in the literature that early physical illness or trauma may have important consequences for future adaptation (23-25). Our study of patterns of hospital use before age 12 sprang from the fact that most delinquent acts occur between ages 12 and 16. Thus, early recognition of certain health patterns and family constellations associated with delinquency might have implications for early intervention and, we hoped, delinquency prevention.

FINDINGS

The mean number of hospital contacts and the nature of hospital contacts over time for the delinquent offspring of criminal parents and for the delinquent offspring of noncriminal parents are presented in table 1. As this table shows, the delinquent children of criminal parents had more hospital visits than did those of noncriminal parents before age 4, before age 12, and through age 16. Of special interest is the fact that the children of criminal parents had significantly more accidents and injuries before age 4 than did children of noncriminal parents. There was a tendency for child abuse to be noted more frequently in the records of delinquent children of criminal parents (20% of criminal parents had an abused child compared with 5% of noncriminal parents (χ^2=2.513, p=.113, Yates correction). Paternal criminality contributed most strongly to this difference (20% of criminal fathers had an abused child compared with 6.7% of criminal mothers).

The mean number and the nature of hospital contacts for children of criminal and noncriminal fathers are presented in table 2. The delinquent children of

FIGURE 1
Mean Number of Hospital Visits of 20 Delinquent Children with Criminal Parents, 85 Delinquent Children with Noncriminal Parents, and 109 Nondelinquent Children



criminal fathers had more hospital visits before ages 4 and 12 and through age 16 than did those of noncriminal fathers. Further, the children of criminal fathers had more accidents and injuries, head injuries, and (at a borderline level of significance) more respiratory illnesses before age 4 than did the children of noncriminal fathers. This same pattern of findings was evident before age 12.

In contrast to these findings, there were no significant differences in the medical histories of the delinquent children of criminal mothers compared with those of noncriminal mothers. When the medical histories of the 15 children of criminal fathers were compared with those of the 5 children of criminal mothers, no differences reached conventional levels of statistical significance because of the small sample sizes. Nevertheless, on each of the variables investigated, the children of criminal fathers had more adverse medical histories than did those of criminal mothers.

Using data from our previous study comparing the medical histories of delinquent and demographically similar nondelinquent children (1), we were able to compare the medical histories of delinquent children who had criminal parents, delinquent children who had noncriminal parents, and nondelinquent children. We found that although delinquent children had more serious medical histories than did nondelinquent children, delinquent children with criminal parents were at greatest medical risk. Paternal criminality contributed most heavily to the differences (see figure 1).

DISCUSSION

Clearly, the delinquent children of criminal parents in this study had more adverse medical histories than did the delinquent children of noncriminal parents. Whatever the measure—number of hospital contacts or nature of hospital contacts—the delinquent children of criminal parents fared extremely badly.

Among the delinquent children of criminal parents, those who suffered most from a medical point of view were the children of criminal fathers. The differences in medical histories between the children of criminal fathers and those of noncriminal fathers were most striking before the age of 4. This finding is particularly alarming because it indicates that physical trauma and medical illness occurred in the lives of these children at a crucial stage of development, when the CNS is growing rapidly. The effects of injury at this time are likely to have a lasting influence on a child's adaptation to school, peers, and society in general. Such CNS injury is often associated with hyperactivity, perceptual-motor disorders, and impulsivity, factors notable in the histories of many delinquent children (2).

Investigators who have observed an association between parental antisocial behavior and children's social deviance have looked primarily to psychodynamic and social learning theory (i.e., modeling) for explanations. Our clinical work suggests several other ways in which paternal criminality may affect both the adverse medical history and the delinquent behaviors of the children of criminals. First, many of the fathers with criminal records in our study were known to have severely beaten and/or tortured their children, causing CNS damage. The uncontrolled behaviors of many of these fathers (e.g., shooting guns out windows, driving recklessly while drunk) encouraged similar recklessness in their children, who then indulged in extremely dangerous behaviors resulting in further injury. Thus, the effects of modeling have physical correlates.

The children of criminal fathers, if not battered, are often neglected. These fathers, sometimes by choice, sometimes as a result of incarceration, are often not present in the home at all. We have documented the tendency of criminal fathers of delinquents to marry women who have been psychiatrically treated and/or hospitalized (2, 3). Therefore, when a criminal father is not in the home, his children are often left in the care of a seriously disturbed mother. Without adequate

nurturance and protection, these children are subject to many accidents, injuries, and illnesses. Of course, battering by distraught, overwhelmed, seriously psychiatrically impaired mothers is also a common finding in the histories of delinquent children.

Along with the prevailing psychodynamic and social learning theories of delinquency there has been a resurgence of interest in the possible genetic transmission of deviance (26–28). We would suggest that the transmission of impulsivity and serious antisocial behavior from father to son may have yet another explanation. The behavioral manifestations resulting from physical abuse or neglect may appear at first glance to be genetic in nature; in actuality they may reflect the consequences of parentally influenced injury

In summary, our clinical and epidemiological findings suggest that one of the ways in which paternal criminality affects children's deviant behaviors is through physical abuse or neglect that results in physical injury. This physical trauma, especially to the CNS at an early age, impedes a child's ability to perceive accurately, make sound judgments, and control impulses.

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Schizophrenic Thought Disorders: Bizarre Associations and Intermingling

BY MARTIN HARROW, PH.D., AND MEL PROSEN, M.D.

The authors assessed bizarre verbalizations elicited from 37 schizophrenic and 16 nonschizophrenic patients. Interviews with subjects indicated that much bizarre schizophrenic language results from patients intermingling material from past and current experiences into their verbalizations. This intermingled material comes from many different problem areas rather than one central emotional complex. It does not arise from emotional overresponsiveness, overinvolved thinking, or delusional ideation. Two factors hypothesized as responsible for bizarre schizophrenic language are the schizophrenic's monitoring problems and difficulty maintaining perspective about his own behavior.

BIZARRE AND DISORDERED THINKING are considered central features in schizophrenia (1-7). At present, their cause is unknown. New techniques are needed to study more directly what factors influence or lead to disordered thinking. We have developed one such technique and have obtained results about intermingling, a factor that may play a role in determining disordered language in schizophrenia.

In recent research, we have explored a formulation about the importance of intermingling in disordered language (8, 9). This formulation was based in part on previous findings of a strong relationship between overpersonalized responses and bizarre verbalizations (10). According to our hypothesis, one overt factor that leads to schizophrenic patients' disordered verbalizations is an intermingling into their language (and possibly into their thinking) of material from their past or current experience (8). We believe intermingling and other types of disordered language are influenced by a difficulty of the schizophrenic in maintaining perspective about, and monitoring, his own language and behavior such that he does not fully recognize when he is inappropriate (11).

In recent research we found positive evidence about the presence of intermingling as a factor in disordered language (8, 9). However, this research was based on a small sample and lacked a control group of nonschizophrenics. The present research had several goals. The first was to investigate intermingling in a larger sample and to compare findings with those from a control sample of nonschizophrenics. Second, we wished to use our technique to explore other factors that might influence intermingling or disordered thinking.

The specific questions on which we will focus in the present report are as follows:

1. Do nonschizophrenics' strange verbalizations reflect intermingling, as we found to be the case for many schizophrenics?
2. Does each schizophrenic's intermingling involve material from one central area of concern, or emotional "complex," or does it represent overinvolvement in a variety of different topics?
3. Does the schizophrenic's intermingling represent a tendency toward overelaborate verbalizations or overinvolved thinking?
4. Does the schizophrenic's intermingling represent interference with language by strong emotional reactions or emotional overresponsivity to the material with which he or she is dealing?
5. Does the intermingling reflect delusional or paranoid material that influences thinking?

METHOD

Sample

The subjects were 53 psychiatric inpatients (37 relatively young schizophrenics and 16 nonschizophrenics) from a multidisciplinary research project on schizophrenia (8, 12-14). The schizophrenic sample included 10 paranoid schizophrenics and 27 schizophrenics whose most prominent symptoms were not primarily paranoid. These patients also met the criteria for a schizophrenic diagnosis on the New Haven Schizophrenia Index (15). The nonschizophrenic

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sample consisted of 7 patients with affective disorders (5 depressed and 2 manic) and 9 patients with severe personality disorders. Only 3 of the 16 nonschizophrenic patients (the 2 manic patients and 1 depressive) showed strong evidence of psychotic thinking.

The mean age of 32 males and 21 females was 22.9 years. Almost all of the schizophrenics (91%) were taking phenothiazines or phenothiazine-like medications (e.g., haloperidol) at the time of assessment. The mean educational level of the sample was 12.4 years. The mean raw score on the WAIS Information Scale was 16.2, which falls within the average range of intelligence (16). There were no significant differences between the schizophrenic and nonschizophrenic samples on age, education, or intelligence.

Data Collection and Ratings of Intermingling

The overall research plan, which has been described more fully in previous reports (8, 9), involved three stages: 1) eliciting bizarre responses from each patient, 2) interviewing each patient about the reasons for his bizarre responses, and 3) rating the reasons for each patient's bizarre responses.

For the first stage, each patient was given the Proverbs Test (17) and the Social Comprehension Test (16) during an acute stage of his or her disorder. The patient's responses were later scored for idiosyncratic, bizarre verbalizations on a standardized scoring system reported previously (18, 19). Three accurate or near accurate responses and five idiosyncratic or unusual responses of each patient were selected for subsequent inquiry to him.

In the next phase, which occurred the following week, a senior psychiatrist conducted a standardized taped interview to explore the patient's reasons for and thinking involved in giving each of the eight responses chosen for analysis. The psychiatrist prefaced the interview by saying he was interested in greater detail about the patient's responses and in learning what the patient had in mind when he gave them and what types of things had led him to give these responses. This was followed by a series of structured questions for each response, such as "Do you remember what you were thinking of when you gave that answer?" "What led you to make that response?"

Third, the taped interviews were rated along standardized dimensions hypothesized to be underlying reasons for disordered thinking in schizophrenia. Thus, patients were rated according to whether their own specific bizarre responses were influenced by 1) faulty logic, 2) an intermingling of material based on their past or present experiences, 3) concrete thinking, 4) emotional overreactions to the questions, 5) delusional or paranoid ideation, etc.

Intermingling was considered present when there was overt evidence in the original response or the taped interview of the patient's blending material from his own experience into his response, or when the patient's answer was obviously guided or influenced by these experiences from the very beginning of the response.

The interviews and ratings also explored the possible presence of a number of factors that might be related to or influence intermingling or bizarre language, and these are reported in the current paper. Satisfactory interrater reliability was achieved for the ratings.

RESULTS AND DISCUSSION

Is Intermingling an Influence on Bizarre Language?

As noted above, intermingling is considered present when the patient blends into his response, or when the response is guided by, material from his past or current experiences that does not "fit" neatly and makes the response seem at least somewhat strange. Half (51%) of the schizophrenics showed strong evidence of overt intermingling and 27% showed milder evidence of intermingling. Overall, in a larger sample than we used in our previous research, the importance of intermingling in schizophrenic bizarre language was suggested. Strong evidence of intermingling was shown in 38% of the nonschizophrenics.

The data indicate that more of the schizophrenics than nonschizophrenics engaged in intermingling, but this difference was not statistically significant. Thus, the data suggest that the processes involved in intermingling are not unique to schizophrenia. They tend to be slightly more common in schizophrenics but also occur in many nonschizophrenics.

In previous research we found evidence that schizophrenics are not the only patients with thought pathology. Nonschizophrenics also show evidence of disordered language, although in nonmanic patients this is less frequent, less flagrant, and less severe (6, 11).

The data raise the possibility that intermingling material from one's own life is one of several general characteristics that might underlie many bizarre verbalizations, regardless of diagnosis. The issue here is whether whatever mechanisms are involved in idiosyncratic language in schizophrenia are also prominent factors for nonschizophrenics on those less frequent occasions when they do give idiosyncratic verbalizations.

Is Schizophrenic Intermingling Due to Preoccupation with One Central Problem Area or Emotional Complex?

The results in this area have some bearing on older theories that schizophrenic patients have one central "complex" or problem area and that preoccupation with this area dominates their lives, leading to emotional disturbance and possibly to interference and disruption of behavior.

The data indicate a clear trend for each schizophrenic's intermingling to be centered around a variety of topics or areas from his experience (three or more areas), rather than only one central problem area. This tendency occurred for 18 of the 25 schizophrenics who

showed evidence of intermingling (72%). Only 1 of 25 (4%) showed intermingling in only one area. Results were similar for nonschizophrenics.

Our results in this area were geared to answer a key question: is there one dominating problem to which the patient responds when he becomes psychotic, or does a vulnerable patient tend to respond strangely to almost any topic of concern that leads to idiosyncratic language? Our data suggest that one important factor in the schizophrenic's strange and idiosyncratic appearance is a tendency to intermingle overtly material from a variety of conflicts and concerns.

Possible Influences on Intermingling or Disordered Language

Overelaborate verbalizations and thinking. The results in table 1 reflect patients' tendency to give extensive and overelaborate verbalizations, which suggests elaborate sets of thoughts and possible overinvolved thinking about a topic. Dramatic examples of this feature in select patients have been noted by many clinicians.

Overelaborate thinking was not common in the schizophrenics (only 30% showed any signs thereof). It did appear, dramatically, in select patients. Detailed analysis indicated a tendency for overinvolved thinking to be associated with a smaller subgroup of paranoid schizophrenics.

These results indicate that overelaborate ruminative thinking is not characteristic of most early schizophrenics and is not an important mechanism in their intermingling or their bizarre behavior. However, a subgroup of early paranoid schizophrenics showed overelaborate verbalizations and thinking. Further exploration to determine whether overelaboration plays an important role for some paranoid schizophrenics might be profitable.

Strong emotional responsivity. Table 1 reports the results on strong emotional responsivity to the relatively neutral proverbs as an influence on patients' bizarre verbalizations. These data do not bear on whether or not the stimuli or emotional environment were emotionally laden. Strong emotional responsivity as an influence on disordered verbalizations tended to occur slightly more frequently among nonschizophrenics than schizophrenics (58% versus 30%), but this difference was not significant. Thus, strong emotional responsivity showed some influence on the nonschizophrenic patients' idiosyncratic language—although it usually was not the only or major influence—and relatively little influence on the schizophrenics.

Paranoid and delusional material. Delusions were very frequent among these young schizophrenics and may be an important influence on their overall behavior, but delusions usually were not the major determinant of their bizarre language (see table 1). In select, very dramatic cases, delusions were the major influence on the patients' bizarre verbalizations. These cases, however, were rare. Surprisingly, the influence of paranoid material on bizarre responses was not greater for paranoid schizophrenics than for nonparanoid schizophrenics. It must be remembered, however, that many nonparanoid schizophrenics also show some degree of paranoid ideation, although it is less frequent and less sustained.

TABLE 1
Patients with Disordered Language Influenced by Overelaborate Verbalizations, Strong Emotional Responsivity, and Delusional or Paranoid Ideation

Influences on Disordered Thinking	Schizophrenics (N=37)		Nonschizophrenics (N=16)	
	N	%	N	%
Overelaborate verbalizations or overinvolved thinking				
Very clear evidence	5	14	3	19
Minor evidence	6	16	0	0
No evidence	26	70	13	81
Strong emotional responsivity				
Very clear evidence	4	15	4	33
Minor evidence	4	15	3	25
No evidence	18	70	5	42
Unavailable for rating	11	—	4	—
Delusional ideation				
Very clear evidence	5	19	0	0
Minor evidence	3	11	0	0
No evidence	19	70	12	100
Unavailable for rating	10	—	4	—
Paranoid ideation				
Very clear evidence	7	19	2	13
Minor evidence	10	27	1	6
No evidence	20	54	13	81

Overall, almost all of the schizophrenics had delusional or paranoid ideas, and some paranoid material entered into the bizarre verbalizations of 46% of the sample. During the early stage, however, delusional or paranoid ideas were not the major factors in shaping the schizophrenics' bizarre idiosyncratic responses.

COMMENT

Intermingling and Disordered Language

The current investigation supports our previous research indicating that one immediate mechanism responsible for many young schizophrenics' bizarre verbalizations is a tendency to intermingle into their language or allow it to be guided by material from their personal experiences. At times the intermingling can often be found in nonschizophrenics who give strange verbalizations.

The results are not at variance with clinical observations and research demonstrating that schizophrenics' language contains features which can be viewed as bizarre, idiosyncratic, autistic, or disordered (1, 3, 7, 10, 19). However, the present data and our previous results (8) suggest that these strange verbalizations are based on rationales which make some sense in terms of patients' own experiences when looked at from their points of view.

Since nonschizophrenics typically show less

thought pathology, we would propose that personal experiences dominate their verbalizations less, and when they do appear they blend into or guide their responses in a more socially acceptable and better disguised fashion. For the nonschizophrenic, the compromise between adhering to the topic and expressing part of one's personal concerns is an imperfect one but is more closely attuned to the listener's probable reaction and the consensually correct subject of discussion.

Aspects of Intermingling and Its Determinants

Other information emerged about intermingling and the type of material involved. Delusional and paranoid ideation usually were not the major determinants of the schizophrenics' bizarre language. The material that intermingled into the patients' verbalizations and made them appear bizarre was not typically a result of overelaborate thinking or of overinvolvement with one central conflict.

The intermingled material usually came from several different personal experiences or conflicts for each schizophrenic. These concerns were not necessarily more serious than those experienced by nonschizophrenics. The major factor was the schizophrenic's tendency to blend the concerns into his or her verbalizations at times when their "fit" with the topics was poor. The intermingling of this material often shifted the schizophrenics' comments away from the consensual response, without any explanation to the listeners.

Perspective-Monitoring Ability and Disordered Language

What is the underlying factor that leads schizophrenics to intermingle material from their personal lives into their verbalizations? We would propose that one important influence involves executive processes, namely, the schizophrenic's difficulty in maintaining perspective about whether his own behavior is socially appropriate and monitoring his own language (perspective-monitoring ability). The normal person is typically able to maintain perspective (about what is socially appropriate) and to monitor his language such that his preoccupations do not enter into his verbalizations in a gross, bizarre manner. At times, however, these concerns do appear in the normal person's language in minor, partially disguised ways.

As formulated here, a prominent factor in schizophrenia is a disorder of that aspect of executive functioning involved in maintaining perspective about the social appropriateness of one's own behavior and monitoring it to conform to the social demands of the situation. At the time of his strange, idiosyncratic behavior the schizophrenic may have temporarily lost the ability 1) to maintain perspective about and recognize what is idiosyncratic to him versus what is appropriate in a social setting, and 2) to monitor his routine, minute-by-minute behavior such that his everyday concerns and wishes do not intermingle inappropriately into it. According to this hypothesis one would expect the schizophrenic to have better perspective about the social appropriateness of other patients' language than about his own, and we are currently undertaking research to assess this assumption. Nonschizophrenic patients and normals may also have some, but less, trouble in monitoring their verbalizations, leading to small amounts of cognitive slippage, especially during periods of stress or upset.

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Acupuncture Treatment of Schizophrenia: Report on Three Cases

BY JONATHAN KANE, M.D., AND WILLIAM J. DI SCIPIO, PH.D.

The authors conducted a 9-week blind controlled study of the effects of acupuncture on schizophrenic illness in three patients on an inpatient ward of a psychiatric hospital. The patients were used as their own controls. The authors compared the effects of acupuncture, pseudo-acupuncture (random needling), and no-treatment control periods. Two patients who had had florid schizophrenic symptoms responded positively to true acupuncture treatments and negatively to pseudo-acupuncture. The third patient, whose symptoms were primarily affective-depressive, showed no significant response to treatment. The authors explore the mechanisms thought to be involved in the etiology of schizophrenia, focusing on the cortical arousal hypothesis.

ACUPUNCTURE, along with Western chemotherapy and Chinese herbal medicine, is being used in mental hospitals in the People's Republic of China to treat schizophrenia and other mental disorders. The Chinese have published material listing acupuncture points used in the treatment of the mental disorders that they diagnose as we do. According to an NIH publication (1), a 1971 Chinese text lists diagnostic categories and criteria for mental disorders along with recommendations for treatment. For example, schizophrenia is diagnosed by "broken thoughts" (loosening of associations), "disharmonious feelings" (withdrawal and inappropriate affect), and "delusions and hallucinations." The book lists body and ear acupuncture loci for the treatment of schizophrenia along with instructions for needling and for giving phenothiazines.

An uncontrolled study conducted at the Hopei Psychiatric Institute in 1957 (2) found that of 30 schizophrenic patients given an average of 8.2 acupuncture treatments, 73.4% "responded best, while the poorer therapeutic responses were obtained in chronic patients." Bresler and associates (3), citing a report of a Norwegian medical group visiting the People's Republic of China, stated that a 74% improvement rate was achieved in schizophrenic patients treated by acupuncture and neuroleptics.

According to a personal communication from Jean-Claude Darras, M.D., former Director of Training, Institut du Centre d'Acupuncture de France, schizophrenia is rarely treated by needling in France, where acupuncture is more widely practiced than it is in other Western countries. However, Soulie de Morant (4) and Van Nghi (5) listed acupuncture points for neurotic as well as psychotic syndromes.

The purpose of the present paper is to present a clinically controlled study of the treatment of three schizophrenic patients by acupuncture in the absence of neuroleptic medication. The design of this study is intended to provide objective assessment of treatment versus placebo effects of acupuncture on a range of psychiatric symptoms occurring during a course of treatment, using each patient as his or her own control.

METHOD

Patient Selection

Consecutive admissions to a 35-bed coed general psychiatric ward of a large urban state psychiatric hospital were screened for possible inclusion in the study. The patients selected had to have one or more of the following psychiatric symptoms on interview: loosening of associations, flat or inappropriate affect, or any two Schneiderian first-rank symptoms. Informed consent was obtained.

All patients were told that although success with this form of treatment has been reported in Eastern countries, no systematic studies were available in the United States. If the patient requested termination of the acupuncture procedure at any time, the treatment regimen was changed to more traditional Western approaches.

Design

Treatments were administered by the ward psychiatrist (J.K.) each weekday in individual 45-minute sessions. The points used are described in a Chinese publication (6). Two sets of points were used in alternation on successive days. The first set was the large intestine

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4, the liver 3, and the governing vessel 26. The second set was the *T'ai-Yang*, the pericardium 6, and the spleen 6. After one week of needling, the following points were added to both sets: the governing vessels 13, 14, and 15 and the small intestine 19. Patients nearly always reported feeling something like an electric current flowing when the needles were placed in the true meridian points. This phenomenon is known as *te Ch'i* and is considered good evidence that the needle is in the correct position.

A pseudo-acupuncture procedure consisted of selecting placebo locations close to the true points but not corresponding to any known meridian, extraordinary, or "new" points. No feelings of *te Ch'i* were described by patients when these placebo locations were used. The equivalent type and number of needles, twirling time, and overall session times were maintained to correspond to the true acupuncture periods. Routine blood tests, urine analysis, and EEGs were within normal limits for all three patients. The no-treatment control periods consisted of scheduled equivalent-time individual sessions with the psychiatrist to maintain a concerned doctor-patient relationship without physical contact.

We used a within-subjects design with each patient acting as his or her own control. Each patient included in the study was rated by means of the Psychotic Reaction Profile (PRP) (7) for a baseline measurement after spending several days on the ward. Weekly PRP measurements were taken for a total of nine weeks. The baseline period lasted five days, and all other treatment and placebo periods were of two weeks' duration. Each patient received the following sequence of treatments staggered among patients: no treatment (baseline), acupuncture, no treatment (control 1), pseudo-acupuncture, and no treatment (control 2). The order of treatment was not randomized because the small sample size did not lend itself to statistical testing of an order effect.

Ward staff were aware of the use of acupuncture in the study but were blind to the design sequence. In addition, neither patients nor staff were aware of the difference between true acupuncture and pseudo-acupuncture procedures.

Measurement

The PRP is a rating scale designed for ward staff administration. It consists of 85 true-false items summarized by four scales: withdrawal, thinking disorganization, paranoid belligerence, and agitated depression. The PRP was rated independently by three paraprofessionals at least weekly. Two of these paraprofessionals were assigned to the day shift and one to the evening shift. Interrater reliability coefficients for the two day staff members were higher than coefficients between day and evening staff. The lower correlations between day and night staff are attributable in part to the different observation periods. To gain an accurate estimate of overall patient behavior, all three PRP raters' scores were averaged for each week for the purpose of the analysis (8). Averaging was necessary because on some occasions only one or two of the raters were present, according to their weekly staffing schedules. A two-way analysis of variance of patient differences, treatment differences, and interaction of these two independent variables was applied to each of the four PRP scales. The minimal acceptable level of statistical significance was .05.

CASE REPORTS

Case 1. A was a 16-year-old high school student who had seen the film *The Exorcist* about two months before admission. After seeing the movie he became more "nervous" than usual. For about a week before admission he refused to go to school. Several days before admission he began to develop florid delusions that the Devil was going to get him. He began talking to himself and felt that he was a policeman. On the day before admission he hallucinated that a giant fly was coming to kill him. He attacked his brother with a knife, causing some superficial throat wounds.

On admission A was fully oriented and showed no loosening of associations. He was withdrawn and guarded in his responses during the admission interview. His affect was inappropriately flat, particularly when he related the series of incidents described above. His history revealed that he had always been a loner and had no girlfriends. At school he could not pay attention to his work and usually engaged in grandiose fantasies about space travel. One year before this admission he had been referred by the school to a psychiatrist, who placed him on thioridazine for an unspecified period of time. The patient reported that his problem related to the fact that he was "bored" with school.

A was diagnosed as having schizo-affective schizophrenia (depressed). His baseline PRP values were as follows: withdrawal, 72nd percentile; thinking disorganization, 68th percentile; paranoid belligerence, 45th percentile; and agitated depression, 76th percentile.

Case 2. B was a 25-year-old single man who was admitted after becoming highly agitated the night before, when he walked home carrying a large cross and saw visions of God and the Devil. He disrobed at home and tried to put his mother into the oven because he thought she was Hitler and the Devil. On admission B was highly agitated and was experiencing auditory and visual hallucinations with religious and rebirth themes. His affect was flat, and he spoke in a monotone. He was fully oriented but had severe loosening of associations. Two months before admission this patient had also seen *The Exorcist*. At that time he started to have insomnia and began to keep a light on when he went to bed. On the day before admission the patient had gone to the cemetery for the unveiling of his father's tombstone.

This was B's first admission. He was diagnosed as having acute schizophrenia. His baseline PRP values were as follows: withdrawal, 32nd percentile; thinking disorganization, 95th percentile; paranoid belligerence, 82nd percentile; and agitated depression, 35th percentile.

Case 3. C was a 45-year-old single woman who was admitted at her father's request after she pulled down his pants to check his prostate "to see if he's a real man." She said that she wanted to come to the hospital because it was "cold at

home." On admission C was grossly psychotic with delusions of a man who would marry her and with whom she would have a baby. (This delusion apparently involved a man who had called her once many years ago.) C showed loosening of associations, was extremely withdrawn and guarded, and responded to hallucinated voices. Her affect was inappropriate with frequent out-of-context chuckling. Her eyes looked wild and glazed, she exhibited bizarre hand mannerisms, and she walked in a stiff, stork-like fashion.

C had been hospitalized many times during the past three decades, living at home with her parents in the intervening periods. Her last psychiatric hospitalization had been four years before this one. She was diagnosed as having chronic paranoid schizophrenia. Her baseline PRP values were as follows: withdrawal, 93rd percentile; thinking disorganization, 80th percentile; paranoid belligerence, 49th percentile; and agitated depression, 93rd percentile.

RESULTS

Case 1

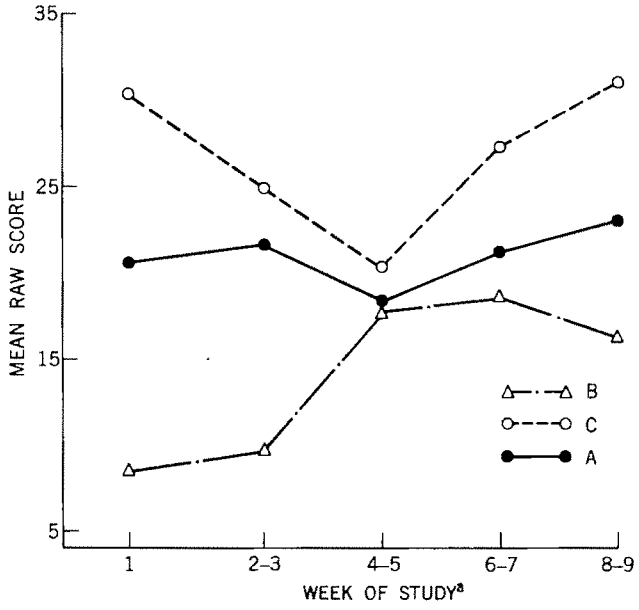
Of the three patients, A seemed to respond the least dramatically to acupuncture treatment. His hallucinations cleared before any of the needling was performed and did not return during the course of the study. During the period of true acupuncture treatments he appeared to be more cooperative, more animated, and less withdrawn. On several occasions immediately following true acupuncture he indicated that he felt calmer and said with a smile that he was no longer bored. During the periods without acupuncture he did not seem to change at all with regard to withdrawal or other symptoms. No change was noted during the pseudo-acupuncture period with the exception that on several occasions he reported feeling more tense.

A's lack of response to treatment is shown in the results of three PRP scales (see figures 1, 2, and 3). Although the agitated depression scale appears to demonstrate a consistent decline in pathology (see figure 4), caution must be used in interpreting this scale because it is the least reliable of the four scales (7). Furthermore, no significant treatment effects emerged for this scale.

Case 2

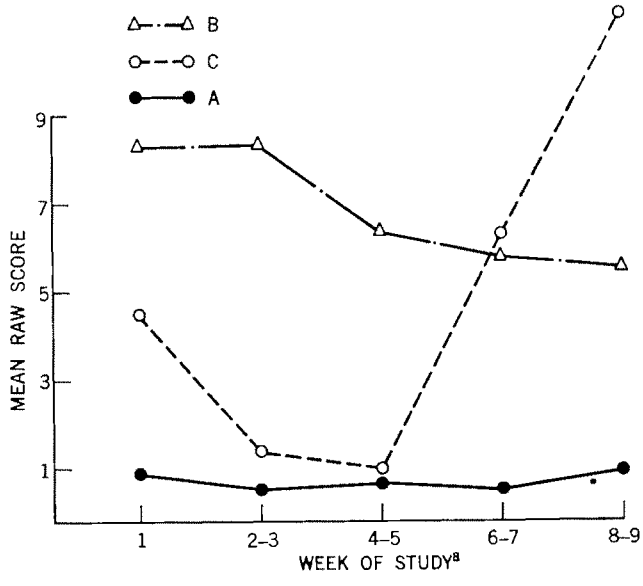
During the several days before the acupuncture treatment B was often placed in the isolation room because of disruptive behavior that was not controllable by staff intervention. As soon as true acupuncture treatments began, the amount of time the patient spent in the isolation room was markedly reduced. During the control 1 period and especially in the second week of this period the patient began leaving the ward without permission and was again placed in the isolation room, although not as often as he had been during the first weeks of admission. During the needling periods, when the patient was asked in a nondirective way how he felt at the end of a session his typical response following a true acupuncture treatment was that he felt calm or relaxed. His responses following pseudo-acupuncture sessions were typically that he felt the same

FIGURE 1
Mean PRP Withdrawal Scores for All Three Patients



[a]Week 1=baseline; weeks 2–3=acupuncture; weeks 4–5=control 1; weeks 6–7=pseudo-acupuncture; weeks 8–9=control 2.

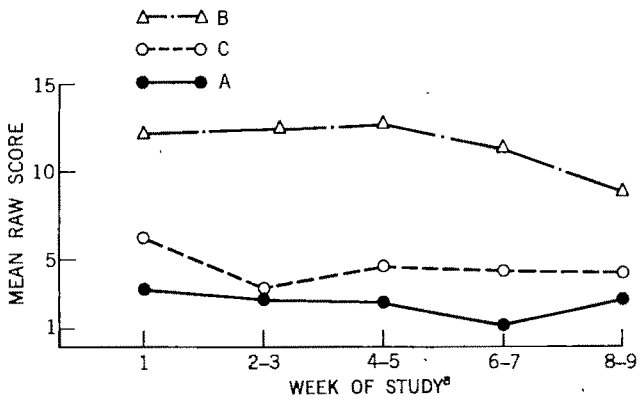
FIGURE 2
Mean PRP Paranoid Belligerence Scores for All Three Patients



[a]See footnote to figure 1.

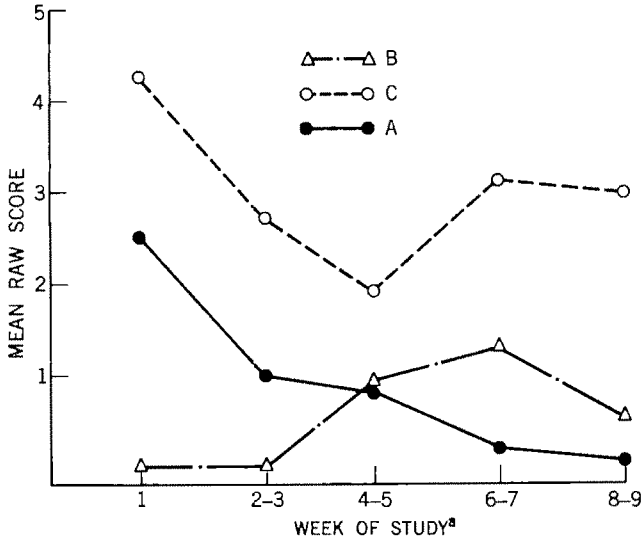
or worse. Toward the end of the second week of pseudo-acupuncture he refused to continue needling treatment. He was then given chlorpromazine, starting at 150 mg t.i.d. and increasing to over 2000 mg within a month. During this month and the following two months there was no noticeable change in the degree of agitation, thought disorder, or hallucinations. It seemed that the behavioral changes occurring during

FIGURE 3
Mean PRP Thinking Disorganization Scores for All Three Patients



[a]See footnote to figure 1.

FIGURE 4
Mean PRP Agitated Depression Scores for All Three Patients



[a]See footnote to figure 1.

the acupuncture period were more pronounced than those during the pseudo-acupuncture and chlorpromazine periods.

The changes in the withdrawal scale for B showed a particularly labile picture (see figure 1). Following true acupuncture his scores increased considerably throughout the control 1 and pseudo-acupuncture periods. They then began to drop off during the control 2 period. The main treatment effect (a measure of pooled patient results among experimental periods) was significant. The interaction effect (patients by treatments) approached significance for this scale ($p<.10$). A planned comparisons test revealed that acupuncture versus pseudo-acupuncture, acupuncture versus control 1, and control 1 versus control 2 were each significantly different. Although these results represent pooled patient effects, B's main score changes were particularly large in magnitude, showing an 86% increase from acupuncture to control 1 periods. There was a consistent decline of B's scores on the paranoid belligerence scale following acupuncture (a decrease of 47% between true acupuncture and control 2 periods). This is reflected in the significant interaction for this scale (see figure 2). The thinking disorganization scale also showed a significant main treatment effect, indicating that there was a significant difference in this variable among experimental periods. A planned comparisons test showed that this effect occurred between control 1 and control 2 periods. B contributed most to this difference, showing a 30% decline in his mean thinking disorganization scores over this interval (see figure 3). The significant interaction effect (patients by treatments) for this scale supports the above analysis of B's contribution to the treatment effect.

Case 3

C was withdrawn, suspicious, paranoid, delusional, and actively hallucinating on the ward before her acupuncture treatments. During the course of the study, especially during the second week of true acupuncture, she became less guarded, began to talk more, and became relaxed enough to smile appropriately on occasion. She interacted much more with the staff and patients. This change lasted for about a week and a half after the first course of acupuncture. She then dramatically deteriorated, again becoming withdrawn and apparently hallucinating. She improved somewhat during pseudo-acupuncture. At the completion of the study she was given the same dosages of chlorpromazine that were given B. C improved within a month, returning to the level she had achieved by the end of the true acupuncture period.

C's deterioration during the middle of the study was corroborated by the PRP finding that a dramatic increase in paranoid belligerence occurred at pseudo-acupuncture and control 2 periods. A significant patient by treatment interaction was found (see figure 2). C also showed a similar deterioration as measured by the withdrawal scale (see figure 1). Although agitated depression scores were similar to the withdrawal scores no statistically significant difference emerged for the agitated depression scale (see figure 4).

DISCUSSION

Trends in improvement after true acupuncture treatments and subsequent deterioration in the direction of baseline values can be seen in figures 1 and 2. In the case of the withdrawal scale the trend achieves statistical significance. The withdrawal scale shows that B was rated by the ward staff as minimally withdrawn on admission because of his demanding excessive interaction. During the true acupuncture and control 1 periods, the staff's ratings indicated that they viewed the patient as having improved (withdrawal for this patient was an improvement). However, their ratings immedi

ately before and during the pseudo-acupuncture periods indicate that they viewed the patient as reverting to his original behavior at admission.

C, who was rated as very withdrawn on admission, was rated as having improved with true acupuncture treatments; her maximum improvement was reached during control 1 and worsened just before and during pseudo-acupuncture.

The fact that both B and C continued to show improvement in their staff ratings during control 1 is consistent with effects often observed in acupuncture treatment of physical conditions. The maximum effect of a session often does not become manifest until a few days later. The fact that both of these patients began to worsen before and during pseudo-acupuncture argues against a placebo or hypnotic effect.

Although research findings with acute schizophrenic patients are not conclusive, there is general agreement that chronic schizophrenic patients display heightened sympathetic tonus and a heightened baseline level of physiological arousal (9). In addition, Goldstein and associates (10) reported a high level of cortical activation in chronic schizophrenic patients.

There is a group of Chinese studies showing that acupuncture points such as those used in this study can influence cortical activation and the responsiveness of the reticular activating system to peripheral stimuli. Researchers at the Shanghai College of Traditional Medicine (11) have shown that electrical responses of the midbrain reticular formulation to peripheral pain stimuli are entirely or partially inhibited by stimulation of the acupuncture point stomach 36. Similarly, a group at the Peking Medical College (12) reported that needling of acupuncture points large intestine 4 and pericardium 6 inhibited cortical evoked potentials produced by stimulating the thyroid region. On the other hand, acupuncture has been successfully used to raise the level of cortical arousal in patients in coma, with awakening occurring in some cases (13).

Other studies have shown that the same points can act in opposite directions depending on the baseline state. Of particular interest is a 1959 report from the People's Republic of China on the effect of acupuncture on the EEGs of epileptic patients (14). Using the same combination of points (including some used in the present study), one group of epileptic patients showed cortical synchronization (decrease in cortical arousal) while another group showed desynchronization (increase in cortical arousal). The authors stated, "The occurrence of the phenomenon of synchronization or desynchronization appears to depend on the level of the cortical excitation state at the time of stimulation." According to Ying-k'un and associates (14), similar results have been reported by Tang for normal subjects.

Further studies have shown that acupuncture can act to correct either a hyperfunctioning or hypofunctioning of the autonomic nervous system. Researchers at the Peking College of Medicine (15) reported that stomach 36 can correct either diarrhea or constipation. Another study from the People's Republic of China (16) reported that stimulation of the upper lip had no effect on normotensive animals but had a powerful effect in reversing hypotension and shock produced by experimental bleeding. This gives credence to the impression of clinicians that a treatment usually has an effect only if there is pathological imbalance.

The present study shows that acupuncture, when correctly performed, exerts therapeutic action, possibly by decreasing the level of sensory input to the reticular activating system and thereby lowering excessive cortical activation and concomitant elevated sympathetic tonus. Whether or not this mechanism applies to other diseases treated with acupuncture remains unanswered.

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APPENDIX 1
The Traditional Chinese Hypothesis

The traditional concepts of mental and physical disease in Chinese medicine arise out of ancient Chinese natural philosophy and science, particularly that of Taoism. According to the theory set forth in the *I Ching* and *Nei Ching* several

thousands of years ago, the universe has arisen out of a primordial energy, *Ch'i*, which evolved by a dialectic process into two polar forces, the yin and the yang. All natural processes of change, at any level of organization, can be understood as a simultaneous dynamic interplay of the yin and the yang elements, which are simultaneously complementary and antagonistic.

Yang corresponds to movement, expansion, sun, spring, summer, heat, and day. In the human body yang corresponds to the upper part of the body, the back, and the "hollow" organs (stomach, gall bladder, urinary bladder, large and small intestines). Yin corresponds to quiescence, condensation, contraction, moon, autumn, winter, cold, and night. In humans it corresponds to the lower part of the body, the abdomen, and the "solid" organs (liver, heart, blood, spleen, lungs, and kidneys).

Traditional Chinese theory states that *Ch'i* flows in cyclic patterns throughout the body by means of a network of superficial meridians connected to the internal hollow and solid organs. Disease arises when the normal homeostatic flow is interrupted, leading to excesses, deficiencies, and stases of energy. Both somatic and psychological consequences can result from such energy dysfunction, since a yin-yang imbalance affects the organism holistically.

The major factors producing imbalance are meteorological influences (cold, humidity, etc.), excessive emotions, and dietetic irregularities. In traditional acupuncture therapy, one acts on the bodily dysfunctions by means of needling the necessary acupuncture points to restore the proper energy balance. In the case of schizophrenia, the cause is a deficiency of yin and an excess of yang. The weakened yin cannot harmonize with the yang, which partially separates from it. The yang, being mobile, agitated, and light, rises to the upper part of the body, including the head, giving rise to psychotic symptoms. To treat this condition one augments the yin and disperses the yang to restore balance and reintegration.

Although the above may seem abstruse or metaphorical, it represents the ancient theoretical explanation for both the etiology of schizophrenia and the mechanism by which acupuncture treatments produce an improvement in symptoms. Interestingly, the modern Western theories referred to above to explain the action of acupuncture share with the Chinese philosophy a dialectic process that views schizophrenia as the result of an imbalance between opposing but normally complementary factors.

Special Issue of *AJP* to Feature Depression and Alcoholism

A special issue of the *American Journal of Psychiatry* will be published in mid-April 1979. This issue, which will be distributed to all subscribers and is in addition to the regular April issue, will consist of a collection of articles on the subjects of alcoholism, depression, or both. Articles will deal with such topics as the treatment of depression with imipramine and ECT; bipolar illness: factors in drinking behavior; physical activity and plasma cyclic AMP in manic-depressive patients; childhood depression; life events at the onset of bipolar affective illness; psychiatric problems in women at alcoholic detoxification; lithium maintenance; efficacy of drugs and psychotherapy in the treatment of acute depressive episodes; and life events, coping, stress, and depressive symptomatology.

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Extra copies of the special issue will be available from the APA Circulation Department at a cost of $2.25 for domestic orders and $2.90 for foreign orders. Payment must be included with order.

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Standards Affecting Mental Health Care: A Review and Commentary

BY SCOTT H. NELSON, M.D., M.P.H.

The author describes the increasing focus on regulation and the standards applied to the field of mental health. He discusses four distinct but overlapping types of standards: 1) clinical, 2) practitioner, 3) program and facility, and 4) payment. He emphasizes the increasing influence of legislation and judicial decisions and points out several trends in standard setting affecting mental health care. Psychiatrists and other mental health practitioners are called on to take a larger role in shaping these standards to ensure that the quality of patient care is not compromised.

THE AGE OF REGULATION is upon the mental health field. No longer can we mental health professionals sit in our private offices, universities, or administrative posts and carry on our activities in the way we were trained to do them. Now institutions and programs must be licensed or accredited, providers must be certified or licensed to practice, and the care we provide is coming under increasing scrutiny by our peers, the public, and third-party payers.

In the past few years an additional influence has joined the regulatory process: the legislative process and the decisions of courts regarding various aspects of mental health care. Some of these judicial decisions have been far-reaching indeed, relating to such fundamental issues as the right to treatment, the right to least restrictive care, and even to such forms of psychiatric treatment as ECT.

The heart of the regulatory process is the application of criteria or standards. According to Webster, a standard is a measure established by authority, custom, or general consent as a model or example—a measure set up by authority as a rule for determining value, extent, or quality. Since the term "standard" denotes a measure, it should be remembered that standards are set by some individual or group for some other individual or group.

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KINDS OF STANDARDS

Because the idea and practice of regulation are new to mental health professionals and because the processes that are involved in regulatory procedures are sometimes complex, the types of standards relevant to the mental health field often appear confusing. In general, however, one can usefully characterize four specific, often mutually interactive kinds of standards that apply to the field of mental health. These include 1) clinical services standards, 2) practitioner standards, 3) programs or facilities standards, and 4) payment systems standards.

Clinical Standards

Clinical standards relate to specific aspects of clinical care. These include diagnosis of the emotional disorder, the appropriate modality for treating the condition, and how long and how often treatment should take place. One of the problems in the development and application of clinical standards is that the depth, reliability, and validity of knowledge about diagnosis, treatment, and outcome in the mental health field are not as objective or scientifically developed as they are in other branches of medicine. For this reason it is often difficult for mental health practitioners to agree on appropriate clinical standards.

Clinical standards in the mental health field are usually set by practitioners of a particular discipline. Examples are the peer review committees of local branches of the American Psychological Association and the American Psychiatric Association and the local clinical standards set by physicians under the Professional Standards Review Organization (PSRO) program. Critics of the peer review process have raised the concern that professional associations cannot and/or will not responsibly apply clinical standards to the care provided by members of their own profession. However, to date the medical professions have been provided the first opportunity to develop standards that will produce much needed improvement in the quality of clinical practice and avoid excessive and inappropriate care.

Over the past century certain treatment philosophies have become well established in the practices of mental health professionals and have been ingrained in the training programs that produce these professionals. However, the anticipated strong resistance of the mental health professions to the development and ac-

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ceptance of clinical standards has not occurred. Indeed, mental health professionals have been the leaders in developing and applying their standards to care. For example, the American Psychiatric Association was among the first professional associations in the field of medicine to prepare its criteria sets to be considered by local PSROs in the review of care (1). This lack of professional resistance may be related primarily to the fact that the field of mental health care has less ability to demonstrate its merits on a scientific basis to policy makers and third-party payers.

Related to the development of clinical standards is the evaluation of treatment outcome. This is another area fraught with difficulty and disagreement in the mental health field, particularly with regard to the standards and criteria that should be used. Although treatment methodology is improving and measurement techniques are becoming more sophisticated, the evaluation of treatment of mental disorder remains considerably inferior to similar evaluation of treatment in other medical practice. In part, this is the nature of the clinical disorders with which the mental health system deals, which are of such complexity that it is usually exceedingly difficult to demonstrate, for example, that the cause of improvement was treatment intervention rather than some other factor in a patient's life.

Practitioner Standards

Standards are applied by at least three different sources to practitioners in the mental health field: agencies of government (usually states), professional associations, and third-party payers. Each of these sources of practitioner standards applies overlapping but distinct standards that may differ according to the discipline of the practitioner.

For example, the process by which a state applies standards to mental health professionals is usually licensure. Licensing gives permission to engage in a profession or occupation after the state has determined that the individual has achieved minimal specified criteria. Nominally these criteria relate to the assurance that public health, safety, and welfare of the citizens of the state will be protected.

Certification is the process used by many professional and other disciplinary groups or associations to grant recognition to an individual who has met certain criteria specified by the association. The criteria may be the same as or different from the criteria used by a state in determining licensure. Certification may or may not be required for licensure and vice versa. For example, clinical psychologists in most states must be certified by their professional association before they can be licensed to practice in the state. On the other hand, physicians who have completed a year of internship and possess a license to practice medicine technically can call themselves psychiatrists and practice psychiatry, although few actually do. Psychiatrists are not required to obtain certification from the American Board of Psychiatry and Neurology in order to practice psychiatry. However, a fully trained psychiatrist must be licensed to practice medicine in a state before he or she can take the examinations for professional certification.

In general, standards used to determine whether or not an individual may be certified or licensed relate to the training, professional degrees, and experience of the individual. In addition, specific attempts at measuring competence of the practitioner may or may not be included. Considerable debate has occurred within the professions as to whether or not competence can be measured, particularly since agreement on standards of clinical care has yet to be reached in the mental health field.

Third-party payers also apply standards to practitioners in that they reimburse services provided by some mental health professionals but not others. For example, under some insurance plans and Medicare, only services provided by physicians can be reimbursed on a fee-for-service, independent-practice basis. Services provided by independently practicing clinical psychologists, social workers, nurses, and others are excluded. Increasingly, however, clinical psychologists have achieved a status similar to that of psychiatrists with insurance companies. In fact, this standard has been taken out of the control of the carriers in the case of the Federal Employees Health Benefits Program by legislation requiring all carriers to provide equal choice for beneficiaries between psychiatrists and psychologists. In addition, Medicaid now reimburses services provided by clinical psychologists in some states.

One of the important issues in the area of practitioner standards in the mental health field is whether or not the various mental health disciplines have done enough to assure that their practitioners are well enough informed and are practicing competently. Although the difficulties inherent in any process of determining competence are admittedly great, critics have pointed out legitimately that the various professions have taken on very little responsibility with regard to such issues as standards of practice or appropriate certification and licensure of members of the profession. In an earlier publication (2) I suggested that one's professional discipline may not be the most appropriate criterion on which competence, and thereby reimbursement, should be based. Rather, the ability to deliver competently a particular kind of service might be more objectively measured on the basis of training, experience, and proficiency tests developed independent of the discipline involved. The debate surrounding practitioner standards is likely to continue, particularly since such standards relate directly to access to reimbursement and the control of the professions over their own destinies.

Program and Facility Standards

Standards for programs or facilities are applied to a wide variety of settings, ranging from such service delivery programs and facilities as state, general, and private hospitals, health maintenance organization

(HMOs), community health and mental health centers, outpatient clinics, halfway houses, and other intermediate care settings to teaching and research programs. As with practitioner standards, program standards impinge on other areas of standards development and are often applied along with such other standards as client and practitioner eligibility.

A sharp distinction has often been made between program standards and standards for facilities. Program standards usually consist of criteria that apply to an organized systematic approach to service, teaching, or research that has specific process and outcome objectives. More often, facility standards have been applied to the mechanics of patient records, institutional sanitation and safety, and the condition of physical plants. Standards that apply to a facility may or may not include program standards. In recent years the review process has tended to scrutinize both physical aspects and the specific programmatic content that is provided by an institution or facility.

Program standards are most commonly applied by states (in the form of licensure), program or institutional associations (which develop standards to assure the quality of the programs or facilities they represent), third-party payers (in the form of requirements for licensure, certification, or accreditation), or teaching or research institutions (commonly in the form of criteria for the receipt of financial aid).

Perhaps the best known organization responsible for promulgating program and facility standards is the Joint Commission on Accreditation of Hospitals (JCAH). This organization was created in 1951 to develop minimum standards of care in hospitals. The JCAH carries on activities started by the American College of Surgeons in 1913 and has evolved to become the accreditation body for institutions and even nonhospital facilities that desire to participate in such third-party payment systems as private insurance, Medicare, and Medicaid. In essence, the JCAH provides a voluntary mechanism through which a facility can be certified as a provider under most of these financing programs.

In the past JCAH has been criticized because of its almost total emphasis on the physical and record-keeping aspects of facilities and its lack of attention to programs. However, recently it has started to develop program standards, including some that relate specifically to mental health, drug abuse, and alcoholism treatment systems. Obviously, changes have been slow and difficult to effect, primarily because of the different procedures and types of individuals needed to develop and promulgate program standards, compared with those needed to develop more technical and physical standards.

The linking of licensing, accreditation, and certification of institutions to reimbursement has been of increasing concern to mental health facilities, particularly those which are nontraditional or are not associated with a hospital setting. Facility and program standards are often not established for such settings; without them, programs are not eligible for most types of reimbursement under insurance and federal financing programs. Each of the three institutes of the Alcohol, Drug Abuse, and Mental Health Administration (ADAMHA) has recognized this fact and has taken steps with JCAH to develop program standards for such new or existing treatment programs as community mental health centers and drug abuse and alcoholism treatment centers. The first surveys and JCAH accreditation of community mental health centers took place in 1976 (3).

Program and facility standards must be distinguished from program evaluation, in which the degree to which program objectives have been achieved is assessed. Meeting standards is often inherent in the process of accomplishing objectives. Conversely, having an adequate evaluation component is often a standard to which programs must adhere.

Carrying out any kind of meaningful program evaluation in the mental health field is a difficult challenge, complicated by lack of agreement on evaluation methodology, measures of program success, and even the specific objectives of the program. In the federal government, the multiple, often conflicting objectives for a program that are held by parties at varying levels of interest and authority make meaningful program evaluation a nearly impossible task.

In the process of establishing mental health program standards and program evaluation procedures, systems for collecting ever-increasing amounts of data have rapidly developed. Serious questions have been raised about the usefulness of these myriad reporting systems in the management and justification of program activities and about the burden they place on respondents in the field.

The standards and other regulations and requirements that are now applied to organized programs of mental health care are so extensive that in the near future it would not be surprising to see a return to less organized forms of mental health care, particularly solo private practice. Clearly, this raises the issue of the way in which standards and other regulatory procedures are applied; it also poses the question of how much is enough.

Payment Standards

The objectives of the standards developed and applied by such third-party payers as insurance companies, Medicare, and Medicaid are dramatically different from those applied to clinical services, practitioners, and service facilities. Payers' standards are spelled out in contracts or other formal mechanisms that describe the benefits and conditions between the payer and the subscriber or eligible client. In the case of most third-party financing programs related to health and mental health care, the goal of the payer is to meet the terms of the contract without incurring excessive costs. The primary mechanism to ensure this cost containment is by actuarial analysis and a claims review process that may or may not be carried out by a

health professional and does not necessarily conform to professional norms and criteria.

Because of the control by third-party payment systems of financial access for both the consumer and the professional, payment standards have been the subject of considerable discussion and controversy in recent years, particularly as the costs and use of both medical and mental health services have continued to rise. The power of payment standards is very great indeed. Third-party payers can determine what facility and program standards must be observed to obtain reimbursement as well as what practitioners will be eligible for reimbursement and under what circumstances. Of increasing concern is that professional standards of clinical care may or may not be used and accepted by third-party payers in reimbursement for service. Rates of payment may also become an issue, particularly in such tax-supported programs as Medicare and Medicaid. There have been substantial difficulties in obtaining practitioner cooperation in such programs because practitioners often feel that reimbursement rates are too low to make service delivery financially feasible.

Payment standards are particularly stringent in the mental health area. The Medicaid program, for example, will not reimburse for care delivered to individuals aged 21–64 in a psychiatric institution. Although some insurance carriers now allow the payment of independently practicing psychologists as well as psychiatrists, Medicare does not allow any practitioner other than a physician to collect reimbursement directly. Service provided by other mental health practitioners may be reimbursed in certain settings but only under the supervision of a physician; in addition, the reimbursement is made to the facility rather than to the individual provider.

One of the major issues relating to payment standards is the extent to which mental health, drug abuse, and alcoholism services are provided as options to subscribers of insurance programs or to clients eligible for other health financing programs. Although some insurance companies provide adequate coverage for mental disorders and substance abuse problems, most limit such coverage more than for physical disease; this is also true under the Medicare and Medicaid programs. In some states the concerns over these limitations have led to legislation requiring minimum mental health benefits in all insurance plans (4).

In part, discrimination against treatment for mental disorders by third-party payers is due to the fact that there continues to be bias and prejudice against individuals with emotional disorders. There is also concern about the overall cost and utilization rates for such conditions, especially if they are provided on an unlimited basis. Insurance companies and federal financing systems are concerned about lack of demonstrated efficacy of the treatment for certain types of mental disorder. The less favorable consideration of mental disorder by payment systems has been a subject of much controversy recently, particularly in the development of arguments for and against coverage of mental disorders in federally assisted HMOs and under national health insurance (NHI) proposals.

Payment standards that are responsive to the objectives of contract obligations and cost control have caused practitioners and concerned others to raise serious questions about the effect of such standards on the quality of care. In 1972, when a well-known insurance carrier was faced with a large potential cost overrun, types of claims that previously had been accepted were routinely denied for the remainder of that fiscal year.

Such a situation clearly delineates the difficulty in reconciling two very different sets of objectives and practices in the application of standards to clinical care—the objectives of cost containment versus quality care. On the one hand, practitioners point out that through the claims review process insurance companies are mandating not only the documentation but also the types of care that can be delivered and reimbursed. Insurance companies, on the other hand, claim that practitioners have failed to provide necessary criteria based on clinical standards and judgments by which claims review procedures can be carried out effectively. As the financial constraints grow tighter, this divergence in approach and viewpoint may widen and may require the intervention of an outside arbitrator for settlement.

THE LEGISLATIVE AND JUDICIAL SYSTEMS

The conflict between third-party payers and practitioners surrounding payment for care may very well be resolved in legislation or in the courts. The legislative and judicial systems have played ever greater roles in the past five years in the shaping of standards in each of the four areas discussed above. The reasons for this increased intervention are complex, but they probably have to do with the fact that health care of all kinds is perceived more and more as a proper concern of society as a whole rather than the sole domain of the physician and other practitioners. Medicine has become very big business indeed and, like other aspects of our free enterprise system, has increasingly evidenced the need for some form of regulation simply because of its size and cost.

There are many examples of the influence of legislation and judicial process on the mental health field. In the area of payment, one can point to the legislation mandating that psychologists be considered equal independent providers of service under the Federal Employees Health Benefits Program, along with several similar pending cases. Whether or not an institution that loses its JCAH accreditation becomes disqualified for Medicare and Medicaid reimbursement is an issue related to both payment and facility standards. Court decisions like *Wyatt v. Stickney* (5) have reaffirmed the rights of patients held against their will in institutions without treatment. In addition, several state legislatures have passed new and more stringent mental

health commitment codes in response to concern about patients' rights. Courts have also helped to set standards for staffing psychiatric facilities and have provided direction on how "treatment" should be defined.

In the clinical area, the number of malpractice claims continues to escalate yearly. The California state legislature has gone so far as to restrict the use of a major psychiatric treatment modality, ECT, by legislation and is considering a similar course for all psychotropic medications, an indication of the potential role of the legislative process in the regulation of day-to-day clinical practice.

FUTURE TRENDS

Current trends seem to indicate that the role of the practitioner in setting standards is decreasing, while the influence of payment and legal systems is increasing. The mental health field is particularly vulnerable in this situation because of the lack of well-defined and commonly accepted criteria for evaluation of clinical care, practitioner practice, and program activity.

The specific concerns of decision makers seem clear. It is widely held that a substantial proportion of practitioners are motivated by financial interest rather than by concern for patients. One insurance company official enjoys citing an example of the psychiatrist who filed a claim for individual psychotherapy for a four-month-old infant as well as separate claims for the child's parents. Insurance companies have also pointed out that it is difficult to determine from the processing of claims that treatment has been at all helpful to individuals, couples, and/or families in improving their functioning. The few instances of gross malpractice and unethical conduct that have occurred are well remembered by critics of the mental health field. In combination with the assumed lack of scientific basis for diagnosis, treatment, and outcome, these instances have tarnished the image of mental health practice as being particularly subject to incompetent and ineffective practice.

The increased influence of third-party payers and the legal system has resulted from the failure of mental health practitioners and other health professionals to regulate themselves. Although as practitioners we complain that the claims review process is arbitrarily managed and does not reflect sufficient professional input, we have made little effort to provide the necessary clinical criteria that would be useful in improving the process. In many instances we have been reluctant to participate directly in the review process. Instead of a collegial attempt to improve the standards of competence for practitioners by delineating which disciplines are capable of performing which kinds of services, there is continued jockeying of professions for standing within the reimbursement system. This contributes little toward winning the confidence of third-party payers, not to mention the needed support of an increasingly skeptical public.

Clearly, part of the difficulty in these areas has to do with the status and independence of the various practitioners to take on new and different roles as mental health professionals. Generally, our training has been in ways to gain clinical knowledge and skills and to apply them to service delivery. We have usually not been trained to deal with financial management, development of clinical standards and review mechanisms, or social policy issues. However, to the extent that mental health professionals continue to withdraw from these areas, third-party payers and the legislative and judicial systems will play an increasingly greater role in the control of clinical services, training, and research activities. The potential for arbitrary decisions in the area of clinical treatment becomes correspondingly greater.

It is encouraging that some attempts are being made by mental health practitioners to help the field become more accountable and to regulate itself. The number of conferences, books, and articles related to accountability of mental health practice has increased dramatically in the past five years. However, the commitment and involvement must be greater, and it must come from all of us if we want to shape the standards and regulatory processes that are applied to us. If we avoid this responsibility we risk significant compromise in the quality of care for our patients.

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BRIEF COMMUNICATIONS

Paranoia and Platelet MAO in Normals and Nonschizophrenic Psychiatric Groups

BY RICHARD J. HAIER, PH.D., DENNIS L. MURPHY, M.D., AND MONTE S. BUCHSBAUM, M.D.

The authors compared the correlation between platelet monoamine oxidase (MAO) activity and the Paranoia (Pa) scale of the Minnesota Multiphasic Personality Inventory in several groups. The data suggest that there is a positive association between high MAO activity and high scores on the Pa scale but only in samples with psychopathology.

As researchers continue to identify tentative relationships between specific biological variables and psychopathology, one strategy for clarifying the meaning of such relationships is to study the same biological variables in nonpsychiatric samples. This strategy not only eliminates possible artifacts of hospitalization and drug treatment but also tests the specificity of the variable's relationship to psychopathology. A series of reports on platelet MAO activity differences between normal controls and three types of psychiatric patients—chronic schizophrenics (1), patients with bipolar affective disorders (2), and alcoholics (3)—has prompted several investigations of MAO correlates in nonpatient samples (4–6). These studies tend to support the existence of a relationship between MAO and psychopathology, but MAO appears to be related to a broad range of psychiatric problems. In view of this nonspecificity, the recent reports that paranoid schizophrenics (7, 8) and schizophrenics with auditory hallucinations (9) have lower platelet MAO activity than nonparanoid chronic schizophrenics are particularly interesting because they suggest a more specific involvement for MAO. Because the low-MAO/paranoia relationship has been questioned in other reports (10, 11), we decided to investigate the suggested relationship in nonpatient samples and in nonschizophrenic psychiatric patients by examining the correlation of platelet MAO activity with the Paranoia (Pa) scale of the Minnesota Multiphasic Personality Inventory (MMPI).

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METHOD

We reviewed all available data collected by NIMH researchers in which psychiatric patients and volunteer controls had completed both MAO and MMPI testing. A total of 352 subjects in five different projects fulfilled this criterion. In the Haier and associates project (12), 385 student volunteers were screened for psychopathology on the MMPI. An index group of the 36 subjects who scored more than 4 standard deviations above the standardized population mean on at least one of the clinical scales was identified. Subsequent psychiatric interviews revealed that 20 of these subjects met Research Diagnostic Criteria (13) for an affective disorder, but none met the criteria for schizophrenia. A control group of subjects who scored within 2 standard deviations of the population mean on all clinical scales also was selected. Major and associates (3) tested a group of Navy enlisted men who were in a special 6-week treatment program for alcoholism. Each subject completed both MAO and MMPI testing at admission and again at discharge. Buchsbaum (unpublished data) and Murphy and associates (14) tested randomly selected normal volunteer subjects. The fifth group consisted of patients with affective disorders who routinely completed the MMPI and MAO testing (during drug-free and diet-controlled periods) as part of their stay in an NIMH research ward.

Platelet MAO activity was assayed as reported by

TABLE 1
Means and Correlations for MAO and the MMPI Paranoia Scale in Psychopathologic and Control Samples

Source	Sample			MAO[a]		Pa		Correlation	
	Description	Sex	N	Mean	SD	Mean	SD	r	p (2-tailed)
Psychopathologic samples									
Haier and associates (12)	MMPI index	M	36	10.2	2.8	14.6	4.0	.39	.02
Major (3)[b]	Alcoholics								
	Admission	M	78	7.5	3.3	12.7[c]	4.0	.30	.01
	Discharge	M	68	8.2	3.1	11.0[c]	5.0	.39	.001
NIMH (unpublished)	Bipolar patients	M	12	7.7	2.5	11.2	4.1	.51	n.s.
		F	22	9.0	4.1	12.6	3.6	−.15	n.s.
	Unipolar patients	M	11	8.8	3.3	14.7	2.7	.39	n.s.
		F	16	12.8	4.3	14.6	3.7	.54	.05
Control samples									
Haier and associates (12)	MMPI controls	M	27	8.8	3.1	9.4	2.1	−.23[d]	n.s.
Buchsbaum (unpublished)	Volunteers	M	9	12.1	5.4	12.3	3.2	−.20	n.s.
		F	16	15.8	5.1	10.1	3.3	−.38	n.s.
Murphy and associates (14)	Volunteers	M	30	11.2	4.7	11.7[c]	3.0	−.02	n.s.
		F	65	13.7	5.6	10.0[c]	3.0	−.33	.01

[a]MAO was assayed with benzylamine and is reported in nmol/10^8 platelets/hour except for control sample data from Buchsbaum and from Murphy and associates, which are benzylamine equivalents derived by transforming the original tryptamine values with a special regression equation (15, p. 258).
[b]Data on MAO are from reference 3; Pa data were provided by the author.
[c]These means and standard deviations are raw score estimates transformed from standard scores.
[d]This correlation is probably attenuated because the sample was selected in a way that would exclude individuals with high scores on the Pa scale.

Murphy and associates (15). ^{14}C-Benzylamine was the substrate in all but the Buchsbaum and Murphy and associates normal volunteer samples, where ^{14}C-tryptamine was used.

The MMPI Pa scale is one of nine original clinical scales developed to assess psychopathology. Low scores on the Pa scale may indicate trustfulness and a lack of personal sensitivity to others and may be related to underachievement in college students. High scores tend to characterize people who are more concerned with themselves than with others and who are unable to deal with criticism. Very high scores indicate suspiciousness or paranoia (see references 16 and 17). The interpretation of MMPI scales depends on the relative elevation among all the scales, so reliance on a single scale typically is not useful. However, the Pa scale is a standard measure that is comparable among the different samples studied here and is a reasonable psychometric index of the paranoid construct.

RESULTS

Table 1 lists the MAO/Pa correlations for the samples defined by some kind of psychopathology and those for normal volunteer and control samples. Five of the six MAO/Pa correlations in the psychopathologic samples are positive and three are statistically significant (alcoholics at admission and discharge were counted only once). All five of the correlations in the control samples are negative and only one is significant. The sign difference of the MAO/Pa correlations between the two kinds of samples is statistically significant (Fisher exact test, $p<.02$).

Four of the significant correlations are in samples of 36 or more subjects. In these samples of satisfactory size, we calculated the correlations between MAO and each of the remaining MMPI clinical scales. In three of these samples, the MAO/Pa correlation was the only one to reach statistical significance. In the fourth sample (Murphy and associates, data for females), the Hysteria and Psychasthenia scales were correlated with platelet MAO (+.25 and +.33, respectively, $p<.05$).

DISCUSSION

The pattern of correlations in these samples suggests that platelet MAO is related to the Pa scale but only in samples defined by criteria of psychopathology. Moreover, the direction of the relationship, i.e., high MAO activity associated with high Pa scores, is opposite of what would be expected on the basis of paranoid schizophrenics having the lowest mean MAO levels of any psychiatric group. The subjects in the psychopathological samples reported here, however, are characterized by affective symptomatology rather than schizophrenia. If the subgroup of the Haier and associates MMPI index sample that meets RDC for affective disorders ($N=20$) is considered separately, the MAO/Pa correlation is .45 ($p<.05$).

The data from schizophrenic patients imply an inverse relationship between MAO and paranoia, but results for these affectively ill samples indicate a positive relationship. However, the MMPI Pa scale may not assess the same construct that is assessed by the clinical diagnosis of paranoia. Furthermore, paranoid individuals can successfully avoid responding to the obvious items on the Pa scale, which can lower their scores (see reference 16). This would attenuate the MAO/Pa correlation, but it would not account for the directional difference. Finally, with respect to the lack of a significant MAO/Pa correlation among normal control

groups, Winter and associates (18) also reported nonsignificant correlations between platelet MAO (tryptamine substrate) and the Pa scale in normal monozygotic and dyzygotic twins.

Differences in the functional relationships among variables within different psychiatric groups may provide clues for understanding the biological bases of certain psychopathological manifestations. How platelet MAO interacts with other neurochemical systems is not known, but a symptom manifestation like paranoia could result in part from a ratio between two or more opposing neurotransmitter systems. Thus, other biological differences between schizophrenics and affectively ill patients may determine the strength and direction of the MAO/Pa relationship. Some considerations of how multiple factors, including genetically based enzyme differences and various situational factors, may interact to yield different clinical phenomena have been discussed elsewhere (see references 3 and 19).

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Phenylethylamine in Rhesus Monkeys: Interactions with α-Methyl-*p*-tyrosine and L-Dopa

BY JARED R. TINKLENBERG, M.D., J. CHRISTIAN GILLIN, M.D., GREER M. MURPHY, JR., RICHARD STAUB, AND RICHARD JED WYATT, M.D.

Rhesus monkeys, pretreated with α-methyl-p-tyrosine (AMPT) and subsequently injected with phenylethylamine (PEA), did not demonstrate the characteristic amphetamine-like PEA effects. However, when AMPT pretreatment was followed with L-dopa and then PEA injection, PEA effects were restored. These results are compatible with a dopamine theory of schizophrenia.

PHENYLETHYLAMINE (PEA), the endogenous parent chemical structure for the catecholamines, and its derivatives are of particular psychiatric interest.

1. PEA is structurally similar to amphetamine, lacking only the side chain methyl group. Recent studies in rodents and monkeys indicate that some, but not all, of the pharmacological and behavioral effects of PEA are similar to those of amphetamine (1–4). Amphetamine-induced behaviors are considered by many as a useful pharmacological model of paranoid schizophrenia (5–8).

2. There is at present no clear evidence that consistent tolerance develops to most behavioral effects of PEA (2, 9), although some pharmacological tolerance may develop (1, 10). Lack of consistent tolerance would be compatible with an endogenous substance that contributes to chronic psychiatric disorders such as schizophrenia.

3. Under certain conditions, neuroleptic drugs modify some of the PEA-induced behaviors in a pattern that approximates the clinical potency of the neuroleptics (11).

4. PEA is a specific substrate for monoamine oxidase (MAO) type B (12). Platelet MAO, most of which is type B, has been low in certain types of schizophrenia and hence in those individuals any effects of PEA might be exaggerated (13, 14).

In the present study, we further defined the effects of PEA in rhesus monkeys by making pharmacological manipulations with α-methyl-*p*-tyrosine (AMPT), L-3,4-dihydroxyphenylalanine (L-dopa), and phenobarbital.

METHOD

Experiment 1: AMPT-PEA Interaction

We studied six subadult *Macaca mulatta* that weighed 4–6 kg and were housed in individual cages arranged so that they could not see each other. Before receiving treatments, the animals were fasted for 24 hours.

Each of the six monkeys was pretreated with 125 mg/kg of AMPT or saline placebo three hours before challenge with 75 mg/kg of PEA or saline placebo. In a randomized block design, the six animals were studied during each of four treatment conditions: saline-saline; AMPT-saline; saline-PEA; AMPT-PEA. At least three days separated each treatment. AMPT and placebo pretreatments were administered by oral intubation in 10 ml of saline.

PEA and placebo challenge doses were given intramuscularly in 0.1 ml/kg of saline. The squeeze bar, a solid panel that could be moved so as to gently restrain the animal against the side of the cage, was used for the I.M. injections.

Two observers, both blind to the treatment conditions, simultaneously recorded two classes of behavior: checking and general activity. Checking was defined as any change in the visual field of the animal due to eye or head movements. The observers counted the number of individual eye movements (or head movements if the eyes were not visible) that occurred during a 15-second observation interval. The general activity score was based on 16 discrete, objectively defined categories of behavior scored as either present or absent during a separate 15-second interval that followed the interval when checking was counted. The activity score quantified the level of nonstereotyped assertive or purposeful behavior such as exploring or grooming. The correlation coefficients of the interrater reliabilities were above .90.

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The procedure for any single injection study involves two pretreatment observation sessions at 30 and 15 minutes before injection, and four observation sessions at 15, 30, 45, and 60 minutes after injection. During each session, checking was counted for 15 seconds, then general behavior was observed for 15 seconds, and during a third 15-second interval the checking count and the general behavioral observations were recorded. A total of six 45-second cycles were carried out during each observation.

For each observation session, the checking counts for the six 15-second intervals were summed. This yielded a total checking count for each 4.5-minute session, which was compared with the totals for other sessions. The activity scores were tabulated by counting the number of 15-second intervals during an observation session in which the behavior occurred and comparing that number with the number of intervals in which it occurred during other sessions.[1]

Experiment 2: AMPT-L-Dopa-PEA Interaction

In the second experiment, we first established that L-dopa at a dose of 5 mg/kg, administered intraperitoneally (I.P.) in 5 ml of buffered solution, had no observable behavioral effects. Then, 2 hours after pretreatment with AMPT, 125 mg/kg p.o., four monkeys were given L-dopa, 5 mg/kg I.P. Three hours after AMPT pretreatment (1 hour after L-dopa), each animal was given PEA, 75 mg/kg I.M. On a separate day, also after pretreatment with AMPT followed by L-dopa, each animal received saline placebo. The order of treatments was counterbalanced. The pretreatments, injections, and behavioral observations were conducted according to the Experiment 1 schedule.

RESULTS

Experiment 1: AMPT-PEA Interactions

We determined for each treatment the mean change in checking—stereotyped behavior defined as repetitious and nonpurposeful, with abrupt eye and head movements—between observation sessions conducted at 15 minutes before and 15 minutes after I.M. injection of PEA or saline. An analysis of variance was significant for treatment ($F(3,15)=13.32$, $p<.01$) but not for subjects ($F(3, 15)=1.17$, n.s.). Following saline pretreatment, checking increased more after injection of PEA than during other conditions. The mean change in checking for saline-PEA was significantly different ($p<.01$) from the mean changes for AMPT-PEA, saline-saline, and AMPT-saline (Duncan multiple range tests for pairwise comparisons). The mean changes among these last three conditions were not significantly different. These findings are compatible with rat studies in which AMPT blocked PEA stereotypy (1).

[1]Details of scoring, reliabilities, and analyses can be obtained from Dr. Tinklenberg.

We also determined for each treatment condition the mean change in the nonstereotyped activity score between observation sessions conducted before and after injection of PEA or saline. The Duncan test indicated that the mean decrease in nonstereotyped activity for the saline-PEA condition was significantly different from the mean changes for the other treatments ($p<.05$). The mean changes for the other treatment conditions were not significantly different.

During the saline-PEA condition, but not during the other treatments, the monkeys clearly showed other PEA-induced behaviors such as an increase in lip smacking—rapid repetitive opening and closing of the mouth—and an increase in other repetitive, non-goal-directed (stereotyped) behavior such as rocking or chewing-gnashing.

As a control, two of the six animals were pretreated with phenobarbital, 25 mg/kg p.o., and then challenged three hours later with PEA, 75 mg/kg I.M. Although obviously sedated, the animals showed the characteristic PEA effects.

An additional study was done to determine if the inhibitory effects of AMPT on the PEA response were transient. Two monkeys were treated with AMPT, 125 mg/kg p.o. twice a day for four days. On the fourth day, three hours after AMPT pretreatment, the animals received PEA, 75 mg/kg I.M. Neither animal showed PEA effects.

Experiment 2: AMPT-L-Dopa-PEA Interactions

Pretreatment with AMPT followed by L-dopa and then by the challenge injection of PEA produced the characteristic PEA-induced mean change in checking. This L-dopa-restored, PEA-induced checking was significantly different from the change in checking during control (saline placebo) conditions (paired t test, two-tailed, $p<.01$). The mean change in activity score during AMPT-L-dopa-PEA conditions did not differ significantly from the change during AMPT-L-dopa-saline conditions. The AMPT-L-dopa-PEA condition was associated, however, with other characteristic PEA effects such as lip-smacking and other stereotyped behavior.

DISCUSSION

The combined results of Experiments 1 and 2 suggest that AMPT does not directly block all the mechanisms through which the PEA effects are mediated in monkeys. Instead, AMPT attenuation of certain PEA effects in monkeys probably operates through prereceptor mechanisms. This finding is important because AMPT inhibits the enzyme tyrosine hydroxylase, which facilitates the conversion of tyrosine to L-dopa; this conversion is the rate-limiting enzymatic step in the formation of catecholamines. Thus, some of the PEA-induced behavioral changes may require an intact catecholamine pathway or at least require the presence of an adequate pool of L-dopa. Since L-dopa

is easily converted to dopamine, PEA-induced stereotyped behavior in monkeys seems to reflect dopaminergic activity.

The lack of association of checking and nonstereotyped activity changes shown in Experiment 2 cannot be interpreted clearly with the available data. Perhaps the measures of checking are more sensitive; alternatively, checking and other behaviors may be mediated through separate neurochemical pathways. This latter possibility is supported by recent rat studies in which AMPT did not block all PEA effects (15).

The present study indicates that PEA effects are associated with dopaminergic activity. This conclusion is buttressed by reports that different neuroleptics attenuate PEA effects in approximately the same rank order of potency as their ability to block dopamine receptors (11). Our findings are compatible with a dopamine theory of schizophrenia, although at present the necessary supporting evidence is not available (14). Further investigation of PEA and related metabolites may further our understanding of psychiatric disorders.

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Gonadotropin Response to Synthetic Gonadotropin Hormone-Releasing Hormone (GnRH) in Heroin Addicts

BY FRANCESCA BRAMBILLA, M.D., LEONARDO RESELE, M.D., DOMENICO DE MAIO, M.D., AND PAOLA NOBILE

To determine whether the pituitary-gonadal deficiency in heroin addicts is related to heroin's effect on the hypothalamus, the authors administered gonadotropin hormone-releasing hormone (GnRH) to 10 male heroin addicts and 5 controls and measured follicle-stimulating hormone (FSH) and luteinizing hormone (LH) response. Basal FSH and LH levels were significantly lower in addicts; after GnRH stimulation the addicts' FSH and LH values increased but not significantly compared to controls. The difference between the two groups' response was highly significant. The authors suggest that heroin causes an incomplete blocking of gonadotropin secretion at the pituitary level, inducing a hypophyseal-gonadal deficiency and a long-lasting depletion of the endogenous releasing factor, which accounts for the reduced response to GnRH.

IN A PREVIOUS STUDY (1) my associates and I observed that male heroin addicts have reduced basal secretion of follicle-stimulating hormone (FSH), luteinizing hormone (LH), and testosterone and that this deficiency is still present 10 days after withdrawal of the drug. These data agree with those of Azizi and associates (2) and Mendelson and associates (3–5) on the testosterone deficiency, which seemed to improve 1 month after withdrawal of the drug. However, in studies by Cushman and associates (6, 7), LH and testosterone levels were normal, with only 12% of the cases showing reduced secretion. Volunteers who were given heroin, either in a single dose or over 10 days, had a significant decrease in testosterone and LH levels, whereas FSH did not seem to be affected (8). Cicero and associates (9) found that heroin addicts had a nonsignificant reduction of testosterone levels, mean volume of ejaculate, and citric acid in the spermatic fluid; a nonsignificant increase in sperm count; and a significant reduction in sperm motility.

Many factors could be responsible for these contrasting results, such as differences in the age of the patients, the daily dose of heroin, the duration of addiction, and previous administration of other drugs of abuse. However, among the addicts hospitalized in our institute for detoxification, we always observe a marked deficiency in the function of the pituitary-gonadal axis; therefore, we thought it worthwhile to study the pituitary response to gonadotropin hormone-releasing hormone (GnRH) stimulation in order to ascertain whether the observed impairment is related to an effect of the drug on the hypothalamus.

METHOD

Sample

The subjects were 10 male heroin addicts, aged 18–28 years, who had a history of addiction to heroin alone that had lasted between 7 months and 7 years. All of the patients had previous histories of intake of other drugs of abuse (amphetamine, LSD, cannabis, morphine) but had been totally off them since they started to use heroin. The daily heroin intake ranged from 300 mg to 3 g of the street preparation (18% pure heroin) and was always taken intravenously.

All of the patients were examined immediately after hospitalization, 8–12 hours after the last heroin intake. All had a normal body build, were without gynecomastia, and had normal masculine hair distribution and normally developed genitalia for their age. All of the patients reported reduced or absent libido and sexual potency. No neurological disorders were found. Three of the subjects had previously had viral hepatitis.

The controls in the study were 5 male patients, matched for age, who were hospitalized for sociofamilial mental deficiency and were endocrinologically normal. All of the addicts and controls gave informed consent.

GnRH Stimulation Test

We used the GnRH stimulation test to evaluate the functioning of the hypothalamo-pituitary axes of our subjects, measuring the FSH and LH responses to the stimulation. The GnRH test was carried out as follows: the patients fasted and rested in bed for at least

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8 hours before the test started. They were given 150 μg of synthetic GnRH I.V. as a bolus. Blood samples were drawn through an indwelling catheter inserted in a forearm vein and kept patent by a saline infusion. The samples were taken immediately before the injection of GnRH and 30, 60, 90, and 120 minutes afterward. Because periodic release and circadian rhythms of gonadotropin secretion have been reported (10), all GnRH tests were carried out between 8:00 and 10:30 a.m. Serum FSH and LH were assayed by the radioimmunological methods of Midgley (11, 12) using double-antibody techniques.

The range of LH values was 1.5–25 ng/ml; the interassay error ranged from ±0.97 (±31%) to ±3.05 (±6%), and the intra-assay error ranged from ±1.29 (±42%) to ±5.4 (±10%).

The FSH values ranged from 1 to 17 ng/ml, and the interassay error ranged from ±0.98 (±32%) to ±1.46 (±3%); the intra-assay error ranged from ±1.57 (±5%) to ±2.61 (±5%).

The analysis of variance for a mixed factorial design was done on our data to test the statistical significance of the differences between groups and between times and for the interaction between these two factors. Comparison of averages was done by means of the Scheffé's test (13). The statistical analysis was done with the 5 controls and only 5 of the addicts, selected at random in order to have numerically similar groups. The addicts who were not included in the statistical evaluation showed the same type of response as the patients who were.

The Blachly scale for signs of abstinence (14) was used to check if the patients were showing phenomena related to the withdrawal from heroin.

RESULTS

The basal levels of FSH were extremely reduced in all the patients. Stimulation with GnRH induced a mean peak increase of 55.15%. The peak levels occurred between 30 and 60 minutes after injection, with a prevalence of individual peaks at 60 minutes. The increase was still demonstrable after 120 minutes. The controls peaked at 30 minutes, had an average increase of 56.9%, and returned to prestimulation levels at 120 minutes (see figure 1).

The analysis of variance of these data showed a highly significant difference between controls and addicts ($F=111.62$, $p<.01$), both in basal levels and response to stimulation. The difference between times ($F=7.24$, $p<.01$) were both highly significant and indicate that the variations of the means during the test differ according to the subject group. In fact, because of the lower basal FSH levels in addicts, the response to the GnRH test, even though having a percentage of increase equal to that of controls, is not statistically significant in absolute terms.

The basal levels of LH were also very low in all of the patients. GnRH induced a mean peak increase of 147%, with a peak between 30 and 60 minutes after injection. At 120 minutes the increase was still apparent. The controls peaked at 30 minutes; their mean increase was 125% (see figure 2). The analysis of variance of these data showed a highly significant difference between controls and addicts ($F=126.75$, $p<.01$), both in basal levels and response to stimulation, for the same reasons reported for FSH. Differences between times ($F=41.81$) and the interaction between times and groups ($F=25.65$) were highly significant.

The comparisons of both FSH and LH values at baseline and at the 4 subsequent intervals show that in the control group there was a highly significant increase at 30 minutes, followed by a decrease. At 120 minutes, levels reached the pretreatment values. The

FIGURE 1
FSH Response to GnRH in 10 Male Heroin Addicts and 5 Controls

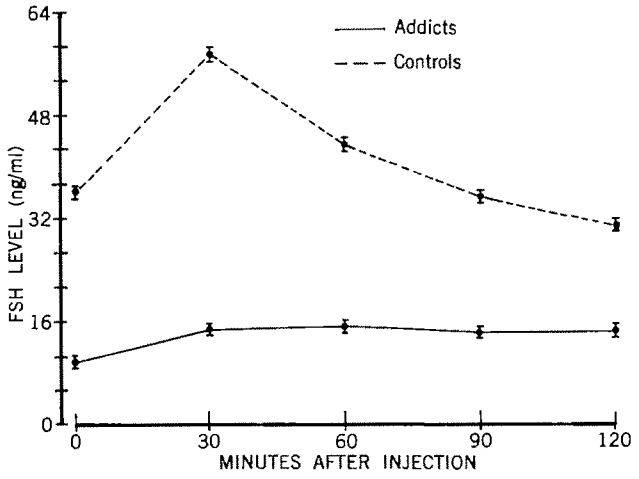
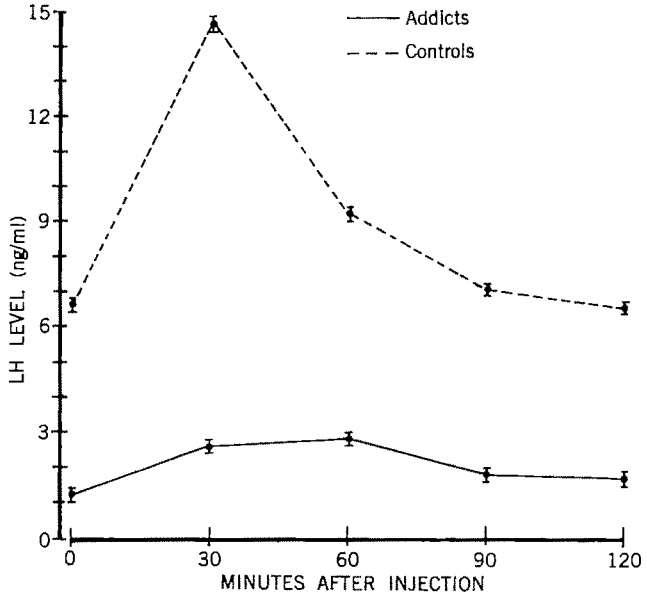


FIGURE 2
LH Response to GnRH in 10 Male Heroin Addicts and 5 Controls



addicts showed no significant difference between times; they had a gradual increase with a peak at 60 minutes and a decrease that had not reached the prestimulation levels after 120 minutes.

The patients did not show significant symptoms of abstinence from heroin.

DISCUSSION AND CONCLUSIONS

Our data again demonstrate that basal LH and FSH levels are greatly reduced in male heroin addicts. GnRH stimulation induced a nonstatistically significant increase in both gonadotropins, with delayed peak times and a slow decrease at 120 minutes. The difference between the response of the addicts and that of the controls was highly significant. The addicts' lower response to GnRH stimulation could have been caused by a primary pituitary impairment (15–20), due to idiopathic pituitary insufficiency, pituitary tumors with decreased levels of basal gonadotropins and an extrasellar expansion, Sheen syndrome, hypothalamopituitary malformations, or acromegaly. We would suggest, therefore, that in these patients heroin acted by blocking the gonadotropin secretion at the pituitary level, thus inducing a hypophyseal-gonadal deficiency. However, even though the GnRH stimulation did not induce a significant response in our subjects, an increase in FSH and LH was observed. This phenomenon suggests that the block of pituitary gonadotropins is not complete.

It has been reported (19–23) that a long-lasting deficiency of endogenous GnRH can induce a reduced and sometimes blunted response to stimulation with the exogenous releasing factor. This phenomenon has been reported in patients suffering from hypogonadotropic-hypogonadism of hypothalamic origin, in hypogonadotropic-hypogonadal patients with anosmia, and in adolescents with delayed puberty. It has been suggested that the degree of prior exposure of the gonadotropes to endogenous GnRH affects both the magnitude and the nature of the FSH and LH response to an acute dose of synthetic releasing factor. In other words, the severity and duration of the hypothalamic deficiency play a role in the type of response to the stimulation by leading to a reduced pituitary reserve of gonadotropimes. In fact, according to Yoshimoto and associates (19), in patients with reduced pituitary response due to prolonged hypothalamic deficiencies, repeated stimulation with GnRH over 7 days normalized the response to the releasing factor.

High levels of androgens reduce the secretion of FSH and LH and the response to GnRH, acting at the hypothalamic levels (20). However, in our patients the testosterone levels were low, so this cannot be the reason for the blunted response. We therefore suggest that the pituitary-gonadal deficiency in our subjects and the reduced response to GnRH stimulation arise from a long-lasting depletion of the endogenous releasing factor related to the heroin addiction. Morphine and its derivatives are known to induce impairments in brain catecholamine levels and turnover in animals (24), although the mechanism of their action has not been completely explained. It is possible that heroin acts at the level of the hypothalamic neurotransmitters in a similar way in humans, thus impairing the secretion of the releasing factor.

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The Trial Use of Clozapine for Abnormal Involuntary Movement Disorders

BY ERIC D. CAINE, M.D., RONALD J. POLINSKY, M.D., RONALD KARTZINEL, M.D., AND MICHAEL H. EBERT, M.D.

Twelve patients with abnormal involuntary movement disorders were treated with clozapine in a double-blind, placebo-controlled trial. The cohort consisted of individuals with Gilles de la Tourette's syndrome, Huntington's disease, and atypical persistent dyskinesia that was drug induced. Two subjects were dropped from the protocol due to complications. Two patients with Huntington's disease showed a marked decrease in movements; other individuals obtained no significant therapeutic benefits. Seven of the 10 patients completing the trial experienced moderate or marked side effects.

THE RISK OF persistent, drug-induced dyskinesia or drug-related deterioration of function is a therapeutic dilemma in the treatment of abnormal involuntary movement disorders. Haloperidol is the drug of choice for most patients with Gilles de la Tourette's syndrome (1) and is used frequently for symptom amelioration in individuals with other abnormal involuntary movement disorders. However, patients with Huntington's disease may experience a drug-induced deterioration in function brought on by the neuroleptics used to suppress their choreoathetoid movements (2), while those individuals with severe neuroleptic-induced movement disorders often require palliative treatment with the same agents that caused their dysfunction. Gilles de la Tourette patients may require haloperidol treatment indefinitely; we recently described a 15-year old patient with Gilles de la Tourette's syndrome who had persistent, drug-induced dyskinesia (3).

Clozapine, an antipsychotic dibenzodiazepine, causes few if any extrapyramidal side effects (4). In hopes of finding an effective agent that did not induce persistent dyskinesia, we instituted a clinical trial of clozapine in patients with abnormal involuntary movements. Clozapine demonstrates dopamine receptor blocking properties in vitro (5). The lack of extrapyramidal side effects may be due to its anticholinergic effects (6) or to its apparent augmentation of γ-aminobutyric acid turnover (7). Although clozapine has not led to any known cases of tardive dyskinesia, it is now being withdrawn from the European market, and further research in the United States is being curtailed.[1] The use of clozapine has been associated with fatal agranulocytosis (8).

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At the time this work was done the authors were all with the Section on Experimental Therapeutics, Laboratory of Clinical Science, National Institute of Mental Health, Bethesda, Md., where Dr. Caine was Medical Officer, Dr. Polinsky is Staff Physician, Dr. Kartzinel is Consultant, and Dr. Ebert is Chief. Dr. Caine is now Assistant Professor of Psychiatry, Neurology, and Pharmacology and Toxicology, University of Rochester Medical Center, 300 Crittenden Blvd., Rochester, N.Y. 14642.

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METHOD

Twelve patients participated in a double-blind, crossover trial. Each person was initially assigned to active drug or placebo group by coin toss; subsequently all were crossed over to the other treatment (patient 4 received active treatment only; her symptom

[1]Honigfeld G (Sandoz, Inc): Personal communication, April 19, 1978.

change was compared with baseline tic levels). Placebo and drug phases lasted 4–7 weeks; each individual who completed the study received clozapine in therapeutically significant doses for 3–5 weeks (see table 1). Maximum daily drug intake was determined primarily by the severity of side effects; the maximum dose was 8–10 mg/kg per day (patient 1 was treated at 5 mg/kg per day prior to approval for higher dose levels). Dosage was increased either by a predetermined schedule (by day 10 patients received at least 200 mg) or as each patient tolerated side effects. Dosage of active medication and placebo was controlled by a physician who was not blind to the protocol.

Diagnostic criteria for the 7 Gilles de la Tourette patients included onset of the syndrome before age 14, multiple motor and vocal tics, gradual replacement of old symptoms by new ones, and waxing and waning symptom severity. Six of these patients had previously benefited from treatment with haloperidol. One (patient 5) had been unable to use haloperidol because she had experienced significantly disturbing akathisia. Three patients with Huntington's disease had typical abnormal movements, positive family histories, and changes on computerized tomographic scans consistent with the known neuropathology of the disease. Two patients with atypical drug-induced dyskinesias were diagnosed by positive history of prolonged exposure to phenothiazines, negative family histories of neurological illness, and unremitting abnormal movements for at least 1 year. Both had shown continued progression of abnormal movements while being treated with phenothiazines. Patient 11 had previously been treated unsuccessfully with multiple agents. During the evolution of his present disorder, patient 12 had manifested choreiform hand and toe movements as well as incessant lateral mandibular movements with an intermittent torticollis.

Informed consent was obtained from all patients and from the parents of individuals under 18 years of age.

The Gilles de la Tourette patients were assessed with twice weekly global ratings and with twice weekly tic counts during which a blind rater observed the patient engaged in conversation with another individual for a 5-minute period. These patients were oriented to the study format during a baseline period to lessen their anxiety. A neurologist blind to the protocol twice weekly evaluated the abnormal involuntary movements and general functional ability of the other subjects. A scale of 0–4 (0=no symptoms, 4=debilitating-incapacitating symptoms) was used to rate the subjects.

The significance of changes in each Gilles de la Tourette patient's tic counts was evaluated with Wilcoxon's two-sample rank sum test.

RESULTS

Patient characteristics, maximum dose, therapeutic response, and complications and side effects are presented in table 1. Two subjects (patients 3 and 8) were dropped from the study before significant therapeutic doses were reached. Patient 3 experienced a progressive decrease in his leukocyte count during a 10-day period while on small doses of clozapine (his granulocyte fraction was routinely low before the study). Although this patient's clinical picture was not typical of drug-induced granulocytopenia, clozapine was discontinued as a precautionary measure. His leukocyte count returned to levels greater than 5,000 in about a week. Patient 8 complained of feeling "slightly confused" for 2–3 hours on the day before his total dose was raised from 40 mg to 60 mg. When the dose was increased to 60 mg, he experienced time disorientation, difficulty comprehending statements or instructions, decreased ability to perform routine self-care tasks, and symptomatic orthostatic hypotension with drops in diastolic blood pressure of more than 30 mm Hg. All of his symptoms cleared completely 48 hours after clozapine was discontinued.

One of the subjects with Gilles de la Tourette's syndrome (patient 1) showed a marked reduction in the frequency of simple motor tics. He experienced a decrease from a mean of 51.0 tics (SD=12.5) per 5 minutes to 20.5 tics (SD=14.7, $p<.001$). However, complex manifestations of his disorder were unaffected. These obsessive-compulsive rituals varied synchronously with his other symptoms and were treated effectively with 4 mg of haloperidol daily. The remaining Gilles de la Tourette patients showed no decrease in tic counts at doses of medication that should have had a therapeutic effect in this young population. Four of these patients exhibited a transient exacerbation of symptoms at doses of 50–125 mg. Patients 4 and 6 slept an additional 2–4 hours daily and experienced drowsiness when awake, a side effect also noted by other patients.

The 2 patients with Huntington's disease who reached therapeutic doses showed decreased abnormal movements; both had mild residual symptoms. Patient 9 was extremely sensitive to sedative effects manifested by sleeping an extra 4–5 hours daily. Patient 10 experienced a dramatic increase in abnormal ballistic and choreoathetoid movements after abrupt discontinuation of clozapine. She was given 5 mg of haloperidol daily to control these symptoms.

The patients with atypical drug-induced dyskinesias showed no benefit from clozapine. Patient 12 slept an extra 3–4 hours daily when maintained on his maximum dose.

DISCUSSION

Despite its apparent pharmacological activity clozapine proved clinically ineffective in treating the Gilles de la Tourette patients and the 2 individuals with atypical drug-induced dyskinesias. Two patients with Huntington's disease showed substantially decreased abnormal movements. However, both of them experi-

TABLE 1
Characteristics of 12 Patients Treated with Clozapine for Abnormal Involuntary Movement Disorders

Patient, by Group	Sex	Age (years)	Clinical Features	Maximum Dose (mg)	Therapeutic Response	Complications and Side Effects
Gilles de la Tourette syndrome						
1	M	18	Multiple simple tics (extremities, trunk) and complex ritualistic movements, mild vocal tics	350	Disappearance of most simple tics; rituals unchanged	Symptoms exacerbated at low doses; mild drowsiness
2	M	15	Multiple simple tics (mouth, face, occasional other), moderate to severe coprolalia, moderate vocal tics	425	Mild decrease in motor tics (not statistically significant) without effect on coprolalia	Symptoms exacerbated at low doses; evaluation complicated by benign cyclic neutropenia
3	M	13	Multiple simple tics (face, extremities, trunk); mild coprolalia, moderate vocal tics	150	No change	White blood cell count dropped to 4,100/cu mm (polys=900); clozapine
4	F	12	Multiple simple tics (limbs, trunk) and complex tics (face), moderate coprolalia, severe vocal tics	300	No change	Symptoms exacerbated at low doses; marked somnolence
5	F	19	Multiple mild simple tics (mouth, face), mild coprolalia, mild vocal tics	500	No change	Increased salivation; moderate somnolence
6	F	12	Multiple simple and complex tics (face, trunk, extremities), associated choreoathetoid movements, mild coprolalia, mild vocal tics	375	No change	Symptoms exacerbated at low doses; pulse rate frequently > 130/minute; marked somnolence
7	M	12	Mild simple tics (face) and rare complex tics (extremities), mild coprolalia, mild vocal tics	500	No change	Moderate somnolence
Huntington's disease						
8	M	52	Mild choreoathetoid movement disorder, mild dementia	60	No change	Disorientation, confusion, slurred speech; severe, debilitating orthostatic hypotension
9	M	54	Moderate choreoathetoid movement disorder, mild dementia	200	Decreased abnormal involuntary movements; no change in mentation	Marked somnolence; "less vigor"
10	F	48	Severe choreoathetoid movement disorder (including hemiballismic movements), moderate dementia	500	Decreased abnormal involuntary movements; no change in mentation	Marked "rebound" after cessation of therapy with disabling abnormal involuntary movements, moderate somnolence, frequent hypotension
Drug-induced disorders						
11	F	22	Severe (often disabling) dystonic trunk and limb postures, facial grimacing, blepharospasm, intermittent retrocollis, frequent opisthotonic postures, choreoathetoid movements of extremities, toe walking	425	No change	Mild somnolence
12	M	46	Persistent torticollis (except when supine), hypertrophied neck muscles, involvement of axial muscles (right>left)	300	No change	Marked somnolence

enced clinically significant side effects that compromised their functioning. Although clozapine was useful in treating the symptomatic abnormalities of these 2 patients, further studies were not undertaken due to the emerging evidence about the drug's potential risks. In addition, both patients had less drug-related impairment when taking haloperidol as a maintenance medication.

Simpson and associates (9) have reported the suppression of involuntary movements in adult patients who show the more typical syndrome of persistent drug-induced dyskinesia. They used up to 900 mg of clozapine in treating some individuals. In contrast, the 2 patients with atypical dyskinesia in this study showed no therapeutic response. The finding that 1 Gilles de la Tourette patient showed a reduction in simple tics while the others failed to demonstrate any change may be indicative of heterogeneous pathophysiologies in this syndrome.[2]

All but 1 of the Gilles de la Tourette patients in this trial had therapeutic responses to haloperidol. Similarly, haloperidol lessens many of the movements of patients with Huntington's disease and persistent drug-induced dyskinesia. Both haloperidol and clozapine possess antipsychotic properties. However, data from this study suggest that while clozapine suppresses some kinds of abnormal involuntary movements, it is ineffective in treating others. When one is studying the pathophysiology of diseases of the central nervous system, it will be essential to carefully consider assumptions about neurochemical mechanisms based principally on the action of diffusely acting pharmacological agents.

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Complex Partial and Pseudoseizure Disorders

BY RONALD A. REMICK, M.D., AND JOHN A. WADA, M.D.

The differentiation of ictal and nonictal seizure disorders is difficult, particularly in patients suffering from partial seizures with complex symptomatology. The authors state that observation of a patient's habitual seizure during EEG recording is the ideal diagnostic tool and describe their method of seizure activation with sphenoidal electrodes and simultaneous audiovisual monitoring. They emphasize the necessity for early, aggressive treatment of both ictal and nonictal seizure disorders, point out risks to the patient if the incorrect diagnosis is made, and urge further cooperation between psychiatrists and neurologists in this borderland area.

SEIZURE ACTIVATION with sphenoidal EEG electrodes and simultaneous audiovisual monitoring developed as an aid in evaluating patients who have a seizure disorder that has not responded to medication and who are being assessed for possible neurosurgical intervention (1). This procedure is also of significant diag-

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nostic assistance in differentiating the ictal and nonictal complaints of patients with seizure disorders, particularly partial seizures with complex symptomatology.

The diagnosis of complex partial seizures, formerly known as temporal lobe epilepsy or psychomotor epilepsy (2), is based on clinical symptomatology, and this diagnosis is supported by the presence of specific electroencephalographic abnormalities. Only the lateral convexity of the cerebral hemispheric surface is accessible to traditional scalp EEG tracings, which has led to the development of a method for electrographic monitoring of the basal cerebral surface. This latter procedure significantly adds to the evaluation and diagnostic assessment of complex partial seizures, as surface EEG monitoring often fails to record irritative disturbances in the more mesial and inferior surfaces of the cerebral hemispheres. Nasopharyngeal electrodes are relatively easy to apply but are subject to several disadvantages (3). Monitoring through sphenoidal electrodes is subject to less artifactual contamination and is preferred for identification of abnormalities, particularly in the anteromesial aspects of the temporal lobes (4, 5).

When focal cerebral abnormalities are suspected, as in a patient with complex partial seizures, seizure activation techniques may provide information as to the site of onset of the habitual seizure. Although traditional activation techniques such as hyperventilation, photic stimulation, and sleep are part of the routine evaluation of patients with a seizure disorder, often pharmacological activation is necessary for further diagnostic clarification. The observation of a patient's habitual seizure during EEG recording is the ideal for diagnosis and treatment. This is essential for those patients considered for neurosurgical treatment, and it greatly assists the diagnosis and management of patients with complicated clinical pictures.

The procedure we use in selected cases is as follows. Under local anesthetic bilateral sphenoidal electrodes are inserted transcutaneously under the zygomatic arch to rest near the foramen ovale (5). Fine wire electrodes are then inserted through the needle electrode, the electrode is removed, and the wire is firmly fixed to the skin with adhesive tape. By use of a split screen in our audiovisual studio, simultaneous monitoring of a 16-channel EEG and the patient occurs throughout the procedure.

An intravenous catheter is inserted and a "cocktail" of pentylenetetrazole (Metrazole), 200 mg in 2 cc (10%), and bemegride (Megimide), 90 mg in 18 cc (0.5%), is infused as follows: 5 cc initially, followed by 1 cc every 30 seconds until activation occurs. Positive activation is an abnormal EEG discharge frequently associated with clinical seizure manifestations that is identical to the patient's described pattern of habitual seizure. When activation occurs the procedure is rapidly terminated by intravenous diazepam (10–20 mg).

We believe this procedure may be of great value to clinicians who have cases in which the differential diagnosis between seizure disorder and conversion symptoms or pseudoseizures is difficult.

CASE REPORT

Ms. A, a 24-year-old woman with an 8-year history of seizures and 9 previous psychiatric hospitalizations related to disturbed interpersonal relationships and impulsive behavior, was readmitted to the psychiatric hospital for further assessment of her seizure disorder. Her "fits" were characterized by voices becoming distant; visual illusions such as faces becoming distorted, depersonalization, and "dreamy" sensations; and feelings of nervousness and panic. They had increased to twice daily despite the regular ingestion of anticonvulsant medicines for 2 years.

Ms. A's seizures began when she was 16 after she experienced a traumatic head injury and lost consciousness. She was placed on anticonvulsants, and her seizures decreased in intensity and stopped completely 2 years later. The seizures recurred when she was 22 years old. An electroencephalogram at this time was minimally abnormal. A sphenoidal EEG revealed slow activity in the left temporal region. It was felt that many of Ms. A's complaints were consistent with a complex partial seizure disorder involving the anteromesial portion of the left temporal lobe. Two years of anticonvulsant treatment had not successfully controlled her seizures.

Ms. A was the second of 3 children. Her parents were alcoholics and had separated when she was 9 years old. Her mother had some form of epilepsy, but at times Ms. A was not sure whether in fact her mother was "faking it." Ms. A began to live with her father when she was 14 and was sexually molested by him when she was 16. This was also the time she began to have seizures. Over the next few years she moved back and forth between her mother and father, until she became pregnant by a man she subsequently married.

Ms. A's relationship with her husband was stormy. They were married for 5 years, during which Ms. A had 8 psychiatric hospitalizations after suicide attempts that seemed to occur at times of severe marital strife.

On admission, Ms. A was detached but clear in her speech. There were no abnormalities in her mood, mental content, or sensorium. Physical and neurological examination was normal. During her hospitalization, a seizure activation with sphenoidal electrodes and audiovisual monitoring was obtained. After 14 cc of the pentylenetetrazole-bemegride cocktail was infused, Ms. A suddenly tensed up, her face became flushed, and tears welled up in her eyes. Her mouth became distorted, and her verbal responses to inquiry became extremely unclear. She stated that she was experiencing perceptual alterations so that things did not appear real around her and voices sounded far away. This state lasted well over 2 minutes and during this time the EEG showed no focal or lateralizing features. Throughout the monitoring there were no specific electrographic events that were related temporally to Ms. A's clinical subjective experiences. She eventually received 70 cc of the cocktail, a dose almost 3 times that required to activate habitual seizures in our patient population. Ms. A stated that she had in fact had her habitual seizure during this procedure.

We discussed the results with Ms. A and explained that she had an electrographic abnormality but that her clinical subjective phenomena did not correlate with the EEG findings. We suggested that she explore her difficulties further with psychiatric assistance, and during the last year she and

her husband have continued in outpatient conjoint psychotherapy. There has been a dramatic reduction in the number of her seizures, even with reduced intake of her anticonvulsant medicines.

DISCUSSION

The purpose of this report is to make others aware of a procedure that may help to differentiate ictal and nonictal symptomatology. We also hope this report will call psychiatrists' attention to complex partial seizures, a disorder in the borderland between neurology and psychiatry.

Although conversion symptoms are not seen by psychiatrists as frequently today as they were in the past, they are still a common occurrence on neurology and general medicine wards. Differentiating complex partial seizures from pseudoseizures can be quite difficult and requires the physician to have a thorough knowledge of the clinical manifestations of seizure disorders (6, 7). That patients with psychiatric disorders have a high percentage of EEG abnormalities adds to the diagnostic dilemma (8, 9).

It is not unusual to see a case like the one described above in clinical practice. Ms. A had an episodic behavioral pattern, nonspecific EEG abnormalities, and a possible family history of a seizure disorder. Her characterological problems were consistent with the observation that patients with seizure disorders, particularly complex partial seizures, frequently have psychiatric disabilities (10, 11).

Did Ms. A have an ictal event with no electrographic correlates? If more midline or deeper brain structures initiated the seizure, EEG abnormalities may not have been recorded, but the clinical pattern would have been quite different. The extremely large dose of convulsant medicine would certainly produce EEG changes and/or induce a generalized convulsion in a patient with a genuine seizure disorder. A review of our patient's records showed nonspecific EEG changes only, and her subsequent positive response to psychotherapy argues for a pseudoseizure disorder.

The separation and clear identification of seizure disorder and psychiatric disorder is most important because the treatments for each are quite different, and early intervention is paramount in both illnesses. We are becoming aware through kindling studies (12–14) that untreated repetitive intermittent electrical stimulation of subcortical structures may lead to focal seizures and, eventually, generalized convulsions. These generalized convulsions are the result of an electrical stimulation that was previously subthreshold. Thus, not recognizing a seizure disorder and failing to institute prompt treatment may result in more widespread irritative cerebral activity, which will be more difficult to treat later. Because patients with complex partial seizures may first consult a psychiatrist, it is imperative that psychiatrists be familiar with this disorder.

The other side of the coin applies as well. Clinical theory would suggest that some types of psychopathology become harder to eradicate the longer they are left untreated (15). Maladaptive patterns become entrenched, and patients become less susceptible to change over time. It would seem that patients would be more accessible to change with early intervention and treatment, and this point is emphasized in the preventive psychiatry literature.

Other difficulties may result if a seizure disorder and psychopathology are not separated. Anticonvulsant drugs, like psychotropic medications, are not innocuous agents, and serious side effects can occur with long-term use (16). Thus, they should only be used when clearly indicated. Another problem would be the demoralization of patients treated for an emotional problem when a physical disorder is the root of the problem. In later reports we will document how, in fact, psychiatric treatment caused an iatrogenic psychiatric disorder in patients with complex partial seizures.

We strongly believe that separating the ictal from the nonictal can only be done when psychiatrists and neurologists work closely together. To do this, the neurologist must be more cognizant of just what a psychiatrist can and cannot do. The patient described had always had various functional labels during her past treatments in addition to her neurological disorder, but it was not until a joint approach was undertaken that meaningful progress was made.

Likewise, psychiatrists must be more aware of what neurologists can offer. A good example is in the field of electroencephalography. A normal EEG does not rule out a seizure disorder. If a clinician suspects complex partial seizures, he or she should pursue this hunch vigorously. This would mean obtaining, in addition to a routine EEG, a collateral history, a sleep electroencephalogram, perhaps sphenoidal and/or nasopharyngeal monitoring, and at times a procedure to monitor the habitual seizure. Although a normal EEG does not exclude a seizure disorder, an abnormal one does not mean epilepsy is present. Electrographic abnormalities are frequent in psychiatric populations (17), but these patterns are usually nonspecific. The diagnosis of a seizure disorder should be based on clinical symptoms, and the EEG should only assist in the diagnosis.

We believe that the seizure activation procedure described is one neurological development that may be helpful to psychiatrists. Sphenoidal electrodes and activation techniques have been shown to be both safe and reliable in the hands of clinicians familiar with these techniques (18). We have outlined the benefits of the correct diagnosis and treatment and the risks to the patient if the incorrect diagnosis is made.

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Schizophrenic Patients and Schneiderian First-Rank Symptoms

BY MOSES PREISER, PH.D., AND WILLIAM JEFFREY, M.D.

Differences between schizophrenic patients with Schneiderian first-rank symptoms (FRS) and those without FRS at the time of hospitalization indicated FRS were associated with behavior consistent with feelings of passivity and helplessness. The two groups responded equally well to treatment in a therapeutic community. Findings support the contention that FRS are not pathognomonic or prognostic in schizophrenia.

SCHNEIDER (1) has described 11 first-rank symptoms (FRS), which he suggests are pathognomonic of schizophrenia when toxic or organic etiology has been ruled out. These symptoms have been used diagnostically in Europe and have recently been the focus of research efforts in the United States. The FRS are the presence of delusional perceptions (normal perceptions given a sudden and idiosyncratic interpretation that cannot be understood in the general context of the patient's ideation); three forms of auditory hallucinations (voices speaking one's thoughts out loud, voices arguing, and voices commenting on one's actions); and seven types of delusions (sensations or other bodily changes imposed by an outside influence, thoughts taken away from one's mind, thoughts inserted into one's mind, one's thoughts being broadcast, one's emotions not being one's own, overwhelming impulses for action imposed by an external source, and actions not one's own).

An initial optimism in the use of FRS as definitive indicators of schizophrenia (2, 3) has lessened, and current studies show their prevalence but not pathognomonicity in schizophrenia (4–8). Because these

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symptoms have been proposed as pathognomonic of schizophrenia, it has been assumed they represent severe psychopathology; this assumption has led to evaluations of their prognostic value.

Taylor (9) first presented data suggesting that the presence of FRS in schizophrenia indicates poor prognosis, but after a further study (10) he determined there was no difference in acute treatment response (measured on a 0–7 scale) once a definite, not presumptive, schizophrenic diagnosis was established.

McCabe (11) felt schizophrenics with FRS often fell in the group of schizophrenics considered to have poor prognosis. Results from the rigorous Washington Field Research Center of the International Pilot Study of Schizophrenia showed no poorer prognosis for schizophrenics with FRS than for those without after 2 and after 5 years (12, 13).

There have been attempts (14, 15) to determine which patients do least well (and who might even react adversely) on intensive short-term treatment units. The question of course and outcome for schizophrenics with FRS on such units has not been fully explored. If schizophrenics with FRS have more severe psychopathology and perhaps psychopathology less amenable to treatment than schizophrenics without FRS, channeling them to less intensive treatment programs might be more effective use of costly treatment.

The present study was undertaken to explore two questions. 1) Do schizophrenics with FRS have more observable pathologic behavior than schizophrenics without FRS? 2) Do schizophrenics with FRS have a poorer response to an intensive short-term treatment program than schizophrenics without FRS?

METHOD

The study was conducted on a 24-bed therapeutic community ward in a large municipal hospital. The patients participated in self-government, and the various therapeutic modalities on the ward included individual and group psychotherapy, psychodrama, and a variety of task-oriented activities. The majority of the patients were medicated.

In most instances, patients were referred from the acute wards of the psychiatric hospital. These were patients who were judged to need hospitalization of at least 2 weeks' duration and to have the potential to profit from the intensive therapeutic experience offered. Maximum stay on the ward was 3 months and the average stay was 6 weeks. More detailed description of the ward can be found in reports by Winston and Papernik (16) and Jeffrey and associates (17).

Evaluations

During a 6-month period all patients admitted to the ward were evaluated in the following ways:

1. One of two nurses completed the Psychotic Inpatient Profile (18) for each patient on admission to the ward and again at discharge. The Psychotic Inpatient Profile (PIP) is an instrument consisting of 74 statements describing manifest ward behaviors and 24 statements describing patients' self-reports. Nurses' ratings of each statement's applicability to a given patient are summed up to yield 12 subscores, each of which reflects a particular dimension of manifest psychotic behavior. Specifically, the subscores reflect excitement, hostile belligerence, paranoid projection, anxious depression, motor retardation, seclusiveness, inability or unwillingness to care for oneself, psychotic thought or motor disorganization, grandiosity, perceptual distortion, depressive mood, and disorientation.

2. When the patients were admitted to the ward and again on discharge, the recreation therapist evaluated them on a 5-point social participation scale that measured a combination of frequency and quality of social interactions.[1]

3. On admission and again at discharge, each patient was evaluated by one of two group therapists[2] on a 5-point scale measuring degree of impairment of functioning (i.e., ability to carry out the basic responsibilities of everyday life.)[1]

4. Each patient's individual therapist evaluated the patient's symptomatology for the presence or absence of first-rank symptoms as enumerated and defined by Mellor (3), and this assessment was reviewed and corroborated by a senior attending psychiatrist.

5. Discharge diagnosis was determined by the individual therapist in consultation with the attending supervisor using Bleulerian (20) and/or ego function (21) criteria, as is customary on the ward.

Evaluations 1, 2, and 3 of the schizophrenic patients were subjected to statistical analyses comparing those with first-rank Schneiderian symptoms (FRS-positive) and those without such symptoms (FRS-negative). The social participation and impairment of functioning measures were subjected to a chi-square analysis. The PIP data were analyzed by one-way ANOVAs comparing the FRS-positive to the FRS-negative groups on each of the 12 subscores at the time of admission. Discharge subscores were subjected to an analysis of covariance in which the respective admission subscores were used as covariates to ensure that significant differences found would reflect response to the treatment program rather than possible preexisting differences between the groups.

RESULTS

Of the 88 patients admitted to the ward over a 6-month period, 52 were given a discharge diagnosis of

[1]The 5 rating statements were identical to those published by Sanders and associates (19, pp. 252, 255). However, in our study the ratings were simply assigned numbers from 1 to 5, unlike the more complicated system used by those authors.

[2]On an independent sample of patients, an interrater reliability of .83 was obtained.

schizophrenia. Of these 52, 25 exhibited at least one Schneiderian first-rank symptom, and 27 exhibited none. Five patients not diagnosed schizophrenic also exhibited FRS. These patients were not included in any of the subsequent analyses reported in this study. The FRS-positive and FRS-negative groups were similar in their demographic composition. The mean age was 25.6 years for the FRS-positive group and 23.3 for the FRS-negative group ($t=1.36$; $p>.10$). The number of black, white, and Hispanic patients was 14, 7, and 4, respectively, in the FRS-positive group and 17, 8, and 2, respectively, in the FRS-negative group ($\chi^2=.95$; $p>.10$). There were 13 males and 12 females in the FRS-positive group and 14 males and 13 females in the FRS-negative group ($\chi^2=.01$, n.s.).

All discharges that occurred before the date agreed on between patient and therapist were considered premature. In the FRS-positive group 4 patients left prematurely and in the FRS-negative group 6 left prematurely, a nonsignificant difference ($\chi^2=.25$; $p>.05$). The groups did not differ significantly in their length of stay (premature discharges were excluded in this analysis); means were 59.6 days and 56.7 days for FRS-positive and FRS-negative groups, respectively ($t=.17$, n.s.).

Evaluations

1. Analysis of variance of the patient's 12 PIP scores at the time of admission yielded two subscale scores that differed significantly. The patients in the FRS-positive group were evaluated significantly higher on the anxious depression subscale ($F(1, 50)=4.02$; $p<.05$) and lower on the excitement subscale ($F(1, 50)=4.33$; $p<.05$). Because it seemed paradoxical that one group should be both more agitated (as seemed implicit in the first finding) and less excited (as suggested by the second finding), individual analyses were run for the items in each scale. The items that significantly differentiated the two groups were, in fact, not contradictory. Thus, from the anxious depression subscale, the FRS-positive group scored significantly higher on the following items (including differences at $p<.10$): number 18, "looks worried or nervous" ($t=3.54$; $p<.01$); number 9, "shows real sadness in face and posture" ($t=2.37$; $p<.05$); number 42, "appears frightened" ($t=1.77$; $p<.10$); number 29, "looks tired and 'all worn out' " ($t=1.70$; $p<.10$). From the excitement subscale the FRS-positive group scored lower on the following items: number 14, "tries to attract attention to self; shows off" ($t=1.97$; $p<.10$); number 23, "jokes, talks, or laughs excitedly; seems high" ($t=1.79$; $p<.10$); number 73, "acts superior to other patients (conceited and boastful)" ($t=1.79$; $p<.10$); number 59, "shouts and yells" ($t=1.75$; $p<.10$). Analyses of the PIP evaluations at the time of discharge for the 12 subscales yielded no significant differences.

2. Comparing the evaluations for the two groups on the social participation scale yielded a significant difference at the time of admission ($\chi^2=10.30$; $p<.05$). The modal rating for the FRS-positive group on admission was number 4: "Prefers to be by himself. Actively avoids contact with others whenever possible but will respond when pressed. Has no recognizable friendships. May have close relationships with family members, but on examination these prove to be pathological (e.g., excessive dependence, etc.). Interaction is infrequent or frequent but inappropriate." The modal rating for the FRS-negative group on admission was number 3: "Participates to a limited extent. Tends to keep his distance from others but will respond appropriately at a superficial level. Has few (if any) genuine friendships, tends not to become deeply involved. Contacts are neither numerous nor frequent. Very few group contacts. Neither seeks nor actively avoids contact with others. Does not initiate conversations." No significant differences between the groups' ratings on this measure were found at the time of discharge.

3. Comparing the evaluations for the two groups on the impairment of functioning scale yielded no significant differences either at the time of admission ($\chi^2=3.18$; $p>.10$) or at the time of discharge ($\chi^2=1.54$; $p>.10$).

Additional Data

In conjunction with another study on the same ward, additional data regarding rehospitalization within 6 months of discharge was obtained for 37 of the patients included in the present study. In the FRS-positive group, 7 of 19 required rehospitalization within 6 months of discharge, and in the FRS-negative group, 6 of 18 required rehospitalization within the same period (1 of the 6 had committed suicide). The difference in rehospitalization rates between the two groups was not significant ($\chi^2=.27$; n.s.).

CONCLUSIONS

Differences found between a group of schizophrenics displaying Schneiderian first-rank symptoms and those without such symptoms were evident only at the time of admission. These differences included the greater degree of worry, sadness, fright, and tiredness evident in the behavior of the FRS-positive group as opposed to the more boisterous and grandiose behavior displayed by the FRS-negative group (as observed and rated by the nursing staff). These differences in behavior may reflect a more intense experience of helplessness and passivity that would be consistent with a concept of more severe ego-boundary dissolution implicit in many of the first-rank symptoms (forced actions, thoughts broadcasted, etc). With the amelioration of symptoms and with recompensation (which both groups were equally able to achieve), significant differences between the groups could no longer be found on any behavioral dimensions measured.

The differences found at the time of admission on the social participation scale seem compatible with the above mentioned behavioral differences; the more

worried and frightened FRS-positive group tended to be more socially withdrawn, and the more boisterous and grandiose FRS-negative group tended to have impaired but more frequent social interactions.

On a variety of measures of course and outcome (length of hospital stay, degree of recovery measured globally as well as in a number of specific areas of functioning, and need for rehospitalization within 6 months) the data corroborate and extend previous findings that among schizophrenics whose diagnosis is established the presence of Schneiderian first-rank symptoms does not indicate poorer response to treatment. Therefore, the presence of first-rank symptoms should not be used as a basis for excluding schizophrenic patients from intensive treatment because of apprehension of poor outcome.

Lastly, 5 of the patients in the overall population on the ward at the time of the study exhibited Schneiderian symptoms but were not diagnosed schizophrenic. These patients represented 9% of the patients with Schneiderian symptoms and 14% of the patients not diagnosed schizophrenic. These percentages are consonant with most previous findings and indicate that first-rank symptoms are prevalent in but not pathognomic of schizophrenia.

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Patient Access to Medical Records on a Psychiatric Inpatient Unit

BY EUGENE J. STEIN, PH.D., RONALD L. FUREDY, M.D., MARY JANE SIMONTON, R.N., AND CYNTHIA H. NEUFFER, M.S.W.

The authors studied the effects of patient access to medical records during hospitalization in a psychiatric unit of a community general hospital. Questionnaires were completed by about 20 staff and 88 patients, and records were compared with those from an earlier period to note any changes in the written record. Patients reported feeling better informed and more involved in their treatment, and staff said that they became more thoughtful about their notes in the chart. The availability of staff seems crucial to this process and facilitates the working alliance.

ALTHOUGH IT has been suggested that patient access to medical records might result in better informed and more actively involved patients, rarely do patients have free access to their medical records during hospitalization (1). Issues such as privacy, confidentiality, costs, accountability, efficiency, patients' rights, and malpractice have fostered a lack of mutual trust in the doctor-patient relationship. However, in psychiatry this trustful relationship, the working alliance, is considered the very basis of therapy. Redlich and Mollica (2), for example, have implied that psychiatrists are ethically "bound by an obligation" to establish such a "partnership." They go on to suggest that "informed consent is the basis of all psychiatric intervention . . ." Shenkin and Warner (3) have suggested, in effect, that patient access to the medical record might result in the patient's being better informed and a more active partner in treatment. These authors implied that patient access to the record would improve the physician-patient relationship, with increased satisfaction for both parties.

In 1973 Shenkin and Warner proposed legislation to require that a complete and unexpurgated copy of all medical records, both inpatient and outpatient, be issued routinely and automatically to patients as soon as the services provided are recorded" (3, p. 688). They felt this proposal might alleviate four serious problems—poor quality of care, unsatisfactory physician-patient relationships, lack of continuity, and excessive bureaucracy—that have been associated with "widespread" dissatisfaction with the functioning of the medical care system. Although Shenkin and Warner were impressed by the generally favorable response to their proposal (4), a search of the literature suggests that few facilities have been willing to put the proposal into practice, and in the more popular medical literature, one finds articles such as "The Patient's Chart Is None of the Patient's Business" (5).

Despite considerable armchair speculation of this sort, our review of the literature revealed no studies of psychiatric patients having access to their records and only two reports in other specialties (6, 7).

The staff of a psychiatric inpatient unit of a community general hospital disagreed as to whether patients should be able to read their records while in the hospital. Some argued that patients would not understand the information or would in some way be harmed by it. Some felt that it would be very difficult to communicate information such as diagnoses. Others felt that patients had a right to know what was being written about them and that openly available records would be more consistent with the treatment philosophy of a therapeutic community, which emphasizes openness and responsibility. The staff agreed to permit patients access to their records on a trial basis provided that the new policy's impact on patient care was evaluated. An interdisciplinary task force consisting of the authors was appointed to study patient access to medical records.

METHOD

Every evening the entire patient group met, and each patient was given his or her current medical record to read. At least one nursing staff member was available to help the patients understand terminology and decipher handwriting. The record was available only to the individual patient and the staff; it was available to the patient for informal review throughout the day, except during doctors' rounds. Family members were not allowed to read the medical records, and pa-

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tients were not allowed to read each other's records. This procedure was followed for a period of approximately five months, during which the following surveys were done.

1. Patient questionnaire. The questionnaire was given to 102 of the 106 patients consecutively admitted to the psychiatric inpatient unit of the hospital. This was usually done by the psychiatric social worker one to two days before discharge. Of the 102 patients, 4 were discharged against medical advice and 5 were transferred to other units without completing the questionnaire. Three patients were unable to complete the questionnaire because of intellectual or language difficulties, and 2 refused to participate in the study. Thus 86% (N=88) of the patients who received the questionnaire completed it.

2. Staff questionnaire. Twenty staff members of the inpatient unit completed this questionnaire during the first month that patients had access to their records. Nineteen staff members completed the questionnaire when it was readministered during the fifth month of the study.

3. Record review. Two random samples of 25 medical records each were drawn from the 1975 period when patients had daily access to their records and from a comparable period in 1973–1974 when patients did not have such access. Two nursing students performed part of the review, and we performed the other part. We had an average interrater agreement of 93% and the nurses, 92%.

RESULTS

Patient Response[1]

The vast majority of the patients (77, or 92%) were in favor of open medical records. They felt that access to their medical records during hospitalization helped them to better understand their problems (75, or 86%) and take a more active role in their treatment (72, or 85%). To a lesser extent, most of the patients said that access to their records afforded them an opportunity to correct inaccurate information (54, or 68%). Most patients felt comfortable about reading their records (72, or 84%), and a majority felt more self-confident as a result (60, or 71%). However, a substantial minority felt more pessimistic after reading their records (26, or 32%) and roughly half of the patients said they were upset by some of what they read (43, or 51%). Although the patients felt better informed, they did not always like what they read.

Although half of the patients were upset by some statements, very few felt that the staff had made untrue statements about them (16, or 19%) or had avoided noting certain things about them because of the open records (9, or 11%). In fact, roughly two-thirds of the patients (55, or 66%) felt that the staff became more accurate in what they wrote in the records. Patients were equally divided on the question of whether the staff became more respectful (41, or 51%). Slightly more than half of the patients thought open records had some impact on what the staff wrote (47, or 56%), and the vast majority of the patients thought that open records made some difference in their treatment (73, or 86%). Few feared for the confidentiality of their records (10, or 12%).

Staff Response

Ninety percent of the staff (N=18) were favorably disposed to open medical records after initial exposure. After approximately five months of experience with daily patient access to records, 79% (N=15) still wanted to continue this policy. The vast majority of the staff said they were comfortable with open records (19, or 95%) and thought this was a useful therapeutic tool (17, or 89%). Initially, 35% (N=7) thought open records had little impact; however, after more exposure only 11% (N=2) came to this conclusion. Most felt that they became more accurate in their observations (13, or 68%) and more thoughtful in their conclusions (about 15, or 79%) because of open records. A sizable proportion did not record information that they felt would upset the patient (4, or 20% initially and 11, or 58% finally), and they found other ways to communicate this information to staff, usually verbally during rounds and in shift-to-shift reports. It is important to note that this information pertained not to diagnosis but to intimate details of the patient's personal life. In addition, an increased number of staff (6, or 33%) reported that they had to seek information from other sources because of open medical records. Despite the tendency for many staff to omit certain information, only 26% overall (N=5) reported that they were less likely to diagnose a patient as psychotic because of open records.

Sixty-five percent of the staff (N=13) thought that open records helped the treatment of most patients, and this proportion increased to 70% (N=14) after five months. Eighty percent (N=16) felt that at least a few patients were helped by open records, and none of the staff felt that open records were generally harmful to patients even after five months of experience with this policy. However, 26% (N=5) knew of at least 1 patient who was harmed by open records. We do not know how many of the patients who participated in this study were thought to have been harmed by open records, but we suspect that there were only 1 or 2. Despite these concerns and changes in the pattern of staff communication, 63% of the staff (N=12) concluded that open records made the chart a more valuable clinical document.

Chart Review

The number of notations of disruptive behavior doubled with open records (mean averages of .68 and 1.55, $p<.10$, t test). The number of cross-outs and erasures appeared to decrease, and the number labeled "error"

[1]Tables presenting the results of the patient questionnaire, staff questionnaire, and the record review have appeared elsewhere (8).

or initialed by a staff member doubled with open records (averages of .52 and 1.00, $p<.05$). No change was detected in the rating of the physician's description of the patient's history and physical examination. These results appear to be consistent with results of the questionnaires.

The increase in the number of cross-outs and erasures labeled "error" and the decrease in the number of cross-outs and erasures not labeled suggest that the staff may well have become more thoughtful and that the patients may indeed have been able to correct some inaccuracies. The increase in the number of notations of disruptive behavior may account for some of the upset feelings noted in patient questionnaires.

DISCUSSION

The results of this study are strikingly similar to those of Golodetz and associates (7) and Bouchard and associates (6), who gave sections of the problem-oriented record to rehabilitation patients. The methodology is also similar. Bouchard and associates found that "in the vast majority patients are reassured and motivated by reviewing their own health care data. Ninety-seven percent of patients feel relieved and less anxious after reviewing their own records" (6, p. 46). Golodetz and associates found that "patients were generally comfortable about reading the record, found it educational and appreciated the trust implied. No substantial difficulties arose. Few records were expurgated" (7, p. 79). We found that even though patients were sometimes upset by what they read, they were generally comfortable with reading their records and felt better informed and more involved in their treatment. The staff felt that patient access to medical records was therapeutic. The staff tended to omit certain details from the record, but they also became more accurate and thoughtful in what they wrote, and the patients had a chance to correct inaccuracies.

All three studies suggest positive benefits including enhanced patients' rights, improved treatment, and improved patient-staff relationships. It appears that few patients were harmed by this policy, and the benefits seem to outweigh the costs. We are aware of no study showing a significant negative impact of patient access to the medical record. One must caution that all three studies took place in settings where the team approach was used. The policy of open medical records is not without difficulties. Specifically, it seems crucial that availability of the record be accompanied by availability of the staff to discuss the record. This process, in itself, appears to be useful in facilitating a working alliance between patient and staff.

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Response of Patients to Participation in Psychiatry Board Examinations

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The patient who volunteers to be a subject for Part II of the certifying examination in psychiatry and neurology is an indispensable but surprisingly invisible participant in a highly controversial process. A survey of the attitudes of 78 patients toward this experience revealed that although they experienced a significant degree of stress, the overwhelming majority viewed their participation in strongly positive terms. The patient's loyalty to the host institution and the manner in which the patient is prepared for the experience appear to be the major factors in determining a positive outcome.

THE PATIENT WHO volunteers to be a subject for the American Board of Psychiatry and Neurology (ABPN) Part II examination (the oral portion) is an indispensable but surprisingly invisible participant in a dramatic and increasingly controversial process. Although the impact of this experience on the candidate has been the subject of much debate (1–3), the impact on the patient has received little attention in the psychiatric literature. To our knowledge the only article in the literature consists of an unreplicated study published in 1972 by Eveloff and McCreary (4), in which the authors found that a middle-class, relatively well-educated group of patients suffered no serious consequences as a result of their participation as Board subjects and tended to view their experience as "slightly positive." Given the increasing emphasis on patients' rights and the rapidly escalating criticism of the value of the Part II examination (5), further investigation of this issue appeared warranted in order to ensure that the examination process was not detrimental to the well-being of the patient.

This report will present the findings of a survey of patients' attitudes toward their participation in the present ABPN oral examination procedure. It is our hope that these findings will prove useful in clarifying one of the many complex issues that must be resolved in order to determine the future format of the certification process (6).

METHOD

The subjects for this study consisted of 78 randomly selected male patients between the ages of 21 and 65 who were on the active treatment roster of a Veterans Administration health care facility and who volunteered to participate in the patient interview section of the Part II examination held on October 17 and 18, 1977. The overwhelming majority of the patients were high-school-educated, blue-collar workers who had received a primary diagnosis of schizophrenia, affective disorder, or alcohol addiction.

Each patient, with the exception of 7 patients on an inpatient research unit, was contacted by a member of the organizing committee approximately 12 weeks before the examination and given an explanation of the examination procedure that stressed the benefit to the candidate and psychiatric profession rather than to the patient. All patients were asked to participate in a minimum of 2 examining sessions. If they agreed, they were asked to sign a statement of consent and to select the times most convenient for them. Of the original 83 patients, 5 refused to participate; the most frequent explanation was an inability to take time off from work. Potential subjects were then contacted at 4-week intervals to inform them of any scheduling changes. Approximately 1 week before the examination the patients were again contacted to confirm the appointment times and to inform them of a small reimbursement for their participation. All of the patients who initially agreed to participate did, in fact, do so, and no patient was asked to be a subject for more than 2 examining sessions.

At the conclusion of the patient's first examination he was asked to complete an anonymous 7-item questionnaire before leaving the examining area. The questionnaire, which consisted of 6 structured items and 1

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open-ended question, was developed by us to assess both the amount of stress experienced by the patient and his attitudes concerning his role as an examination subject. All of the 78 participants completed the questionnaire.

FINDINGS

The first item called for the patient to answer yes or no to 3 questions related to the statement, "Some people have said that patients are often asked to do things that they don't agree with or understand." Thirty-eight percent of the respondents indicated that they felt this way in general, 9% felt this way about the interview that had just been completed, and 96% stated that the purpose of the interview was explained satisfactorily.

Item 2, a multiple-choice question, sought to determine how stressful the interview was for the patient. Thirteen percent of the patients found it very stressful; 26%, moderately stressful; 5% minimally stressful; and 56%, not stressful.

The third item asked whether the patient felt that he had been treated with courtesy and respect. Ninety-six percent of the patients answered yes.

Items 4, 5, and 6 were multiple-choice questions that sought to measure the patient's feelings about his participation in the examination and his willingness to be involved in similar activities in the future. Two of the choices for each item had a positive loading and two a negative loading. Of the 234 potential responses to the 3 items, 229 choices were actually made, with only 11 (5%) being negative responses. Of these 11 responses, 6 were contributed by 2 patients on the research ward.

Item 4 asked the patient to choose the answer that best described his feelings about participating in the examinations. Ninety-six percent of the patients selected the positive choices; 30 said that it was "Worthwhile because it helped out the hospital," and 43 said that it was "An interesting and helpful experience to me personally." Three patients selected the negative choices; 1 said that it was "Something I could do without," and 2 said that it was "Tolerable because of the five dollars I was paid for the interview."

The fifth item asked whether the patient would make himself available for similar examinations in the future. Ninety-five percent of the patients selected the positive choices; 29 said that "I would make myself available as a subject whether they paid me or not," and 44 said that "I would do it again because it was important to me and the hospital." Four patients selected the negative choices; 3 chose the statement, "I would make myself available only if they paid me," and 1 the statement, "I wouldn't participate the next time."

Item 6 asked the patient to indicate how he felt about participating in activities that did not directly contribute to patient care. Ninety-five percent of the patients selected the positive choices—"They provide an interesting change in the normal routine" (N=14) and "They serve an important purpose and therefore should be encouraged" (N=58). Four patients selected the negative choices—"They don't contribute to patient care and therefore should be banned" (N=1) and "They don't mean much to me one way or the other" (N=3).

The open-ended item, number 7, invited the patient to suggest ways in which future examinations could be improved. Of the 28 responses, 12 were expressions of support for the activity (e.g., "Very good," "Keep up the good work"). Six patients commented on the beneficial nature of the examination ("Good for the upcoming psychiatrists," "Beneficial for people with problems like mine," "It shows the hospital is improving itself"). The remaining 10 comments were related to aspects of the interview. Although 2 of these were decidedly positive, the other 8 suggested that the respondents had negative feelings about the examining session. Illustrative of this group are such statements as "Make it longer," "Start the interview on time," "The doctors asked the same questions," and "The doctors should be better acquainted with the history in order to ask more helpful and logical questions."

DISCUSSION

The results of our survey indicate that although 39% of our subjects described their experience as moderately to extremely stressful, the overwhelming majority viewed their participation as a strongly positive and potentially beneficial personal experience. Almost all of them felt that they had been treated with courtesy and respect, and they indicated a willingness to participate in future examinations regardless of possible compensation. They took pride in the fact that they had been instrumental in a process that was beneficial to both their hospital and the education of the candidate.

These strongly positive responses confirm and strengthen the findings reported 7 years ago by Eveloff and McCreary (4). Despite significant differences in setting, patient population, methodology, response rate, and questionnaire format, the basic similarity in these two sets of results strongly supports the validity of these findings and suggests their applicability to other groups of patient-subjects.

In comparing the actual findings of these two studies, we found that the factors which seem to have been most responsible for the significantly higher frequency of positive responses in our sample were the loyalty of our patients to the institution and our emphasis on the preparation of the patients through continuing personal contact with a member of the staff responsible for the organization of the examination. During this preparatory process the patient was encouraged to feel that he was an important contributor to the success of the examination process. In addition, the presentation of the examination as a source of benefit to others en-

abled the patient to "use" his difficulties in an adaptive manner, thus increasing his sense of self-esteem.

Although our sample was small, confirmation of the importance of this preparatory process is suggested by the fact that most of the negative responses to the experience were reported by the patients on the research unit, who were not directly exposed to this approach. It is also interesting to note that after the interview, some of our patients spontaneously reported evaluations of the candidates' skill. Issues of confidentiality precluded an investigation of this observation, but it was our impression as well as that of some examiners that the patients' assessments were often quite accurate, and it seemed possible that the degree of stress reported by some of the patients was related to the degree of skill exhibited by the candidate.

CONCLUSIONS

If the patient is viewed as an important contributor to the success of the endeavor, his or her participation as a subject in the ABPN Part II examination can be a positive, even beneficial experience despite a significant degree of stress. Although many questions regarding the value of this examination remain unanswered, the response of most patients is perhaps best expressed in the words of the patient who concluded his questionnaire with the statement, "More examinations!"

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1979 Fall Committee Meetings to Be Held in Baltimore

The 1979 APA Fall Committee Meetings will be held September 19–22, 1979, at the Baltimore Hilton Hotel in Baltimore, Maryland.

Educating Staff to Manage Threatening Paranoid Patients

BY G.A. WILLISTON DI BELLA, M.D.

The threatening paranoid patient is one of the most frightening and difficult patients to treat. The author points out the need for all levels of hospital staff to become more adept at recognizing their own feelings, which often keep them from appropriately managing these patients. He summarizes several crucial points in teaching staff to cope successfully, dividing the process into diagnosis, management of one's emotions, and practice of appropriate therapeutic techniques.

FEW PATIENTS stir up as much fear and anxiety as those who are hostile and paranoid (1, 2). The patient's threats of violence, violent acts, and terror all induce fright among all levels of hospital staff. Too often staff members have not been trained to prevent violence and maintain treatment when confronted with threatening patients (3).

The psychiatrist can take a role in changing this situation. He or she can educate the staff to deal with the hostile paranoid patient. The three-step process outlined in this paper is based on my years of experience and on a review of the literature. Although my focus is on paranoid patients, many of these recommendations can be applied to all threatening patients. To make this education maximally effective, the psychiatrist must be attuned to the times when the staff is most receptive, i.e., at times of crisis. An example of a crisis is when the staff ask permission to transfer the patient out of their care.

DIAGNOSIS

The first step in helping staff deal with the threatening patient involves enabling them to recognize this type of patient. Too often this process is neglected in multidisciplinary work.

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The meaning of "paranoid" needs to be made clear. The APA glossary (4) defines it as oversuspiciousness. *DSM-II* lists six paranoid diagnoses. As in any syndrome, it is the combination of signs and symptoms that confirms the diagnosis of paranoid pathology. The additional feature in hostile paranoid patients is that they threaten violence or are violent.

To help staff appreciate the diagnostic features of the hostile paranoid patient, the teacher should select actual patients and review with staff specific examples of verbalization and other behavior that show how the paranoid patient thinks and feels.

I have selected from among the paranoid patients I have treated a number who exemplify the seven types of paranoid thinking described by Swanson and associates (1). Some of the examples could fall into more than one category.

Projective thinking. After knocking the ward psychiatrist to the floor one patient said, "God told me to beat him up."

Suspiciousness. To any friendly approach, a patient said something like, "Don't come off like you are my friend when I know you just want me to stay locked up in this miserable jail."

Viewing the world as a hostile place. Particularly frightening to staff, and often indicative of imminent violence, is the "paranoid stare" (I call it "devouring with the eyes"). This stare is a peculiar, penetrating, unblinking look with raised upper and lower eyelids.

Fear of loss of autonomy and control. One patient refused to get involved in activities: "Activities are for children," he said.

Feeling of self as a pivotal point of events. One day the staff gave a patient's refrigerator a much-needed cleaning. In the process, the patient's food was shifted from one shelf to another. Without really looking, this patient exploded violently, saying, "I knew they were out to get me; they took my food."

Grandiosity. At times one patient condescendingly displayed his feelings of self-importance as he expounded on the "one true way" of socialism. Sometimes he tried to show that he was a staff member rather than a patient by helping to manage other patients or doing other ward work.

Delusions. One patient hated medications. He firmly believed that they were poison. Consequently, he did everything possible to avoid receiving medication.

There are many examples of paranoid behavior available to get staff involved in an exploratory process, not only to understand the paranoid patient but also to begin exploring themselves.

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SELF-MANAGEMENT

The second step in learning how to deal with threatening patients is learning how to manage oneself. The therapist, for example, must become aware of, and master, his or her own feelings before he or she can manage a patient.

Staff members often react to threatening patients with fear, anxiety, anger, frustration, helplessness, guilt and hopelessness. Each staff member reacts differently, has habitual ways of dealing with dysphoric feelings, and has different physiological responses to dysphoria. The teacher can help staff members control their reactions and mental mechanisms.

The first help the teacher can provide is to reassure the staff member that strong reactions to hostile paranoid patients are normal. Second, the teacher can outline what happens to staff, perhaps by drawing a graph listing the expected emotional reactions to a hostile patient (fear and anxiety, anger, frustration and helplessness, guilt, and self-doubt) and the subsequent mental mechanisms used to cope with these reactions (denial, withdrawal, identification with the aggressor, displacement, reaction formation, submissive-seductive maneuvers, and projection).

Third, the teacher can review some specific examples of staff behavior that illustrate the mechanisms in action. This allows them to focus in on these and begin to ask themselves what emotion led to what behavior. Examples follow:

Denial. Denial was the mechanism used most often by staff. They "forgot" the patient's episodes of violence, delusions, and hallucinations. Before being beaten up by a patient, one staff member said, "He is just misunderstood; he would never hit me."

Withdrawal. Some staff members were aware that they stayed away from hostile patients because of fear. Others neglected the hostile patients and really seemed to forget that they existed. Others withdrew from certain crucial interactions, e.g., setting firm, fair limits.

Reaction formation. Some staff members took the attitude of, "I don't hate or fear you; I want to mother and rescue you." Taken too far, this reaction would only further agitate the typical paranoid patient.

Displacement of anger. Anger was usually displaced toward authority figures among the staff itself. Staff members protested limit setting and thus encouraged paranoid attitudes in the patient: "Of course, the patient is angry and violent. Who wouldn't be? All he needs is to be free!" Paranoid patients induce extreme divisiveness among hospital staff as well as in their own families.

Acting out. Punitive retaliation is not uncommon among staff members dealing with "bothersome, problematic" patients. The paranoid patient tends to push staff into such acting out. Actually, all patients tend to behave in ways that provoke responses in other people that just confirm their predetermined views of the world.•

Finally, the teacher can make sure that individual staff members do not have to deal with their dysphoria alone. He or she should repeatedly state that therapists are expected to have problems and see to it that opportunities to discuss these with supervisors and in conferences are readily available.

PRACTICAL TECHNIQUES

The final step in ensuring that staff learn to treat hostile patients appropriately is to teach them and help them practice techniques and strategies of managing the hostile paranoid patient.

The therapist must take a scrupulously frank, honest, straightforward, and structured approach to the threatening patient. He or she must not be too warm and friendly or attempt to get close quickly. Touching the patient must be curtailed. Similarly, staff members should not approach the paranoid patient suddenly and rapidly. These patients often attack in response to minimal surprises.

The paranoid patient is exquisitely sensitive to admitting deficiencies and often will not seek out help; thus, the staff will have to take the initiative in offering their resources to the patient. Issues of controlling violence must be discussed very early in therapy. The patient must be strongly encouraged to take responsibility for his or her actions, to become aware of anger mounting to a dangerous degree, and to have contingency plans prepared long in advance to handle this in an acceptable way. The patient must be told openly that in no circumstance is violence acceptable. The staff must make it very clear to the patient what will happen if he or she becomes violent, i.e., the patient's lack of control will be replaced by the staff's firm control. As a general rule, all patients fear and exaggerate unknown "punishments" for breaking rules. They will often break rules just to find out what the "penalty" is.

The patient's family should always be actively engaged in the therapeutic process. They need to learn how to avoid provoking the patient. Hostile patients are more often violent toward close relatives than toward others.

Medication is another crucial area that is usually neglected. The staff fails to consider the implications of the fact that the paranoid patient might really believe that the medication is poison. Too often the staff ask, "What should we do? The patient is on huge doses of medication but is not responding." In the majority of these cases the patient has found an ingenious way not to ingest the medication. The staff should thoroughly explore the patient's feelings about medication and help the patient work through his resentment and/or his feeling that taking medications relieves him of any responsibility for his behavior. A long-acting injectable medication, such as fluphenazine decanoate, might be considered over oral medication.

Managing imminent or actual violence has been fairly well covered in the literature (5, 6). However, a few

points bear emphasizing. The paranoid person does not just explode "out of the blue." There is almost always a period of mounting tension and turmoil and/or excessive angry brooding. Optimally, intercession should occur during this period. Staff will have to know how to restrain a patient, and they will not be able to unless someone trains them, including role playing in the training. Training films are available (7).

When an episode of violence actually occurs, it should be discussed openly and thoroughly with the patient. In addition, a staff discussion should always be held as soon as possible so that staff have the opportunity to identify their fear, ventilate, and then discuss ways of doing better. Otherwise the fright will remain or be rapidly converted and crystallized into inappropriate behavior and attitudes. Similarly, the episode must be discussed with the rest of the patients on the ward.

Staff members may wish to read other suggestions in the literature (1–3, 5–8). A general rule to judge whether appropriate therapeutic techniques are being used is the following: if the staff's behavior consistently produces appropriate patient behavior, it is correct.

Reviewing the information provided here with staff in a concerted way, along with providing specific examples of inappropriate behavior by staff, is essential in the process of enabling staff to deal with the hostile patient in a therapeutic way.

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CLINICAL AND RESEARCH REPORTS

This section contains 1) new research findings, including preliminary data from pilot studies, either clinical or laboratory; 2) worthwhile replication studies; 3) case reports that describe a truly new syndrome or cast new light on established ones; and 4) case reports that indicate a new therapeutic procedure of potential value or call attention to adverse effects of drugs or previously unreported complications of therapeutic interventions. Program descriptions and literature reviews cannot be printed in this section. Criteria for format are listed in "Information for Contributors" in each issue; papers that do not adhere to these criteria will be returned to the author.

EEG Observations During Combined Lithium and Neuroleptic Treatment

BY RICHARD ABRAMS, M.D., AND MICHAEL ALAN TAYLOR, M.D.

In a recent report, Spring (1) recommended EEG monitoring for patients receiving combined lithium and neuroleptic drug therapy on the basis of EEG findings in three patients who purportedly developed encephalopathy as a result of a combination of lithium and thioridazine.

As part of a larger study, we collected EEGs on a sample of patients receiving lithium and/or neuroleptics, in order to provide data on the EEG effects of such drug treatment.

Method

The subjects included all consecutive admissions to an adult, acute-treatment university psychiatric service for 22 months ending May 1976.

Twenty-minute scalp-recorded resting EEGs were obtained in all cooperative patients, usually with sleep. The International 10–20 system and unipolar and bipolar montages were used. EEGs were read by an electroencephalographer (R.A.) who was blind to any information that could identify the patient or indicate psychotropic drug status. All records were classified as normal, borderline, or abnormal, according to the criteria of Gibbs and Gibbs (2). Borderline and abnormal records were subsequently combined for analysis.

Results

A total of 131 patients provided EEGs. Of this group, 68 were drug-free, 17 were taking lithium, 33 were taking neuroleptics, and 13 were receiving lithium and neuroleptics at the time of recording. Each of the 13 patients in the combined-treatment group was receiving haloperidol. Of the 33 neuroleptic-only patients, 30 were receiving haloperidol and 3 chlorpromazine. For purposes of calculating mean dosages of neuroleptics received, chlorpromazine was converted to haloperidol at a 10 to 1 ratio.

The mean lithium dosage for the combined therapy group was 1431 mg/day (range=900–1800 mg/day), with a mean serum lithium level of 0.94 mEq/liter (SD=0.32). The mean haloperidol dosages were 39 mg/day (range=20–60 mg/day) for the combined-treatment group and 38 mg/day for the neuroleptic-only group.

There was no significant difference in the distribution of EEG abnormalities for the four groups or for the 3 drug treatment groups considered alone (see table 1).

Comparisons of the groups separately revealed no differences in EEG abnormalities between the lithium-only and the combined-treatment groups or between the neuroleptic-only and the combined-treatment groups. Finally, there were no significant correlations between serum lithium level and presence of EEG abnormalities for patients in the lithium-only or combined-treatment groups or for the entire lithium sample.

Conclusions

The combination of lithium and haloperidol produced no more EEG abnormalities than either drug given alone or than no drug at all. This finding, which is consistent with our previous report of a lack of clinical neurotoxicity for combined therapy (3), fails to support the need for EEG monitoring of such treatment. It is possible that Spring's patients (1) may have demonstrated anticholinergic delirious states secondary to thioridazine, the most potent anticholinergic

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TABLE 1
EEG Abnormality and Drug Therapy

Drug Treatment	EEG[a] Normal N	Normal %	Abnormal N	Abnormal %
None	54	79	14	21
Lithium only	10	59	7	41
Neuroleptics only	28	85	5	15
Lithium plus neuroleptics	9	69	4	31

[a] $\chi^2 = 5$ (n.s.).

neuroleptic available. This is suggested by his description of the first and fourth patients, who showed no abnormalities when lithium was combined with other neuroleptics but developed a transient encephalopathy when lithium was combined with thioridazine. The case of the first patient described by Spring was particularly unfortunate; she had done well on combined lithium and haloperidol for a year and was changed to the more toxic lithium/thioridazine combination because of apprehension engendered by the report of Cohen and Cohen (4).

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Postcardiotomy Delirium and Cardiac Output

BY STANLEY S. HELLER, M.D., DONALD S. KORNFELD, M.D., KENNETH A. FRANK, PH.D., AND PAUL F. HOAR, M.D.

Thirteen years following its initial description, delirium after open-heart surgery continues to occur. The phenomenon consists of a lucid interval followed by illusions and/or disorientation and at times progresses to hallucinations and/or delusions. A number of factors have been found to correlate with this delirium, and a multicausal etiology has been postulated. The existence of the lucid interval led to the hypothesis that the delirium was a reaction to the impact of the open-heart surgery recovery room (OHRR) experience in patients made vulnerable by the physical effects of cardiac disease and surgery (1–3).

An association between postcardiotomy delirium and postoperative cardiac index (cardiac output/surface area) has also been shown. In 1966, Blachly and Kloster (4) studied 37 cardiotomy patients who had undergone valve replacement. Cardiac output was measured pre- and postoperatively by a radioisotope dilution technique with ^{131}I-labeled albumin. Charts were evaluated blindly as to the presence of either organic brain syndrome or hallucinosis. The authors found that the great majority (81%) of patients in their series had some evidence of organic brain syndrome. It was marked in 22%, moderate in 32%, and mild in 27%. A marked brain syndrome was generally associated with cardiac index below 2.5 liters per minute per meter squared during the early postoperative period. Conversely, higher outputs were associated with less cerebral dysfunction. Forty-six percent of the patients experienced hallucinosis, some without evidence of brain syndrome. The hallucinosis tended to occur later and was suggestively related to periods of rising cardiac output.

The improvements in cardiac surgery and cardiopulmonary bypass techniques and the development of a thermodilution method (5, 6) that more accurately measures actual cardiac output called for a new study of the relationship between cardiac output and delirium.

Method

We studied 48 patients who had a single valve replacement at Columbia-Presbyterian Medical Center between July and November 1977. Patients were equally divided between aortic and mitral valve cases receiving glutaraldehyde-treated porcine heterografts. A triple-lumen flow-directed thermodilution catheter was introduced and the average cardiac output was determined via the thermodilution principle by using a

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TABLE 1
Relationship Between Cardiac Index and Delirium

Patient Group	N	Preoperative	Postoperative					Change (%)	p
			Close Chest	Recovery Room	Day 1	Day 2	Mean		
Aortic									
Clear	13	2.91	2.38	2.77	3.06	3.17	2.85	− 2.1	<.05
Delirious	11	3.01	2.37	2.15	2.46	2.44	2.36	−21.6	
Mitral									
Clear	17	2.33	2.47	2.55	2.63	2.66	2.58	+10.7	<.07
Delirious	7	2.22	2.33	2.05	2.03	2.06	2.12	− 4.5	
Combined									
Clear	30	2.58	2.43	2.65	2.82	2.88	2.70	+ 4.7	<.005
Delirious	18	2.70	2.35	2.11	2.29	2.29	2.26	−16.3	

cardiac output computer. During cardiopulmonary bypass, the adult Bentley oxygenator and cardiotomy reservoir were used. A nonhemic prime and hypothermia to 28° were employed. The cardiac index was sequentially measured preoperatively, at the time the chest was closed, on admission to the recovery room, and on the first two postoperative days. A mean postoperative cardiac index score was calculated. Informed consent was obtained from the patients after the procedure had been fully explained.

Delirium was diagnosed by blind chart review (3). Patients were judged to have a delirium if they experienced a lucid interval and then doctors' or nurses' notes in the postoperative week described either illusions, inability to distinguish dreams from reality, disorientation, hallucinations, or delusions. As a result of our earlier work (1–3), inquiry into these issues is done routinely in the OHRR. Student's paired and unpaired t tests were used to determine statistical significance.

Sample. The aortic valve replacement group (18 men, 6 women) ranged in age from 28 to 75 years, with a mean age of 60.8 years. Aortic stenosis was the main problem in 21 patients, insufficiency in 3. Four patients had concomitant coronary bypass procedures. Mitral patients (17 women, 7 men) were younger, with a mean age of 54.2 years and a range of 30–76 years. Mitral stenosis was the main problem in 14 patients, insufficiency in 10.

Results

Delirium was seen in 38% of our total sample. Eleven of the aortic patients (46%) developed delirium. Mean ages were 56.6 years for clear aortic patients and 65.8 for delirious patients. Bypass times were comparable (115.1 minutes for clear patients and 118.3 for delirious patients). Differences in cardiac index between the clear and delirious patients are shown in table 1. There was an overall decrease in the cardiac index for the whole aortic group, but the delirious group had a significantly greater decrease than the clear patients ($p<.02$). The delirious patients also showed a significant decline in cardiac index compared to their preoperative values ($p<.003$), while the clear patients showed only a slight change.

Among the mitral patients, the incidence of delirium was less (29%). Mean ages were 53.5 years for clear patients and 55.9 years for delirious patients. Unlike the aortic patients, cardiopulmonary bypass times differed between clear and delirious mitral patients (94.1 versus 121.7 minutes). There was a significant increase in cardiac index postoperatively for the clear mitral patients ($p<.05$), while delirious mitral patients had a statistically suggestive ($p<.07$) decrease. Mitral patients with delirium were the only group whose cardiac indices did not increase in the first 2 postoperative days. When the aortic and mitral valve groups were combined and the change in cardiac index was studied for the entire sample, a significant difference was found: clear patients' cardiac indices increased, whereas those of delirious patients decreased ($p<.005$).

Discussion

An association between postcardiotomy delirium and a low postoperative cardiac index has been confirmed in this preliminary study. This relationship is probably due to an associated impairment of cerebral perfusion. Our patients had a lower incidence of cerebral dysfunction than that reported by Blachly and Kloster (38% versus 81%), which is consistent with other studies showing a decrease in incidence since the earliest reports (2, 7).

Aortic valve patients with a clear sensorium showed a relatively unchanged cardiac index postoperatively, while delirious aortic patients showed a decrease of almost one-fifth. Clear mitral valve patients had increased cardiac output, while delirious mitral patients showed a slight decrease.

Although mitral valve patients had lower cardiac indices (8), the incidence of delirium was higher for aortic valve patients. It may be that aortic valve patients, who are accustomed to better cardiac function, were less able to tolerate low postoperative cardiac outputs. Mitral patients, on the other hand, may be adapted to low cardiac output associated with cardiac failure. Aortic valve patients were also older than mitral valve patients, and age could have also contributed to the higher delirium incidence.

Blachly and Kloster (4) noted that a rapidly rising output was associated with hallucinosis, although they did not report statistical confirmation. We were unable

to study cardiac output beyond the second postoperative day, so we can make only limited comments on this issue. However, all patients except the delirious mitral group had increases in cardiac output during the first 2 postoperative days. Additional measurements over the postoperative week would be necessary to confirm Blachly and Kloster's observation. It is also possible that the later occurrence of hallucinosis in their series was associated with patients' continued stay in the OHRR, rather than the continued rise in cardiac index.

It has been postulated that in the stressful OHRR environment the patient with an impaired brain is most delirium-prone (1–3). Age, time on the cardiopulmonary bypass pump, intraoperative hypotension, pre- and postoperative severity of illness, sleep deprivation, sensory monotony, and the personality traits of dominance, confidence, and composure have been associated with delirium (1–3, 7, 9, 10). Cardiac index is therefore another associated postoperative physical variable.

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Clinical Utility of Tricyclic Antidepressant Blood Levels: A Case Report

BY PAUL S. APPELBAUM, M.D., RUSSELL G. VASILE, M.D., PAUL J. ORSULAK, PH.D., AND JOSEPH J. SCHILDKRAUT, M.D.

The recent availability of procedures for measuring plasma levels of tricyclic antidepressants promises to increase the precision of pharmacologic treatment of depressions (1). An unusual example is provided in the following case report.

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Case Report

Ms. A, a 68-year-old woman with a history of recurrent unipolar depressions since her teens, has had 13 hospitalizations at several institutions during the last 12 years. During that period she received 4 separate courses of ECT, totaling 100 treatments, with modest response. Phenothiazines in large doses had limited impact on her illness. Lithium therapy was attempted twice but had to be withdrawn because of ataxia and tremulousness.

Tricyclic antidepressant therapy was first initiated in 1969, and Ms. A exhibited some clinical response to a combination of amitriptyline (75 mg/day) and perphenazine (6 mg/day). She showed complete recovery when the amitriptyline was increased to 200 mg/day. However, from 1970 to 1975 she had many recurrent episodes of depression, some of which were treated with amitriptyline in doses of 75–125 mg/day. The maximum clinical benefit during this period occurred when the patient received perphenazine (12 mg/day) in addition to amitriptyline (125 mg/day). From 1972 to 1975, tricyclic antidepressants were discontinued and Ms. A received phenothiazines and ECT.

In 1975 Ms. A was admitted to the Massachusetts Mental Health Center for the first time, and treatment with tricyclic

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antidepressants was reinstated. She responded to a very low dose of amitriptyline (50 mg/day) without concomitant use of phenothiazines. In June 1976, the patient stopped taking the amitriptyline and experienced a recurrence of depression. Upon rehospitalization, she was placed on amitriptyline (100 mg/day) without phenothiazines. She began to improve but soon became disoriented, with severe memory deficit, incontinence, somnolence, and, ultimately, coma. Four days after amitriptyline had been discontinued she had a pulse of 160, respiratory rate of 22/minute, temperature of 101° F, and no focal neurologic signs. An extensive neurologic work-up, including lumbar puncture, brain scan, and EEG, revealed no cause for her symptoms. Physostigmine was not administered. Eight days after the discontinuation of amitriptyline, the syndrome had resolved. Her depression persisted despite treatment with acetophenazine (60 mg/day) but responded to the addition of imipramine (50 mg/day).

In September 1977 Ms. A again stopped taking her medications, became depressed, and was readmitted to the Massachusetts Mental Health Center. She improved on imipramine (75 mg/day) and acetophenazine (40 mg/day), but when the imipramine dose was increased to 100 mg/day she experienced intermittent relapses that responded to dosage decreases.

She had been successfully maintained after discharge on imipramine (75 mg/day) until a sudden decompensation led to her hospitalization on Feb. 21, 1978. As in previous depressive episodes, she was agitated and diaphoretic, she responded to questions with "no" or "I don't want to" and she walked with what appeared to be an antalgic gait, which in the past had resolved as the depression cleared. She would not cooperate with formal mental status testing. Physical examination, liver function tests, and the remainder of her routine clinical chemistry tests were all within normal limits. Her weight was approximately 49 kg.

At the time of this admission the dose of imipramine was increased to 100 mg/day and acetophenazine, 20 mg/day, was added. The patient's failure to respond to this regimen after 2 weeks prompted her physician on March 7 to obtain plasma levels of tricyclic antidepressants, which were determined by gas-liquid chromatography using a nitrogen detector (2). He expected to find low levels and was surprised to discover that her total plasma tricyclic antidepressant level was 805 ng/ml (imipramine=193, desipramine=612), which is far above the usual therapeutic range of 180–250 ng/ml (3, 4). Imipramine was stopped on March 8, but acetophenazine (20 mg/day) was continued. A repeat sample drawn on March 9 revealed a total plasma tricyclic level of 696 ng/ml (imipramine=174, desipramine=522). It is noteworthy that physical examination at this point failed to reveal evidence of atropinic toxicity: pulse was 120, skin was diaphoretic, pupils were reactive to light and were not dilated, and there was no facial flushing, dry mouth, or difficulty with urination. On March 12, 3 days after imipramine was discontinued, Ms. A became less agitated, cooperated with nursing staff for the first time, and began to converse socially in an appropriate fashion and to play the piano. Mental status examination revealed a clear sensorium, marked improvement in depressive mood, and no evidence of psychosis. The next day, treatment with imipramine was resumed at a dose of 25 mg/day. On March 20, the patient was stable and recompensated, and her total plasma tricyclic level was 252 ng/ml (imipramine=51, desipramine=201). At this point Ms. A was discharged to a nursing home.

Ms. A was readmitted to the hospital on March 31 because her depressive syndrome had reemerged. The total plasma tricyclic level had fallen to 143 ng/ml (imipramine=23, desipramine=120), which is below the therapeutic range. Poor medication compliance may have caused this decrease. On April 1, the dose of imipramine was increased to 25 mg and 50 mg on alternate days. On April 20, she remained unimproved and acetophenazine was increased from 20 mg/day to 40 mg/day. One week later, the patient recompensated, becoming pleasant and talkative. On May 2, the plasma tricyclic level was 307 ng/ml (imipramine=85, desipramine=222). Ms. A was discharged on May 12 and 3 months later remained in remission while continuing to receive acetophenazine, 40 mg/day, and imipramine, 25 mg/day alternating with 50 mg/day.

Discussion

During the patient's most recent admission, accepted clinical practice would have called for an increase in the dosage of imipramine in the face of a failure to respond to a modest dose (100 mg/day). However, when plasma tricyclic levels were obtained on March 7, it became clear that they were approaching the toxic range and that a decrease in dosage was indicated. In the absence of the determination of plasma tricyclic antidepressant levels, neither clinical psychiatric evaluation nor physical examination would have led to the proper psychopharmacologic response.

The exceptionally high plasma levels that were found in spite of the relatively low daily doses shed new light on the patient's toxic episode in 1976 and suggest the possibility of a similar etiology then. Of note is an apparent decrease in the patient's tolerance for tricyclic antidepressants as she aged, a finding that has been reported elsewhere and that should emphasize the need for caution in prescribing for geriatric patients (5). An additional factor in the attainment of toxic levels in this case may have been the concomitant administration of acetophenazine. Phenothiazines have been reported to inhibit the metabolism of tricyclic antidepressants, especially of demethylated derivatives such as desipramine, thereby elevating effective blood levels despite stable oral doses (6, 7). In this regard it is of interest that every determination in the present case showed much higher plasma levels of desipramine than imipramine. Finally, this patient's failure to respond at high plasma levels and subsequent improvement at lower doses raises the possibility that, at least for some patients, there may be a therapeutic window for imipramine or desipramine similar to that previously reported for nortriptyline (1).

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Rapid-Cycling Affective Disorder During Imipramine Treatment: A Case Report

BY SAMUEL G. SIRIS, M.D., HARVEY R. CHERTOFF, M.D., AND JAMES M. PEREL, PH.D.

Rapid-cycling mood disorders have been recognized as rare but dramatic and potentially informative manifestations of affective disease. A review of the world literature in 1965 by Bunney and Hartmann (1) revealed clinical descriptions of 10 48-hour cyclers, and more recently several other cases of rapidly cycling bipolar disease have been studied biologically (2-4). None of these reports, however, has linked a rapidly cycling course with the use of an antidepressant agent. The present communication details a rapidly fluctuating affective disorder which occurred after treatment with imipramine had been initiated in a woman with a postpartum depression.

Case Report

Ms. A, a 30-year-old married mother of two, had no notable psychiatric difficulties until age 27. At that time, several weeks after the birth of her first child, Ms. A experienced conflict because her son seemed to do better on formula than on breast milk. This was soon superseded by persistent feelings of depression, irritability, anxiety, worthlessness, and loss of sleep. After several weeks of suffering, she sought psychiatric help and was treated first with amitriptyline, 75 mg/day, and then with desipramine, 75 mg/day. Although her sleep improved, her dysphoric mood, lack of interest, and diminished ability to function persisted for 4 months, at which time they gradually abated and she felt like her "normal self" again. Medication was then discontinued. She had no history of episodes of acceleration, euphoria, or irritability.

She remained well until the birth of her daughter 3 years later. When the child was 3 months old, Ms. A again experienced a similar conflict over breast feeding, coupled with apprehension about her parents retiring and moving to another state. She became depressed once again, with prominent tearfulness, doubting, preoccupations about her lack of capacity, feelings of guilt, decreased energy, decreased ability to concentrate, poor appetite, and middle and late insomnia. Her symptoms were worse in the morning than in the evening. After 3 weeks of steady depression, she consulted a psychiatrist once again and started taking imipramine. After 12 days of imipramine treatment at a dosage of 100/mg day, she had a 48-hour period of cheerfulness and increased energy, which made her think temporarily she was "back to her old self." This episode ended abruptly, however, and she returned to her depressed state for the next 2 weeks. On the 28th day, this time on a dosage of 175 mg/day of imipramine, she again came out of her depressed state for approximately 48 hours. During that interval she had a night in which she slept only 2 hours but woke feeling fully rested. She described feeling "better than my usual self" and decided to join a bowling league but did nothing else unusual. This signaled the start of a 2-month period during which she emerged from and then relapsed into a depressed state seven separate times. During this period her imipramine dose was raised gradually to 300 mg/day (4.4 mg/kg, with a steady-state plasma concentration of 180 ng/ml) without alteration of her cyclic mood pattern. Her nondepressed intervals ranged from 2 to 6 days and her depressed episodes from 3 to 7 days. The transitions between these states seemed unrelated to external events and occurred abruptly over a few hours, during which time Ms. A felt herself either "slipping" or "snapping out of it." When she was not depressed, Ms. A was at times euthymic and at other times mildly hypomanic, with more than her normal level of energy and enthusiasm, and she occasionally required as few as 2 hours of sleep. Her judgment remained sound, however, and she was neither giddy nor irritable. She was unmistakably different when depressed: hopeless, devoid of pleasure, self-critical, nonfunctional, and thoroughly miserable.

On the 85th day of imipramine treatment, when Ms. A was "due" to come out of one of her depressed intervals, oral lithium carbonate was begun. The dose was rapidly stabilized to give a serum level of 0.7-0.8 mEq/liter. On day 86 of imipramine treatment, Ms. A switched back to her normal euthymic state and remained there. Imipramine was continued at 300 mg/day until day 122, when the dosage began to be tapered off. Imipramine was completely discontinued on day 186. Lithium treatment was continued for an additional 12 months, then discontinued, and the patient has remained well.

Ms. A's family history is remarkable in that her mother suffered two clinical depressions. One, which occurred during the first trimester of an unplanned pregnancy, resolved spontaneously. The second depression, at age 65, was treated with imipramine for 5 months before responding

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promptly to ECT. Retrospective analysis of the mother's case history revealed two abrupt and otherwise unexplained 48-hour episodes of feeling active, confident, and enthusiastic while on imipramine. These episodes disappeared as suddenly as they had started.

Discussion

Ms. A had a postpartum depression that was phenotypically unremarkable. She met the Research Diagnostic Criteria (5) for a simple primary endogenous recurrent major unipolar depression. For 3 weeks her course had been well established. However, after starting imipramine, Ms. A underwent 15 major mood changes between depression and either euthymia or hypomania over a 10-week period. To our knowledge, such a cyclic response to imipramine in an otherwise unipolar depressed patient has not been described previously, although clear switches into persistent mania in response to imipramine have been well-documented (6, 7). It is also known that more manic attacks occur in bipolar patients being treated prophylactically with imipramine than in those being treated with lithium or placebo (6), and preliminary evidence suggests that the cycle time of some bipolar patients may be shortened by tricyclic antidepressants (8). Imipramine, of course, interacts with biogenic amine mechanisms which have been implicated in the manic-depressive "switch" process (7, 9), so that a causal relationship linking imipramine administration and the clinical syndrome exhibited by Ms. A is not implausible.

Dunner and associates (10) examined the case records of 390 bipolar patients and found that 13% were "rapid-cyclers" according to their criterion of 4 or more affective episodes per year. As a group these patients were observed to benefit less from lithium than other bipolar patients. However, this represented a group of patients who were known to be bipolar before treatment and were not being treated with a tricyclic antidepressant. The subset of affectively ill patients whose only manifestations of hypomanic or manic characteristics occur while they are under treatment with tricyclic antidepressants has not yet been separated out for study. Ms. A's favorable response to lithium, as a single case, is of course inconclusive. Nevertheless, our experience would encourage us to identify similar cases and attempt a lithium trial.

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Effects of Training Schedule and Biofeedback on Speech Dysfluency

BY THEODORE WEISS, M.D., LINDA F. CARSON, M.D., AND JOHN PAUL BRADY, M.D.

Masseter muscle electromyographic (EMG) biofeedback reportedly improves speech fluency in stutterers (1). We examined the efficacy of this method and the effects of training session frequency in an older patient with speech dysfluency.

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Case Report

The patient was a 64-year-old woman with mild congenita choreoathetoid encephalopathy (cerebral palsy). The cerebral palsy had not impaired her functioning, but at age 62 her speech had worsened, and her jaws started clamping shu when she spoke. She had to compress her cheeks inwarc with both hands to facilitate speech flow. Inpatient evaluation revealed no clear reason for her deterioration, and several medications (haloperidol, perphenazine, amantadine

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TABLE 1
Patient's Speech Performance

Biofeedback Training Schedule[a]	Number of Recordings	Words in 5 Minutes		Dysfluencies in 5 Minutes		Dysfluencies/ 100 Words	
		Mean	SD	Mean	SD	Mean	SD
Baseline	1	332		88		26.5	
Daily	2	439.0	55.2	33.0	21.2	7.9	5.8
Weekly	10	361.6	50.5	32.6	14.5	8.9	3.6
Biweekly	8	341.8	61.6	48.6	22.5	15.0	7.7
Weekly	10	375.5	38.1	35.2	11.1	9.6	3.5
Weekly (no biofeedback)	10	395.8	45.0	34.3	7.9	8.8	2.5

[a]There were 10 sessions in each training phase.

diazepam, and doxepin) were administered without benefit. She was taking doxepin, 75 mg/day, for mild depression at the beginning of the study. Because her speech problem involved masseter muscle contraction, we trained her with EMG biofeedback from this area. After 20 informal training sessions, at varying intervals, we initiated the present study to examine our impression that improved speech occurred with more frequent biofeedback.

Method

EMG activity (100–1,000 Hz) from the right or left (in alternate sessions) masseter muscle was recorded with surface electrodes (silver/silver chloride) using a commercially available biofeedback device. The machine's threshold criterion was set empirically so that the patient turned on a green or yellow light when she relaxed her masseter muscle and a red light when she tensed it. She practiced this several times during the 1-hour training session. The rest of the time she spoke with a technician and was drilled in pronouncing problematic words or syllables while attempting to keep the yellow or green lights lit.

Performance was assessed by a 10-minute recording during which the same experimenter had the patient talk about a subject that interested her. Five minutes of her speech was analyzed for numbers of words and dysfluencies, defined as clearly discernible instances of words or syllables the patient had difficulty emitting.

The training sequence was as follows: 1) 10 twice-daily biofeedback sessions over 5 days, 2) 10 weekly biofeedback sessions, 3) 10 biweekly biofeedback sessions, 4) 10 weekly biofeedback sessions, and 5) 10 weekly sessions without biofeedback. In no-biofeedback sessions, after the initial recording, the patient and technician talked for 50 minutes, with no biofeedback or drill on problematic words and syllables. The tape recording was done at the beginning of every session, except during the daily biofeedback phase, when it was done only before sessions 1 (baseline, see table 1), 5, and 9. Between biofeedback sessions the patient was instructed to practice relaxing and clenching her jaw several times daily at home and to imagine the green or yellow lights when she had difficulty speaking. For the no-biofeedback sessions she was given no instructions about home practice. She gave informed consent to participate in the study.

Results

During daily biofeedback training, the patient's speech fluency improved markedly. Dysfluencies/100 words fell from 26.5 before training to 3.8 at the beginning of session 9. Speech data generally were best during this period. In the following weekly biofeedback phase speech remained good, but when biofeedback was decreased to every 2 weeks, her speech worsened. Her performance during biweekly training was worse than any other period. Of 8 sessions, 4 (numbers 2, 3, 4, and 9) showed dysfluency rates over 18.0, a high level not seen in any other sessions (except the prestudy baseline). During this biweekly biofeedback phase doxepin was discontinued after session 4 because the patient no longer seemed depressed. Speech fluency worsened dramatically in the next 2 sessions (5 and 6), with data in almost every category by far the worst seen (e.g., dysfluency rates of 53.3 and 43.9). We felt these sessions reflected aftereffects of the drug's discontinuation and dropped them from the data analysis. (These data would have strengthened our argument regarding the effect of session frequency.)

In the subsequent weekly biofeedback phase speech again improved. To assess the importance of biofeedback, we then gave 10 weekly sessions without biofeedback as described above. Speech performance was quite similar to that in the weekly biofeedback training phases.

Discussion

Weekly training sessions were more beneficial than less frequent ones for this dysfluent patient. However, we found no evidence that EMG biofeedback from masseter muscles was necessary for this improvement. Masseter biofeedback improved fluency in stutterers when the feedback-on and feedback-off conditions followed one another within a few minutes (1). Our data on a related but different issue—the persistence of benefit from masseter biofeedback during a 1-week period between training sessions—indicate that weekly practice speaking with a technician was as therapeutic as weekly biofeedback.

We cannot exclude the possibility that improved speech during the no-biofeedback phase reflected persistence of an over-learned response from many prior

biofeedback sessions. However, the patient's worsening in speech during biweekly biofeedback followed 40 prior biofeedback sessions, whereas persistently good performance in the weekly no-biofeedback phase followed 60 prior biofeedback sessions. It seems unlikely that the 20 biofeedback sessions between the first weekly biofeedback phase and the final weekly no-biofeedback phase—during a number of which the patient was quite dysfluent—were critical in her acquiring persistently improved speech.

Elder and Eustis (2) found that daily blood pressure biofeedback was more effective in lowering hypertensives' pressures than a similar number of biofeedback training sessions spread over several months. However, our data in the no-biofeedback phase suggest that an appropriate control procedure in their study—relaxation training, rapping, sitting quietly—carried out with the same frequency as blood pressure biofeedback might have been as effective in lowering patients' pressures.

The issue of nonspecific or placebo factors in biofeedback training (3) is relevant. An opportunity to speak with a familiar person was beneficial to our dysfluent patient's speech. Biofeedback was merely the mechanism by which this interaction occurred rather than an important therapeutic ingredient.

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Unconscious Communication Between Father and Son

BY JESSE O. CAVENAR, JR., M.D., AND NANCY T. BUTTS, M.D.

The influence of parents and their neuroses on the developing child has long been recognized. The importance of the neurotic conflicts of the parents in the psychoanalysis or psychotherapy of the child has been appreciated since at least 1909, when Freud capitalized on his patient's relationship with his father in "The Analysis of a Phobia in a Five-year-old Boy" (1). Freud conducted the entire treatment of the boy through the father.

The importance of unconscious conflicts in the parents as a cause of conflicts in the child gained more acceptance as more experience was gathered in child psychoanalysis. By 1935, Burlingham (2) stated that "the power of unconscious forces is especially marked in its interplay between parent and child. It is so subtle and uncanny that it seems at times to approach the supernatural."

Johnson and Szurek (3) suggested that unconscious communication between the child and the parents, particularly the mother, was instrumental in the development of antisocial personality disorders. It was believed that the parent or parents unconsciously derived pleasure from the child's antisocial acts and subtly encouraged them. At the same time, the parents could discharge their unconscious hostility, sadism, and destructiveness toward the child.

Child psychoanalysts observe the intensity of unconscious parental forces when they undertake simultaneous analysis of both parent and child. Such simultaneous analysis is occasionally necessary when a parent and child are locked in a severe neurotic pattern. There are several case reports in the literature that describe the simultaneous psychoanalysis of mother and child (4, 5); we are aware of only one article (6) on the simultaneous psychoanalysis of a father and son. None of these case reports refers to specific dreams of either parent or child during the treatment.

The purpose of this paper is 1) to report a case in which unconscious communication between father and son resulted in both having dreams that used the same symbols and 2) to demonstrate the degree to which the father's conflicts influenced his son.

Case Report

The father, Mr. A, is a 38-year-old professional man who had sought psychoanalysis 10 years earlier because of anxiety, self-defeating behavior, and a fear of success. An obsessive-compulsive personality structure had led to a psychoanalysis of 6 years' duration. There was excellent resolution of his anxiety and other symptoms.

The psychoanalysis had terminated 4 years previously.

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Mr. A had continued self-analysis primarily through interpreting his dreams. He had been very successful in his chosen field and had had many advancements and promotions since the analysis ended.

As a result of several significant object losses, Mr. A regressed and became clinically depressed, with weight loss, sadness, and early morning awakening. He consulted his previous analyst for assistance in understanding the depression. Anxiety dreams indicating superego tension were frequent. He described one typical dream as follows: "A snake was charging at me. I had a sling-type weed cutter in my hands, and tried to cut the snake in half. I couldn't get the weed cutter to land solidly, and kept missing the snake. The snake would crawl past, then charge from another direction." He awakened from the dream and discussed it with his wife early the following morning.

That evening, the son, who was 7 years and 10 months old, came to Mr. A to discuss his own dream of the previous night. He reported, "A snake was chasing me, and I couldn't get away from it. Every time I thought I was safe, the snake would chase me more." The son had no knowledge that his father had had a similar dream the same night.

Four nights later, Mr. A had the following dream: "A tornado was approaching; I could see the funnel. I was very anxious and hurried about telling various people that a tornado was imminent. The people wouldn't heed my warning, and continued their routine activity. I shouted, 'Believe me, this is serious.' In desperation, I grabbed a child under each arm and ran." The father awoke with anxiety and again discussed his dream with his wife early that morning.

That evening, his son came to talk to Mr. A about his own dream of the previous night, which he described as follows: "A tornado was coming after me. I was scared at first; I got sucked up in the funnel, like Dorothy in *The Wizard of Oz*. I was thrown around by the tornado, but it was fun. After a while, the tornado let me back down to the ground; I wasn't hurt." Again, the son had no knowledge that his father had had a dream using the same symbols on the same night. The son had never seen a tornado except on television and had only seen snakes in a zoo. He had no reason based in his own experience to associate either with anxiety.

Mr. A was seen for 8 hours in face-to-face psychotherapy and rapidly understood the dynamics of his depression. The depression cleared, and the anxiety dreams stopped. The son's dreams apparently also stopped; he has not mentioned a dream to his parents in many months. Obviously the atmosphere in the home is such that the son feels free to discuss dreams which are of concern to him, so we assume that he has not had further troublesome dreams.

It is important to note that Mr. A's recollection of his dreams was not retrospective falsification; he had discussed his dreams with his wife before his son reported his own dreams. We are also certain that Ms. A did not tell him of the father's dreams. She was positive that she had not told her son about them. Ms. A had also completed a successful psychoanalysis and found the similarity in dreams fascinating.

Comment

In the multiple case reports of simultaneous parent-child psychoanalysis, there are to our knowledge none that include reported dreams in which parent and child used the same dream symbols. The choice of dream symbols is poorly understood, but if parent and child employ the same symbols, simultaneous psychoanalysis should demonstrate this tendency.

Altman (7, p. 18) noted, "Each dreamer employs a preferred set of symbols chosen from the vast number available to him and uses them with considerable regularity and consistency so that they become a sort of mental fingerprint." He further stated, "A recurrent use of the same symbol must have a special significance but, to date, in not a single instance have I been able to trace any to its source in the patient's personal experience. Why does anybody use a particular symbol with such consistency that one could call it a mannerism?"

Certainly we cannot answer this question on the basis of one case report, but our work tends to suggest that persons within the same biological lines may employ the same symbols. This should be further investigated in parent-child simultaneous psychoanalysis or psychotherapy or in the therapy of twins or siblings when dreams are a focus of the treatment.

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Central Nervous System Toxicity of Cimetidine: A Case of Depression

BY JAMES W. JEFFERSON, M.D.

In a relatively brief period of time, the competitive histamine H_2 receptor antagonist cimetidine has become a drug widely used to decrease gastric acid secretion (1, 2). The Food and Drug Administration has approved its use for the short-term treatment of duodenal ulcer and the treatment of pathological hypersecretory syndromes (Zollinger-Ellison syndrome, systemic mastocytosis, multiple endocrine adenomas). Other uses which require further experimental confirmation include benign gastric ulcer, gastroesophageal reflux disease, acute gastric hemorrhage, pancreatic insufficiency, and the maintenance treatment of duodenal ulcer.

The drug is generally well tolerated, but there have been a growing number of reports of neuropsychiatric toxicity associated with its use (3–10). Included in these reports are manifestations such as lethargy, coma, restlessness, agitation, disorientation, confusion, delirium, unsteadiness, slurred speech, visual impairment, and incontinence. The following case adds depression to the list of neuropsychiatric complications.

Case Report

An 81-year-old man was referred from a retirement home to the medical service of our hospital for evaluation and treatment of depression and possible recurrent peptic ulcer. Over a period of several months he had become "disgusted with life," complaining of difficulty concentrating, especially when reading and playing cards, loss of interest, a decreased ability to enjoy himself, lack of energy, difficulty falling asleep, bizarre nightmares, anorexia, and weight loss. He had no previous history of psychiatric difficulties.

Two years earlier he had developed gastric outlet obstruction secondary to peptic ulcer disease, which was treated surgically. For 5–6 weeks before the current hospitalization he complained of epigastric pains and vomiting after meals, which raised the possibility of recurrent ulceration and obstruction. He had been receiving cimetidine, 300 mg. q.i.d., for a number of months.

Psychiatric evaluation was obtained shortly after admission, and the consultant felt that the cimetidine could be responsible for the patient's symptoms and recommended its discontinuation. At follow-up 8 days later, however, the symptoms were unchanged and he was continuing to receive the drug. When it was finally stopped, the patient improved rapidly and in 2 days was more optimistic, had lost his fatalistic outlook, was sleeping and eating better, and was reading magazines and actively looking for a cribbage partner. His gastrointestinal symptoms completely resolved and at discharge he was cheerful and looked forward to resuming his prior activities.

Discussion

It appears that cimetidine can cause CNS side effects, although they are uncommon. Usually these reactions take the form of a reversible organic brain syndrome, although, as the present case illustrates, a drug-induced depressive syndrome is also possible.

Neuropsychiatric complications appear more likely to occur in the presence of 1) advanced age, 2) impaired renal function (up to 70% of an oral dose of cimetidine is excreted unchanged by the kidneys), 3) associated serious medical problems, and 4) a dosage higher than that recommended. The absence of predisposing factors does not, however, exclude the possibility of cimetidine-induced CNS effects.

With the increasing use of cimetidine in a variety of clinical settings, there may well be a steady increase in the number of neuropsychiatric complications. Prompt recognition of the apparent causative role of the drug in such situations is essential because the syndrome is readily reversible upon drug discontinuation, and if it is unrecognized, the patient will be subjected to needless suffering, increased risk of morbidity, unnecessary diagnostic procedures, and inappropriate and often dangerous treatment.

The association of CNS toxicity with the use of a histamine H_2 receptor antagonist should also stimulate further interest in the role of histamine in the CNS.

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Autocastration in Transsexualism

BY MICHAEL A. HABERMAN, M.D., AND RICHARD P. MICHAEL, M.D., PH.D.

Forty-four cases of autocastration (removal of the testes rather than mutilation or amputation of the penis) have been reported in the English language literature since 1900 (1-3). In 1951, Hemphill (4) wrote about the personality traits of patients who castrate themselves, including an abnormal attitude toward the genitals, a tendency to regard the genitals as exercising influence over the whole organism, an abnormal integration of the body image that permits mutilation, a sense of intactness and relief thereafter, and a strong, conscious motivation. Hemphill contrasted genital self-mutilation with other self-inflicted injuries by pointing out that this act gives both satisfaction and relief and is not regretted by the individual.

Before the 1954 report by Esman (5), all published cases were of psychotic individuals with organized delusions. Esman described a 44-year-old nonpsychotic schizoid man who was secretly a transvestite and who impulsively castrated himself while intoxicated. The patient's complacent attitude toward "so catastrophic an occurrence" suggested that the act might have in some way gratified an unconscious wish and solved an unconscious conflict. The patient had subsequent admissions for depression and alcoholism, but 5 months later he seemed improved and was not heard from further. Cleveland (6) reported three cases of autocastration in men who were not considered overtly psychotic and who also reacted with bland acceptance. In 1971, Lowy and Kolivakis (2) described the first recorded case in which a transsexual man castrated himself. This man dated his desire to be a girl from age 6 and had started nonfetishistic cross-dressing before puberty. He claimed his sexual organs had always disgusted him and he had desired sex change surgery since adolescence. He castrated himself at age 35: the procedure was "skillfully performed," and he flushed the testes down the toilet to ensure that they could not be reattached. He seemed happier after castration and "relentlessly and successfully continued to establish his identity as a woman." The authors described him as "probably schizophrenic" for reasons that are not entirely clear.

Transsexualism has now emerged as a more distinct diagnostic entity, and increasing numbers of transsexual patients have sought help from clinics. In general, societal attitudes have been fairly permissive, and it is becoming easier to fulfill many of the criteria that must be met before surgery can be contemplated. However, the cost of surgery ($4,000 to $5,000) constitutes a major obstacle. Further, few psychiatrists are prepared to recommend surgery unequivocally unless a series of important desiderata are satisfied, which may take several years. We will describe two cases of autocastration by male transsexuals who attended the gender identity clinic of the Emory University School of Medicine and the Georgia Mental Health Institute.

Case 1

Mr. A, a 25-year-old unmarried man, came to the clinic requesting hormones to prepare for sex-reassignment surgery, a course he had decided on at age 19. For the previous three years he had lived cross-dressed, used a feminine name, and obtained hormones intermittently from several physicians in the community. At age 2, he began to dress himself in some of his mother's clothes, and a maternal aunt is said to have cross-dressed him frequently. He played with dolls and became a "loner" when school began. Sexual activity was initiated in the preschool years by a male cousin 10 years his senior, and this relationship progressed to fellatio and anal intercourse at puberty. In all his sexual encounters he assumed the receptive position, and he denied any heterosexual activity, either real or fantasied. At age 10, he was referred by the school to a psychiatrist because he was shy and withdrawn. Therapy was brief, but he maintained episodic contact with the therapist over the next 10 years. At age 15, he started to wear makeup and earrings to school. At age 19, he cut his wrists while he was using amphetamines and was hospitalized for a short time. A diagnosis of paranoid schizophrenia was made then, largely because of his paranoid ideation about the inability of others to see him as a female. Treatment with chlorpromazine and psychotherapy for 1 year had no effect on his gender dysphoria.

On his admission to our clinic, physical examination and laboratory studies, including EEG, karyotyping, and plasma hormone levels, were all within normal limits. He felt very uncomfortable among homosexuals and thought he was not accepted by them. His main desire was to have dates with very masculine heterosexual men. He had no police record or evidence of recent drug abuse and denied having engaged in prostitution. There was no evidence of any psychotic process, unless one regards the fixed transsexual belief as a delusional manifestation of psychosis. He was capable of earning his living and passed very well as a female in the community. He was totally convinced that surgery would dramatically alter his life and explained that normal relationships were impossible for him because he was a woman with male genitalia. We began the feminization process slowly with controlled doses of estrogen and progesterone and monitored his plasma hormone levels by radioimmunoassay. He warned us he might castrate himself because he felt we were proceeding too slowly but said he could control the impulse since such an act might jeopardize subsequent genital

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surgery. Soon after New Year's Day he went to a lavatory in a local hospital, injected lidocaine into the skin of the scrotum, incised the median raphe with a razor blade, excised both testes, and flushed them down the toilet. He stuffed a towel between his legs to control the hemorrhage and walked to the emergency room for further treatment. He had read about genital anatomy and had, with some skill, performed a bilateral orchiectomy. He called our clinic shortly thereafter and said, "I told you I would do it and I have." At this time there was no evidence of psychosis and the sensorium was clear. He felt no remorse or guilt and had no thoughts of further self-injury or suicide. With quiet satisfaction he stated, "No one can call me a man now." Twenty-one months later we supported this patient's request for full sex-reassignment surgery and, despite her somewhat bizarre lifestyle and obvious personality problems, she has made a satisfactory adaptation during the 12-month follow-up since surgery.

Case 2

Mr. B, a 23-year-old unmarried man, came to the clinic requesting sex reassignment surgery. He reported that he had castrated himself 2 years previously and had felt very much better since that time. For the past 3 years he had been taking feminizing hormones and had lived completely cross-dressed as a female. He described himself as having been feminine, quiet, and a "sissy" since early childhood. He cross-dressed at home before puberty without protests from his parents and began cross-dressing in public after high school graduation. His first homosexual intercourse occurred at age 14, and all of his subsequent sexual encounters were with men. He invariably took the receptive position. He denied any heterosexual desires or fantasies, but the fantasy of actually becoming a woman did not develop until late adolescence. He claimed to have had many sexual relationships with heterosexual men that lasted from a few to several months. He had castrated himself, following the simultaneous loss of both his job and boyfriend, after he took methaqualone tablets and drank a quantity of alcohol. Like the previous patient, he had acquainted himself with genital anatomy, but he relied on alcohol for anesthesia. He lost a considerable amount of blood, alerted his roommate, and was taken to a hospital. Despite his intoxication, he had placed the incision carefully in the midline of the scrotum, and the scar was quite difficult to detect. Psychiatric evaluation at the time of the castration did not reveal any evidence of psychosis; the patient expressed no regrets and felt no subsequent depression or anxiety. Psychiatric examination at the time of admission again failed to reveal any evidence of psychosis, and the diagnosis was gender identity dysphoria and hysterical personality disorder. This patient was strikingly feminine and had no difficulty passing as a woman in the community. We have provided her with hormones for the last 3 years, and 12 months ago complete penectomy and vaginoplasty were done at the patient's expense. Despite some emotional ups and downs, her adaptation has steadily improved and she has become quite successful in her career.

Discussion

These two patients demonstrate many of the features discussed by Blacker and Wong (1): they showed strong feminine identifications, an absent or uninvolved father during the formative years, a positive relationship with a dominant mother, and physical repudiation of their male genitalia. However, unlike the two schizophrenic men described by MacLean and Robertson (7) who performed self-enucleation, neither of our patients was thought to be psychotic at the time of the autocastration or thereafter. In 1976, Money and DePriest (3) described the only three cases of genital self-mutilation seen at the Johns Hopkins Clinic since 1951. Two of these patients were overtly psychotic, but the third case involved a planned autocastration by a 51-year-old transvestite. The authors noted that the autocastration did not result in any psychiatric deterioration. We believe that this act is qualitatively different from the self-mutilations occasionally seen in disturbed schizophrenics. Except in regard to their genitals the patients were not delusional, they had studied the anatomy of the area, did not butcher themselves, and had long-term goals that both eventually attained. Such patients perceive their genitalia as a severe impediment to effective social and sexual functioning. The penis and testes appear to be excluded from the body image by the male transsexual, probably as a result of years of continuing conflict between early feminine introjects and the peer and community responses to the individual as a male.

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Am J Psychiatry 136:3, March 1979

LETTERS TO THE EDITOR

This section contains reactions to Journal *articles, statements of opinion, comments on Association activities, requests for information, etc. Letters should be submitted in duplicate to the Editor, who makes all decisions regarding publication. Letters must be typed double spaced throughout and should not contain more than 500 words and 5 pertinent references. Criticisms of published articles will automatically be sent to the author(s) for response. Letters will be edited for clarity and conformance with* Journal *style. We regret that we cannot inform writers of the disposition of letters or return those which are not printed.*

"Hypohypomania" and Tricyclic Antidepressant Choice

SIR: Ample evidence suggests that tricyclic antidepressants, when indicated for unipolar depression, cannot be chosen at random. Some patients respond to amitriptyline (a serotonergic drug) and others to imipramine (noradrenergic), with little or no overlap between the two groups (1). To date, these groups are discriminable only by urinary 3-methoxy-4-hydroxyphenylglycol (MHPG) excretion or by response to *d*-amphetamine. However, MHPG assay is highly technical and not widely available, and many clinicians hesitate to use the widely abused amphetamines with outpatients, even briefly. Clinical guidelines such as agitation versus retardation, despite their popularity, lack discriminant value in controlled investigations (2). To avoid fruitless drug trials and prolonged patient suffering, the practitioner needs a means of distinguishing the two types of unipolar depression—amitriptyline-responsive and imipramine-responsive—that requires no more than the clinical interview. We would like to suggest such a method.

We have found by close questioning of patients diagnosed as unipolar that many have experienced episodes of increased energy and decreased need for sleep (even without increased sense of well-being), irritability in the absence of depression, and an increase in behaviors that in more full-blown form would obviously be hypomanic or manic (e.g., impulsive traveling, money spending, or making long-distance phone calls). These patients, many of whom were previously treated unsuccessfully with adequate courses of amitriptyline, have responded well to imipramine. They tend to have had earlier illness onset (20s to mid-30s) than patients who lack such histories. In this, they resemble depressed patients with distinct manic episodes (3). In contrast, patients without a history of these "hypohypomanic" symptoms (many of whom had received adequate imipramine trials) have responded well to amitriptyline. This guideline has successfully predicted the proper antidepressant in approximately 90% of 50 consecutive outpatients we have seen recently.

A recent study (4) suggested that one group of unipolar depressives, chosen on the basis of nonresponse to tricyclics, more closely resembled patients with more clearly developed bipolar affective disorder, but the authors did not say which tricyclics were used. Others (5) have noted that cyclothymic disorders represent a less severe point on the spectrum that ends in bipolar affective disorder and have emphasized the importance of searching for the more subtle symptoms of affective illness. Although both studies clearly document, on phenomenologic grounds, the nonhomogeneity of unipolar depression first suggested by MHPG studies, neither provides guidance for selecting a particular tricyclic.

One might determine on logical grounds alone, from extant studies, the correct tricyclic antidepressant for unipolar patients. There are two types of unipolar depression (1, 2, 4, 5): one responds to imipramine and the other to amitriptyline (1). Decreased MHPG predicts positive imipramine response (1) and depressed bipolar patients have decreased MHPG (2). Therefore, those patients with unipolar depressions who resemble bipolar patients, slight as the resemblance may seem, should respond to imipramine, while depressed patients without obvious or subtle hypomanic histories should respond to amitriptyline. Our recent experience supports this logic and has allowed us to predict, based only on the interview, the proper tricyclic antidepressant.

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THOMAS E. STEELE, M.D.
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Mental Health Standards and Stereotype Traps

SIR: I have enjoyed reading the *Journal* for several years, but now I must register a criticism. I find it paradoxical and distressing that one author (Robert Butler, M.D., "Thoughts on Aging," July 1978 supplement) presented an articulate outcry against behavior that conforms to "society's norms or negative stereotypes," while another (Richard Green, M.D., "Sexual Identity of 37 Children Raised by Homosexual or Transsexual Parents," June 1978 issue) used "cultural

stereotypes" as criteria for implied psychological health. I am neither homosexual nor transsexual, but as a woman I shudder to think that my daughter would be considered "unfeminine," "neurotic," or "maladaptive" if her favorite toy were not a doll, her ambition were not to be a waitress, secretary, or nurse, or if she were interested in athletic endeavors (i.e., a "tomboy")—or that my son would be considered "unmasculine" if he enjoyed dolls and did not care for sports.

Certainly, differences of opinion on cultural matters are welcome and stimulating to your readers, but if psychiatry is attempting to attain scientific status it would be well advised to free itself of health criteria that are based on anachronistic cultural rules of behavior. In fact, many individuals throughout history have suffered loss of ego integrity as a result of being made to feel "abnormal" because they failed to fit cultural sex-role stereotypes. These stereotypic expectations have always been damaging among less sophisticated societal levels; to see them expressed in the *American Journal of Psychiatry* is very disappointing and—dare I say it—depressing.

FAITH FRANKEL
Hampton, N.J.

Trifluoperazine and Haloperidol: Sources of Ejaculatory Pain?

SIR: Impotence is a known side effect of neuroleptic and antidepressant drugs, but I know of no published reports of ejaculatory pain resulting from either of these classes of medications. Thus it may be helpful to record the case of a patient who experienced ejaculatory pain when taking trifluoperazine HCl and when taking haloperidol.

The patient was a physically healthy 30-year-old father of two. I first met him when he was admitted to a hospital for an acute psychotic episode. He had been admitted once before, 3 years earlier, for the same problem. These acute psychotic episodes, as well as several temporary psychotic episodes, had apparently been precipitated by his use of marijuana. The patient said he had been treated with lithium carbonate during the previous hospitalization (the record is not available). He reported symptoms of feeling lost, getting messages from television, and, in general, feeling not himself. His wife added that his appetite and sleep were drastically impaired. Haloperidol, 15 mg/day, was prescribed. One month after admission the patient had responded and was discharged, still on haloperidol. The following month, he complained of fatigue and slurred speech and stopped taking the drug. Over the next months, he used haloperidol intermittently (approximately every 2 months) when he felt his symptoms returning.

About a year after his discharge, the patient suffered a particularly threatening episode of temporary psychosis. Nevertheless, he refused to resume haloperidol but agreed to a daily 10-mg dose of trifluoperazine. One week after taking trifluoperazine, he complained of pain on ejaculation. He described it as a deep, sharp pain experienced when the muscles contracted during ejaculation. The pain occurred whether ejaculation was induced by intercourse or by masturbation. He stopped taking trifluoperazine and 3 days later reported that he no longer experienced the pain. At that point, the patient agreed to resume haloperidol instead of trifluoperazine. However, on resuming haloperidol, 5 mg/day, he complained of the same pain upon ejaculation. As a result, haloperidol was discontinued. The patient had had a urologic examination 2 months after he was discharged, and no abnormality was found. Molindone hydrochloride, 50 mg/day, was substituted for the haloperidol.

In the more than 2 months since the patient has been on molindone, he has reported no pain on ejaculation and no other side effects. This raises the question of whether the pain was due to haloperidol or to trifluoperazine.

STEVEN H. BERGER, M.D.
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Psychiatry and Culture

SIR: Had I understood the article "Overview: Foundations of Cultural Psychiatry" by Armando R. Favazza, M.D., M.P.H., and Mary Oman, M.A. (March 1978 issue) it would not have been necessary for me to write this letter.

The authors assume that we all agree on a definition of "psychiatry." However, only about one-tenth of their article is devoted to "mental health and illness," so they must have had difficulty defining psychiatry. There is also some vagueness in their definition of "culture." Psychiatrists in general seem to rely, either from insecurity or pretense, on references outside their discipline. This may create the feeling that we are the first ones to see something new, making us innovators or discoverers. Why not quote the definition provided by Sir Aubrey Lewis, which would preserve and further the development and continuity of views and thoughts in psychiatry? Sir Aubrey, discussing the "ill-defined" concept of "culture," said it was "the sum total of persisting values, ideas, and other symbolic systems that shape the patterns of behavior" (1, p. 235). The last words in this definition are the link with our discipline. Closer to the roots of the word "culture" is a statement by Williams regarding its "main meaning of cultivation or tending, though with subsidiary medieval meanings of honor and worship" (2). The statement by Dr. Favazza and Ms. Oman that "the study of culture has not yet been established as a cornerstone of psychiatry" is misleading. Culture envelops psychiatry and psychiatry must find its place within culture—a place that will be different, and of varying importance, in different cultures. Mental illness may be looked at, assessed, and dealt with in varying ways. The authors have not dealt with these issues, and have ignored large areas of culture—art, literature, religion, and, particularly, history. If these areas had been included, then the discussion of the appropriate term might have had some justification. "Cultural psychiatry" at first glance might be superior to "cross-cultural" or "transcultural." My preference would be "comparative psychiatry," which implies that the psychiatrist, trained and working in one cultural sphere, moves to another and studies the existence and manifestations of psychiatric disorders therein.

The authors bypassed the first psychiatrist who undertook such comparative study, Kraepelin (3). The insistence on Freud's "Totem and Taboo" (4) and Jung's "Symbols of Transformation" (5) is hardly relevant to the study of psychiatric disorders today. Furthermore, there is a *petio principii* built into the paper: the authors quoted anthropologists and sociologists who rely on psychoanalytic and psychiatric findings to support their hypotheses, and then psychoanalysts and psychiatrists cite the same anthropologists, sociologists, and ethnologists, and these findings support their own theories.

The conclusion that the authors have reviewed prominent concepts and findings is an understatement. They did not mention Jaspers' *General Psychopathology* (6), which contains a section on "The Abnormal Psyche in Society and History." This section provides a glimpse of the relevant literature as well as indicating ways of making it important to the clinical psychiatrist. The strangest feature of their overview is that only the English literature—actually, only the American one—was quoted and even this was done one-sidedly. Could the authors not open their (and our) horizon a bit wider?

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DIMITRIJE PIVNICKI, M.D.
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Dr. Favazza and Ms. Oman Reply

SIR: In our article, we included only English language references because *Journal* editorial policy limits the number of references that may be cited. We chose what we felt to be salient references which would be available to most *Journal* readers. A number of our references are to books and articles that do provide an entrée into the foreign language literature. We think our approach in this matter was the proper one for an overview article. We did not discuss Kraepelin's Javanese data because we were not writing a historical article. As for the charge that we built a *petio principii* into the article, we can only deny it. *Res ipsa loquitor.*

We stated in our paper that we would deal with "concepts basic to psychiatry." Dr. Pivnicki seems to feel that our choice of child rearing and basic personality, cognition, family and social networks, sex roles and behavior, alcohol use, communication, and therapy are not basic psychiatric concepts because they do not fit in with his definition of psychiatry. Of course, we are all entitled to define psychiatry as we see fit, but our critic's definition would appear to be exceptionally narrow.

Dr. Pivnicki would have us further the continuity of psychiatric work and not reach out to anthropology or sociology. With this line of reasoning psychiatrists should also refrain from learning from biochemists, neurophysiologists, psychologists, etc. The study of culture is complex and complicated. If anthropologists, sociologists, and social psychologists have developed useful methods for studying culture, it seems unwise to reject this knowledge just because it was not produced by psychiatrists.

After reviewing over 3,600 mental health articles and books with anthropological or cross-cultural themes, we stand by our assertion that the study of culture, in comparison with the study of psychology or of the neurosciences, has not been fully established as a cornerstone of psychiatry. Dr. Pivnicki states that he learned little from our article. He is certainly to be congratulated heartily on his fund of cultural knowledge. We wish that all *Journal* readers were as well-read as he because it certainly would advance the sophistication of work in cultural psychiatry.

ARMANDO R. FAVAZZA, M.D., M.P.H.
MARY OMAN, M.A.
Columbia, Mo.

The Military Family: Who Has Been Scapegoated?

SIR: Nietzsche noted that every word represents a bias. Certainly a published study of opinion generalized from a small undefined military population in the midwest to the total military community, done without serious consideration of statistical tests of validity, reliability, and standardization, represents a subjective bias. My own experience and the relevant literature on the military family (1–3) fail to confirm the ideas presented by Don M. LaGrone, M.D., in "The Military Family Syndrome" (September 1978 issue). Such caricaturization represents the error of extreme views. The author stresses the inadequacy of intrapsychic models and professes an environmentalism that is startling in its simplicity. Do we need to remind the author that the individual personality is never determined by the group as a whole, and no individual is representative of all the characteristics imputed to any particular group?

The delineation of a syndrome in its simplest operational definition as a "set of symptoms which occur together" is not even attempted. There is an attempt to impugn all military families as in some way pathological by designating them as a syndrome. The idea that military psychotherapists should not treat disturbed military families and their children because they themselves are in the military is absurd and speaks to a peculiar belief system and environmentalism which holds that people in the military are less human than others.

Relevant published research on the military family that stands in contrast to the author's views was ignored (1, 2).

There is some indication that psychopathology in the military child tends 1) to run more toward ego restrictiveness and inhibitions than impulse-ridden behavior and 2) to be less frequent than that in civilian children (2, 3). The author states that the incidence of child abuse in the military is five times the national rate. This might be interpreted as a testimonial to good case finding, medical care, and reporting procedures rather than a negative indicator. Recent studies at Ft. Lewis, Wash., and San Antonio, Tex., suggest the opposite finding (4, 5). The San Antonio study found that while the military was 20% of the area's population, it accounted for only 11% of all child abuse cases (4, 5).

The author did not consider the possible confounding of his data by the high incidence of noncommissioned officers' families in his sample. What he designated as the child-rearing techniques of the military family are probably a function more of socioeconomic level than of the specific nature of the military milieu, if we regard it in the light of Kohn's classic study (6).

There is little substantiating evidence that scapegoating is more common in military families. The vague notion of a coalition between fathers and the military, with dependents

excluded as the third part of the triangle, runs counter to all we know of family relations. Certainly, the lower divorce rate in the military community would point to greater family stability. The military commitment to child care is represented in AR 600-48, which requires that every military installation of more than 2,000 dependents establish a child advocacy program for the purpose of monitoring and coordinating all programs and services for children: Dr. LaGrone ignored this. In that sense, the military is the only community which has attempted to implement the inherent philosophy of the 1970 report of the Joint Commission on the Mental Health of Children. If scapegoating is an issue in this paper, it is the military and the military family that have been scapegoated.

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JON A. SHAW, M.D.
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Dr. LaGrone Replies

SIR: Dr. Shaw refers to Nietzsche's thoughts on bias and then betrays his own. His long list of complaints reveals an objection not just to what was said but to the fact that it was said at all. He seems to feel that an interactional model is too simplistic. I would suggest that an intrapsychic model is at least as restrictive and simplistic. I did not advocate an either-or stand with the use of theoretical models in my paper; I simply suggested that a systems model might assist the therapist in his understanding of military families.

There was no attempt to assign pathology to all military families, but I do feel that there are conditions in the military from which pathologic relationships might naturally evolve. The idea that military therapists should not treat military families because they are members of the same system is not absurd, and I believe the literature on family process would support my point.

Dr. Shaw refers to the "relevant" literature. I would ask him for the military definition of the word "relevant." While I was still in the military, I attempted to publish "The Military Family Syndrome." After eight attempts to have it cleared through "channels," I was finally told that it would not be cleared because "it would hurt the recruiting program." It was only after I left the military that I was able to send it to the *Journal*. Just how valid is military research when it is derived from a system that represses "unofficial" points of view?

Dr. Shaw implies that because there is a regulation (AR 600-48) for a commitment to child advocacy, this commitment actually exists. There is a difference. Perhaps Dr. Shaw would like to outline for us what actually has been put into practice and how effective it has been, using "statistical tests of validity, reliability, and standardization." Better yet, perhaps the military would do well to allow a neutral body of investigators to look at its mental health problems and to publish its report without repression.

Finally, there have been other studies that have reached conclusions similar to mine. Bloom's study was mentioned in my paper (1). Incidentally, his population was similar to the one in San Antonio that Dr. Shaw mentioned. White (2) studied a military population in Portsmouth, Va., and found that 59% of his patients were diagnosed as having a behavior disorder. The family dynamics he described were similar to those in my sample.

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DON M. LAGRONE, M.D.
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Psychotherapy or Consultation for Women's Conflicts?

SIR: In "Success or Failure: Psychotherapeutic Considerations for Women in Conflict" Carol Nadelson, M.D., and associates (September 1978 issue) discussed the conflicts experienced by many women today as they make career choices and the choice between a serious involvement in their work and family obligations. It has also been my experience that more and more women are facing career-related crises. I doubt, however, that the best source of help for these women is psychotherapy. To be sure, a short series of consultations may be needed to help some of them regain their balance or to ascertain that some are actually psychologically immature and have unrealistic expectations of work. In the case of married women, spouses should also participate in the consultation.

Prescribing psychotherapy carries the implication that a person is sick or has failed in some way because of her own conflicts. Actually, many of these women's problems are the result of their attempts to cope with adverse circumstances in a culture that is still highly ambivalent about women's success.

Many women have not internalized cultural values that assume that they, too, must achieve in the work world to realize their potential. This is further complicated by the fact that we know much less about female-male differences than we realize. More research in this area, free from cultural and political biases, is urgently needed if we are to avoid repeating the mistakes of the recent past in terms of men's and women's roles.

Like the authors, I see many single women approaching 30 who are torn between pursuing a successful career—for which one may need not only time but geographic mobility—and having a family. My view is that most of these women are no longer concerned about feeling deviant or different if they do not marry and have a family. Rather, they face a choice between satisfying their deep emotional needs to be part of a committed marital relationship and to have children and the realization that family obligations may pose a serious handicap to their career advancement. This dilemma was il-

lustrated in a recent article in *Fortune* magazine (1) which discussed a group of women who graduated with M.B.A.s from Harvard Business School in 1973. Some of them said they had decided not to have children; others who had children or planned to do so had given up ambitious career goals for which they are certainly as well prepared as their male counterparts.

Middle-aged women who opt for a career after raising a family are particularly vulnerable. Age *is* a handicap in the labor force unless one decides on a low-echelon job. I know women who are self-assertive, have the support of their husbands, and have prepared themselves adequately to enter the job market, but who cannot find suitable jobs or do not know how to compete in highly competitive arenas. Some of them end up accepting jobs that give them little satisfaction and that do not utilize their skills and qualifications. Others endure a life of discontent, unable to go back to their former emotional stance, to the times when they were looking forward to that "room of one's own" our society has been promising, and also unable to negotiate from a position of powerlessness. Their psychological paralysis, which is due to reality factors, is at the root of their depression and other health problems that seriously hamper their family and social relationships.

What these women need is not psychotherapy. They need to see consultants from the business and professional fields who are trained to present to them in a peer setting the realities they will confront, so they can make choices and plan realistically, according to their individual capacities and their emotional make-up. They need training in problem-solving strategies, in gamesmanship, and in realistic goal setting, and they need a working knowledge of the vicissitudes of upward mobility.

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CLARA G. LIVSEY, M.D.
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Dr. Nadelson and Associates Reply

SIR: We agree with Dr. Livsey that the problems of integrating career and family are complex and that alternative therapeutic approaches are indicated. We would, however, like to focus on some points where we differ.

We disagree with the idea that suggesting psychotherapy implies that the problem is one of illness. The importance of psychotherapy in facilitating coping or crisis resolution should not be overlooked. A person may use coping and defensive styles that are not in themselves pathologic but can be maladaptive to the new life situations presented by a career.

Conflicts that create difficulties in work achievement and satisfaction have long been considered appropriate for psychotherapy, which is then oriented toward facilitating successful adaptation. Traditionally, more men have been seen for problems centering around "work" and more women for concerns about "love." With recent cultural changes in roles for women, we are seeing more women who seek psychotherapy for conflicts about achievement and career.

We do not advocate that all women with work-related difficulties be seen in psychotherapy; we are making two main points in this regard. First, we are concerned that when reality issues concerning work are raised by a woman who is in psychotherapy the therapist consider them seriously and not see them as psychopathologic. Second, when a woman presents with symptoms (e.g., anxiety, depression, paralyzing ambivalence) concerning her work, which relate to intrapsychic conflicts and/or early life experiences and expectations, these should not be dismissed or seen as merely the reality problem of a working woman.

The career woman who experiences conflict between her commitment to her work and to her family may well seek and benefit from psychotherapy so that she can have a better understanding of herself and be able to find a more optimal situation. We do not believe, as Dr. Livsey implies, that family obligations necessarily impose a serious handicap on career advancement; on the contrary, the support and fulfillment of marriage and children may enhance one's energy and creativity. We heartily agree that research in these areas is needed. We believe that we must address issues of existing sex roles and their implications, as well as the process of formulation of goals and expectations for women. Although current evidence indicates that women are often conflicted and do experience role strain, we do not feel that this is a necessary condition.

MONA BENNETT, M.D.
MALKAH NOTMAN, M.D.
CAROL NADELSON, M.D.
Boston, Mass.

A Call for Clarification

SIR: In his article "Psychiatrists and Physical Examinations: A Survey" (August 1978 issue), Charles W. Patterson, M.D., stated that Karl Menninger (1) has advised caution in performing physical examinations of psychiatric patients. This statement is potentially misleading because it does not refer to the fact that Dr. Menninger, in the very work cited, emphasized that "the Physical Examination of his patient is one of the psychiatrist's responsibilities and one of his professional privileges." He qualified this statement to reemphasize the obvious—that physical examinations are part of the relationship between doctor and patient and must be done tactfully. In fact, he even emphasized the positive effect a physical examination may have on a psychiatric patient when he stated that the examination "disproves the common error of belief that psychiatrists are only 'talking doctors,' not real, honest-to-goodness 'touching' doctors."

In actual practice, physical examinations are always done on the day of admission to the C.F. Menninger Memorial Hospital and in outpatient cases prior to assignment of a therapist partially because of Dr. Menninger's influence.

It is important to clarify this matter so as not to discredit Dr. Menninger and other well-rounded psychoanalysts and to avoid widening the artificial gap between psychoanalytically oriented psychiatrists and other psychiatrists.

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Dr. Patterson Replies

SIR: Drs. Hamilton and Fisher believe that a statement in my article attributed to Dr. Karl Menninger is potentially misleading. They request it be clarified to avoid discrediting him. The statement in question was "Menninger . . . advised caution in performing physical examinations under certain circumstances because of potentially adverse consequences." Although Drs. Hamilton and Fisher do not indicate specifically how it is misleading, they may fear the reader will believe Dr. Menninger advises that physical examinations not be performed. They unnecessarily defend Dr. Menninger by quoting his statements about the usefulness of such examinations and by indicating the emphasis given to examinations at the Menninger Foundation. I did not state or wish to imply that Dr. Menninger advised against performing physical examinations; however, I am quite willing to have this point clarified.

The rest of their criticism reflects excessive sensitivity. They are concerned that lack of clarification could not only discredit Dr. Menninger but also "other well-rounded psychoanalysts" and widen the "artificial gap between psychoanalytically oriented psychiatrists and other psychiatrists." While my article does mention that the psychiatrist's future role is a topic of active speculation, it contains no criticism of "psychoanalysis" or "well-rounded psychoanalysts"; in fact, neither "psychoanalysis" nor "psychoanalyst" was mentioned.

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Sex and Psychiatry

SIR: In my considered opinion the "sexual revolution" in psychiatry has gone much too far, and it is time to regain a sense of balance.

The *Comprehensive Textbook of Psychiatry* (1) deals with schizophrenia in 141 pages but devotes 260 pages to sexuality—a curious, if not absurd, ratio. In a recent article, Renshaw (2) advocated "the 30-minute sexological," during which the psychiatrist would demonstrate the structural and functional intimacies of the genital apparatus to couples with sexual problems who had obviously missed out on sex education.

Psychiatry has been repeatedly chastised for having moved away from the mainstream of medicine. One of the main stumbling blocks in efforts to develop a stronger identification with medical specialties appears to be the grandiosity of the psychiatric profession. In the last quarter of a century, American psychiatrists have been getting involved and claiming to be experts in many areas that go far beyond the boundaries of traditional psychiatry. The list includes poverty, crime, violence, education, politics, minorities, women's issues, and sexual problems. Since the basic training and experience of a psychiatrist does not endow him with special expertise in any of these areas, the image of the contemporary American psychiatrist is evolving toward "Jack of all trades and master of none."

The recent heavy involvement of psychiatry in problems of human sexuality is the latest example of this trend. I think the assessment and treatment of sexual problems belong to "sexology," which could be construed as a medical or even a nonmedical specialty but not as a subspecialty within psychiatry. None of the pioneers of sexology was a psychiatrist, and Freud certainly never advocated sexual counseling as the treatment of choice for sexual conflicts.

Masters and Johnson and other respectable sex therapists deserve our admiration, support, and referrals of appropriate patients. Let us separate sexology from psychiatry to everyone's benefit. Let sexual counseling and therapy be done by sexologists and let psychiatrists conduct psychotherapy. I for one am hoping to see the day when the American psychiatrist will return to basics and be expected to focus his efforts on patients with neuroses, psychoses, or character problems, where his training and experience enable him to be a true expert. There is enough work to be done in these areas to keep psychiatrists busy perhaps for centuries to come.

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GEORGE GARDOS, M.D.
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Monocytosis and Agranulocytosis Due to Neuroleptics

SIR: The article "Routine WBCs and Agranulocytosis in Children" by Leonard R. Piggott, M.D. (August 1978 issue) correctly reiterated the conclusions of Ayd (1), Pisciotta (2), and Litvak and Kaelbling (3) that routine interim hematologic surveillance is not very helpful in detecting incipient agranulocytosis during phenothiazine treatment. The physician familiar with its pathogenesis, knowing the rather anaphylactoid or apoplectic onset of so fulminating a disease as agranulocytosis, is aware of the relative uselessness of this measure. In 1965, however, I reported a probable relationship between the increased occurrence of monocytes in the peripheral blood of patients treated with chlorpromazine and chlorpromazine toxicity (4). It had been my observation that certain patients who demonstrated an increase of monocytes during chlorpromazine treatment had a relative intolerance and inadequate clinical response to this drug. This might reflect its toxic effect on the bone marrow, especially the myeloblastic series of it. I observed monocytosis beyond the individual norm prior to treatment most commonly with phenothiazines that belong to the chlorpromazine model group, which have in common three carbons in a straight chain (diaminophenothiazines), the group of phenothiazines most capable of producing agranulocytosis. I observed this trend to a much lesser degree with thioridazine but not with other phenothiazines, which in the same context are not generally associated with agranulocytosis.

The monocytic response appears to coincide with gradual intolerance or sensitization to the chlorpromazine model phenothiazines, culminating, in extreme cases, in the anaphylactoid response of agranulocytosis or pancytopenia, especially if it occurs with or is precipitated by virus infections or concomitant administration of other drugs (additive or potentiating effect). Although mechanisms of bone marrow suppression and antibody-mediated destruction of leucocytes (drug-dependent leukoagglutinating activity) have

been described, the pathogenesis of most cases remains obscure. An immunologic mechanism for WBC destruction is supported by strong evidence in cases involving aminopyrine, which shows some structural similarity to the chlorpromazine model group of phenothiazines and has a high potential for causing agranulocytosis.

The genesis and function of monocytes is not completely clear. Their chief function, apparently by virtue of their active motility and phagocytosis, is the removal of bacteria, protozoa, and substances that act as foreign bodies (lipoid substances, tissue debris, pigments). Tissue deposits of mentioned phenothiazines could be considered foreign bodies and explain in part the monocytosis, but my observation in two cases of agranulocytosis (4) seems to indicate that the increased number of monocytes prior to and following the agranulocytic stage may reflect compromised neutrophile function. A recent study (5) stressed the importance of monocyte functional capacity in patients compromised by granulocytopenia or neutrophile defects.

In the light of a possible causative relationship between monocytosis and chlorpromazine model phenothiazine toxicity on the hematopoietic system, a periodic white blood count, at least in patients treated with these neuroleptics, would become more meaningful and possibly more helpful to detect preclinically a predisposition to agranulocytosis. It would be of general clinical interest to test this relationship mentioned also for other drugs frequently associated with blood dyscrasia.

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Dr. Piggott Replies

SIR: I appreciate Dr. Lutz's communication. The possibility that a monocytosis may herald an agranulocytic reaction is of considerable interest and may, if demonstrated in a larger number of cases, justify periodic white blood and differential counts on patients who are taking drugs which are known to have caused agranulocytosis. However, the actual incidence of agranulocytic reactions is so low that one would still have to consider carefully whether the chances of early diagnosis of an impending agranulocytic reaction would justify the expense and discomfort of repeated blood counts. Additionally, one must consider the possibility of finding false positive monocytic responses and the subsequent discontinuation of a needed medication. This might be of value in selected populations, such as women over 40, where there is a greater potential for the development of agranulocytosis. For the younger adults, adolescents, and children, where the reported incidence is exceedingly low, the routine use of predictive blood tests seems difficult to justify.

LEONARD R. PIGGOTT, M.D.
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Still More on Dream Theory and Neurology

SIR: J. Allan Hobson, M.D., and Robert W. McCarley, M.D., are to be congratulated on their two stimulating and elegant papers, "The Neurobiological Origins of Psychoanalytic Dream Theory" and "The Brain as a Dream State Generator: An Activation-Synthesis Hypothesis of the Dream Process" (November and December 1977 issues). The authors arrived conceptually at formulations very similar to those I put forth in a paper delivered to the Medical Section of the British Psychological Society in June 1964 and subsequently published in an article entitled "A Review of Psychoanalytic Dream Theory in the Light of Recent Psychophysiological Studies of Sleep and Dreaming" (1). I subsequently developed these concepts further and discussed possible functions of the dream in a number of papers, chief of which were "Implications for Psychoanalytic Theory of Psychophysiologic Sleep Research" (2) and a chapter entitled "A Freudian View" in *Dream Psychology and the New Biology of Dreaming (3).*

I agree with some of the criticisms expressed in the series of Letters to the Editor in the May 1978 issue of the *Journal.* I do not believe that Drs. McCarley and Hobson were clear in their differentiation of the D state as a neurophysiologic state, the dream process, and the dream as a psychological or subjective event. Moreover, I cannot agree with their essentially linear attempt at interpretation of Freud's psychoanalytic (i.e., psychological) theory as deriving directly from his neurobiologic model in the "Project for a Scientific Psychology" (4).

In my papers, I emphasized that new neurophysiologic data required a revision of classical psychoanalytic theory, particularly with regard to the notions of dream process, and noted that "during the D state there are both heightened arousal and input of internally generated signals." I emphasized that thresholds for permitting external stimuli to reach the brain were raised and indicated (based on the evidence then existing and now more amply confirmed) that inhibitory impulses originating in the locus coeruleus essentially paralyzed the organism. Freud was aware of the importance of motor paralysis, although he had no notion of the actual neurophysiology: his first statement about sleep and dreaming in the "Project" emphasizes that in sleep one is paralyzed. I also pointed out that bilateral extirpation of the locus coeruleus in cats results in motor behavior during the D state, which suggests that the cats are acting out hallucinations.

As did Drs. McCarley and Hobson, I pointed out that new knowledge indicated that the D state occurred cyclically throughout every night's sleep. Hence, one could not conceptualize the motor force for the dream process as instinctual drives and infantile wishes becoming connected to memory traces of the day's residue. I also emphasized that Freud's notion of the dream as the guardian of sleep required revision and that in fact the evidence indicated that there are complicated neurophysiologic functions which operate to ensure that sleep is preserved under ordinary circumstances so that dreaming can occur. However, this is not to say that the dream as a psychological event, i.e., dream content, is

not determined predominately by memories and human drives or motivation. In a fashion similar to their notion that Freud's psychological theory or model evolved directly out of his neural model, Hobson and McCarley implied that the dream evolves almost as a direct event out of the neurobiologic state, although they did allow for psychological input into the dream. However, if one looks closely at their figures and concepts, it seems clear that they conceive of the signals generated in the pontine reticular formation as information, and they emphasize that the derivation of dreams is related to vestibular impulses. I have conceptualized this differently as the internally generating signals allowing a framework upon which psychological perceptions can be constructed. Evidence suggests that signals originating in the pons activate the lateral geniculate and then in turn the occipital cortex along with the general activation of the forebrain. Those neurons would perceive the signals as if originating in the retina. We know that when imprecise percepts are presented, such as Rorschach ink blots or tachistoscopic stimuli, the human organizes them into meaningful percepts. Because of the special quality of the D state, these are then raised to consciousness (what Freud calls "quality") and are perceived for the moment as real events. In this sense we would not talk of the direct dream generator but rather the dream process generator that enables psychological processes to develop the dream.

I have suggested that the internal signal generation by the pontine reticular formation, which reaches sensory areas such as the occipital cortex as if coming from external receptors, is a neurophysiologic process akin to Freud's notion of movement in a regressive direction in the mental apparatus during the dream process and activating the system perception from within. I share the notion of the dream as a synthesizing process.

A brief comment on the wish fulfillment hypothesis, which I have discussed in detail, is in order. Certainly, the infantile wish can no longer be conceived of as the driving force for the creation of a dream. However, when one samples human mental activity, either waking or sleeping, one finds evidence of motivation. Hence, the notion of wish fulfillment may not be entirely in error in that sense.

In my papers, I also discussed the evidence that much of the forgetting of a dream is to be explained as a state-dependent phenomenon. However, I do not believe we can eliminate the notion of repression entirely. Both with dreams of patients and in my own experience, I have noted that a well-recalled dream told to another person, whether analyst or spouse, is often then forgotten by the dreamer but not by the person to whom he or she has told it. In therapy the dream memory may return following appropriate interpretation. Freud himself changed his mind about the rapid forgetting of dreams. In the "Project" he explained poor memory by emphasizing that dreams follow old facilitations and thus make no change. Also, because of the motor paralysis, they leave no traces of discharge. Very shortly thereafter, in "The Interpretation of Dreams" (5), Freud took exactly the opposite position that the forgetting of dreams was inexplicable unless one took into account the resistance or censor.

Drs. McCarley and Hobson suggest that Freud's psychological theory was a direct derivation of his neurologic model. My own view is that in the "Project" Freud was trying to develop a neurologic model to explain his clinical psychological observations. He clearly gave it up as being premature in terms of the state of neurologic knowledge and turned to what is the basic psychoanalytic model, especially as elucidated in Chapter 7 of "The Interpretation of Dreams" (5). Clearly, most of the notions developed in Chapter 7 and many not mentioned after the "Project" for many years are to be seen in the "Project" (including some, such as issues concerning intelligence, with which Freud never dealt in psychoanalytic terms). This does not mean that these ideas developed as a consequence of a neurologic theory; rather, they were attempts to explain observed clinical behavior and communications. Strachey corresponded with me after reading my initial paper and it is clear that this was his notion as well.

In my view, much of psychoanalytic theory needs revision, and modern sleep research has indicated some of the necessary revisions. Freud's psychoanalytic theory derives by analogy from nineteenth century physics. He did not hesitate to use nonscientific analogies, as witness his brilliant paper, "Notes on A Mystic Writing Pad," which provides insight into the possible function of dreaming and anticipates the cybernetic notion of the brain as a computer. The necessity for constant revision of psychoanalytic theory does not rest on the notion that it grew directly out of a neurologic model. The most direct and erroneous derivation from the neurologic, in my opinion, is the literal notion of psychic energy, which clearly must be discarded. Unfortunately, space does not permit a greater elaboration of my ideas and their relation to those of Drs. Hobson and McCarley.

The Hobson and McCarley papers, in spite of needing some rethinking and revision, are elegant and draw on data unavailable 14 years ago, particularly their own brilliant studies on the mechanism of the sleep cycle oscillation. I wish they had searched the literature more carefully in preparation for their papers and had not presented their concepts as entirely new ones.

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Drs. McCarley and Hobson Reply

SIR: We thank Dr. Hawkins for the opportunity to call to the attention of *Journal* readers his and others' valuable contributions to the study of the relationships between physiology and psychology, both in Freud's writings and in modern sleep and dream research. Our paper on the physiology and psychology of REM sleep and dreams was not intended to be a claim that efforts at this kind of relationship had never been attempted before; indeed, we made repeated references to the efforts in Freud's day to make it clear that these attempts have a long and continuing history. They were especially common during the 1960s, when the first electrophysiologi

data about REM sleep became available. We were unable to cite many of these articles because of space limitations, and we are pleased to have the opportunity to point to additional discussions of the literature of this era (1–4). We wish also to reemphasize the importance of the works of Holt and Amacher that were cited in our paper.

Our paper was rooted in the conviction that advances during the past decade in the understanding of cellular and specific neurophysiology of REM sleep were sufficiently rich and complete to form a new theoretic orientation, to lay a foundation for specific interpretations about the relationship between physiologic and psychological events during dreaming sleep. Both of our papers emphasized this sense of a need for a new theory and a new orientation, a need that Dr. Hawkins and others evidently did not recognize. This is illustrated in Dr. Hawkins' summary statement and conclusion in the last paragraph of his 1966 paper (his reference 1), which stated that, while some revisions were needed, "the basic psychoanalytic theory of the dream process is in the main correct . . .," a conclusion far from our view. Indeed, all other previous psychophysiologic papers of which we are aware have concluded that little, if any, serious change in psychoanalytic theory was necessitated by the new data.

Dr. Hawkins' thoughtful letter makes clear further specific differences in theoretical viewpoints, which we will discuss below.

1. Freud's neurology and Freud's psychology: We are uncertain about Dr. Hawkins' exact viewpoint on Freud's "Project." Dr. Hawkins states at one point, "Freud was trying to develop a neurologic model to explain his clinical psychological observations," but he then states that "Freud's psychoanalytic theory derives by analogy from nineteenth century physics." We think this uncertainty over whether the project is a neurologic theory derived from psychology or a psychoanalytic and psychological theory derived from physics well illustrates our own viewpoint: Freud used the concepts of natural science (physiology, with its links to physics and chemistry) and those of psychology interchangeably because he thought that reconciliation of physical and mental concepts would be simple, or to phrase it more technically, that there were identity isomorphisms between the two domains. Now, 80 years later, it does not seem so simple, but we still believe in the fruitfulness of the search for isomorphisms between physiology and psychology.

2. Mind-body relationships: Do we confuse them? Our forthcoming paper on mind-body isomorphisms and the study of dreams (5) emphasizes and makes specific our concepts of the separate nature of psychological and physiologic data and theory. In it we state,

> The most useful strategy in the scientific study of mind-brain relations is to regard mind and brain concepts as coming from different linguistic and conceptual systems. In accord with the phenomenologists, we see the common origin of all percepts, whether of objects or of emotions, in raw experiential consciousness and suggest this initial event furnishes the foundation for elaboration of both mind and body or brain concepts. . . . We do not seek to reduce physiological to psychological events or psychological events to physical events. Nor do we view psychological events as causing physiological events or vice versa. Instead we view the proper strategy in mind-body study as seeking areas of contact between the conceptualizations of physiological and psychological theory. Mind-body isomorphism is our name for this strategy; it implies a conviction that such matching or mapping between operations and objects in the two conceptual systems of mind and brain can be found.

We have attempted to be extremely careful in trying to separate mind concepts from body concepts. Although it is entirely possible that we have erred somewhere in our discussion of mind and body and not kept these distinctions, we have attempted to avoid this pitfall and will be grateful to anyone who points out specific lapses of this kind.

We think the mind-body distinction is an important one and one that is too often blurred in everyday speech. Dr. Hawkins says something he might not mean when he notes that "neurons perceive the signals as if originating in the retina." Perception is obviously a psychological concept. Neurons may be activated in some way, but they never perceive, sense, or feel. Our way of phrasing it, and the way that is consonant with our specific theory of isomorphisms, is that the psychological concomitants of neuronal activation of the same temporal and spatial characteristics are the same, regardless of whether the stimuli come from the retina or from the brainstem. More abstractly, we would say that psychological event X is isomorphic to physiologic event Y, regardless of whether event Y is caused by physiologic event Z_1 or physiologic event Z_2.

3. The importance of the specificity gained by using information from cellular neurophysiology. Dr. Hawkins suggests that the signals the visual cortex receives during REM sleep are "imprecise," analogous to Rorschach ink blots. Unfortunately, at this moment neither he nor we knows exactly how precise or imprecise they are. The important fact is that this is amenable to investigation: our most recent physiologic studies, done in collaboration with Dr. Jack Nelson, suggest that the information received by the forebrain from the brainstem during REM sleep includes specific coding for laterality of the eye movements of REM sleep. We look forward to continuing physiologic investigation that will enable us to make more definite statements about whether the information is precise or imprecise. Another difference concerns the interpretation of the physiologic basis of motor behavior of cats who have lesions abolishing the muscle paralysis of REM sleep. Dr. Hawkins describes their motor behavior as "acting out hallucinations," a concept difficult to specify in physiologic terms. Our interpretation is that the motor behavior represents activation of various systems for patterned motor and instinctual acts, independent of visual system activation, and that the psychological concomitants of information about these motor commands during human REM sleep form an important dream substrate.

4. How does one assay the correctness of Freud's model of dream hallucinations in the "Project"? We think the only similarity between Freud's model as sketched in the "Project" and the current view of visual cortex excitation during REM sleep is that both regard it as coming from internal rather than from external sources. Three differences are particularly notable. First, there is no regression: Freud precisely and clearly defined regression as backward conduction of nervous activity. In fact, neurons conduct in their usual directions during REM sleep. Freud was writing at a time when there was no notion of the unidirectional nature of neuronal activation, which proceeds from the neuronal body down the axon. Second, Freud's concept of what is regressing to the visual system, i.e., "nervous energy," is not at all compatible with modern physiology and must also be viewed as an error. Third, even if one were to substitute neuronal activity for nervous energy, the source is not instinctual

wishes as Freud thought but motivationally neutral pontine activation. Thus in regard to conclusions about this aspect of Freud's theory, we leave it to readers to decide which of the following statements they prefer: 1) With the above exceptions, Freud's physiology of the visual system excitation during REM sleep was correct, or 2) Eighty years of work in physiology have rendered obsolete most of Freud's ideas on the physiology of visual cortex activation in REM sleep.

We do have important areas of agreement with Dr. Hawkins. Sleep research has indicated necessary revisions in psychoanalytic theory. We think that the impact of discoveries in physiology will become increasingly important and that interchange between physiology and psychology will grow as we become able to discover more and more of the physiologic concomitants of relatively complex psychological events. It is our hope to be able to present increasingly specific relationships between dreams and the physiology of REM sleep. We are thus taking up the long-abandoned tradition of Freud's "Project" and welcome a continuing dialogue with progressive psychoanalysts like Dr. Hawkins.

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Withdrawal from Endogenous Opiates

SIR: In reading "Possible Withdrawal from Endogenous Opiates in Schizophrenics" by Michael Gitlin, M.D., and Michael Rosenblatt, M.D., (March 1978 issue) I was interested in the authors' observations on the possibility of "induced" withdrawal symptoms resulting from an interaction of naltrexone with the endogenous opiate system.

While I was with the National Institute on Drug Abuse (NIDA), I was Project Officer on the naltrexone development program and had the opportunity to have a continuing overview of all the patients who were taking naltrexone in the phase 2 research study. This included over 1,000 individuals who had taken at least a one-time dosage of naltrexone.

In September 1976 I coedited an NIDA research monograph on the progress of naltrexone development that included a paper entitled "NIDA's Naltrexone Research Program." In that paper I put forth the hypothesis for which Drs. Gitlin and Rosenblatt have now demonstrated possible confirmatory evidence. The relevant section read as follows:

> The only occasional side effect with some subjects seems to be an abdominal and gastrointestinal discomfort. When this was found at the beginning stages of treatment, it was attributed to minor withdrawal symptoms, because opiates were presumably still in the addict's system. However, these symptoms have been reported later on in treatment as well . . . .
>
> An intriguing scientific question, however, is: what interaction could naltrexone be having with the endogenously occurring opiate-like compound that has recently been isolated by researchers? Could, in fact, these abdominal symptoms be related to such an interaction? If an individual who is being maintained on naltrexone has a quantity of this endogenous substance secreted into his system and it is, in fact, opioid in nature, is it not possible to assume that some of the same symptomatology might occur as if an external opiate were entering this individual's system in a sufficient amount to cause such physical effects? This symptomatology might therefore include minor withdrawal symptoms characterized by the abdominal discomfort described. (1, pp 8–9)

I feel that this is an important line of research that needs further follow-up. We now have a glimpse into the possible interaction of antagonists with the endogenous opiates. This possibility should be pursued and further clarified. As narcotic antagonists evolve out of the research area and into the marketing/treatment area, we need to know how they might be affecting a naturally occurring human opioid system and what further ramifications such an effect might have.

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DEMETRIOS A. JULIUS, M.D.
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Drs. Gitlin and Rosenblatt Reply

SIR: We appreciate Dr. Julius' correspondence. The NIDA monograph he coedited came to our attention after our paper had been submitted to the *Journal*. We are indeed pleased that our work has given preliminary comfirmatory evidence to Dr. Julius' hypothesis. The recent work of Watson and associates (1) replicating in a double-blind design the earlier work of Gunne and associates (2) which showed diminution of auditory hallucinations in a subset of schizophrenics given naloxone intravenously, further strengthens the importance of opiate antagonist research in the etiology and treatment of psychiatric disorders. A possible approach to clarifying this issue, one which we have incorporated into our present research, is to correlate withdrawal and/or therapeutic effects in patients given opiate antagonists with changes in CSF or plasma endorphin levels. However, given the conflicting data obtained to date in opiate antagonist research concerning both therapeutic (3, 4) and side effects only multiple investigations using varied protocols will help clarify these issues.

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Correction

In the January 1979 issue there was an error in "Panic Attacks: Diagnostic Evaluations of 17 Patients" by Stephen F. Pariser, M.D., Bruce A. Jones, M.D., Emil R. Pinta, M.D., Elizabeth A. Young, M.D., and Mary E. Fontana, M.D. The sixth sentence of the Comment section should read, "Therefore, if MVPS is a cause of panic attacks, only 4 of the 17 patients would qualify for the diagnosis of panic disorder, because 6 patients who met other RDC for panic disorder also had definite MVPS."

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BOOK REVIEWS

The Psychopath in Society, *by Robert J. Smith. New York, N.Y., Academic Press, 1978, 157 pp., $7.80.*

Given the prominence of the diagnosis of psychopathy and the legal and moral questions it raises, the literature on the psychopath is relatively sparse. Prominent analysts of the caliber of Alexander and Karpman, along with Sheldon Glueck, have written on the subject, but the author states that Cleckley is the most charming to read as well as the most quoted authority. Cleckley views the psychopath "as basically insane, a sad creature who in spite of frequently superior endowment inevitably comes a cropper because of his failed moral development, dragging those who have trusted him down into the abyss of his own inevitable denouement."

Research on the subject has been confounded by casual labeling and the fact that the psychopath defies traditional mental health categories. The author notes that recognition of psychopathy as a specific disorder is typically dated to Pinel in 1801. In 1888 Koch introduced the idea of "psychopathic inferiority" in Germany and attributed it to hereditary weakness. Bleuler viewed psychopathic behavior as an emotional and moral deviation based on constitutional factors. Psychoanalysts are generally unsuccessful in treating psychopathic individuals because the analytic aim of loosening an overly strict superego is inconsistent with the fact that it is the psychopath's lack of inhibition that highlights his or her behavior.

In his second chapter, "The Charm and Winning Ways of the Psychopath," Smith notes that the psychopath's "most disarming aspect is a display of thoroughly beguiling charm." Sometimes aggression gets the upper hand, but even then there are flashes of charm that seem honest. Were he not a charmer, "he would be in no position to manipulate others, whether it is only to win a confidence or to use the other as perhaps a sexual or financial object." It is an innocent, even angelic, display that gives the observer a feeling that the psychopath is a person capable of full devotion and loyalty.

The search for a constitutional explanation for psychopathy, the subject of chapter three, has turned up very little if anything in the way of causation. In chapter four the author examines the social role and the learning theory formulations of psychopathy. In sharp contrast to those who seek an abnormality in bodily states stand those who view the individual as a product of his specific experiences of life. Something in the basic training of the sociopath is missing—the full steps of socialization have not been traversed. According to learning theory, the qualities of the sociopath come from imitation of a cold and distant parent, but Smith also discusses the paradox of how a psychopath manages to be so finely tuned to the needs of others that he can manipulate them.

In chapter five the author discusses "Cleckley and the Psychopath," noting that Cleckley is the recognized expert in the Anglo-American literature. It is not necessary to repeat Cleckley's concepts here; suffice it to recall the basic characteristics for his diagnosis: 1) superficial charm and good intelligence, 2) lack of remorse or shame, 3) untruthfulness and insincerity, and 4) pathological egocentricity and incapacity for love. As an example of psychopathy Smith describes Demara, who successfully posed as a politician, a naval officer, a psychologist, and a naval surgeon. The model thus posed for American psychopaths is, "Think big enough and the world can be yours."

In chapters titled "Psychopathy: Qualitative or Quantitative Entity?" "The Philosophy of the Marketplace and Psychopathy," "Psychopathy in Varied Cultures," and "Psychopathy and the Future" the author examines this condition, which is responsible for so much heartache and which plagued military psychiatrists throughout World War II. Smith cites Harrington's book titled *Psychopaths* (1), published in 1972, stating that Harrington took the fearsome view that the psychopath is the man of the future. Apparently, Harrington had written an article in *Playboy* as well, and Cleckley's withering comment on that article was, "Perhaps in this general and heedless effort to reverse the basic values anything traditionally regarded as undesirable or despicable might be stamped with the sign of approval."

The author discusses the views of other writers and of the counterculture. Little is said, however, about treatment. It was the opinion of Navy psychiatrists in wartime that the condition burns itself out when men get into their 50s. By that time parents have died and there is no one left to keep psychopathic individuals out of jail.

As to the present volume, I can recommend it for its complete survey of psychopathy. As I see the situation, it is pretty much the same as when the first edition of Cleckley's first book, *Mask of Sanity* (2), was published in 1941.

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F.J.B.

Organic Psychiatry: The Psychological Consequences of Cerebral Disorder, *by William Alwyn Lishman, B.Sc., D.P.M. Oxford, England, Blackwell Scientific Publications (Philadelphia, Pa., J.B. Lippincott Co., distributor), 1978, 967 pp., $75.00.*

In recent decades there has been a strong tendency for psychiatry to develop separately from internal medicine and neurology. This has been accompanied not only by changes in the theoretical and practical attitudes of physicians but also by the development of a clinical area that is usually referred to as a borderland, although the title No-man's-land may be more appropriate at times. *Organic Psychiatry* endeavors to cover this gap.

The author defines organic psychiatry as both a clinical

field and a clinical approach, with emphasis on the former. The first part (Principles) covers general aspects, such as the general psychological features of cerebral disorder, regional disturbances, clinical assessment, and differential diagnosis. There is also a section on terminology, although unfortunately the term "organic" is not further discussed. The second part, Specific Disorders, includes head injury, tumors, epilepsy, intracranial infections, cerebrovascular disorders, senile dementia and related disorders, endocrine and metabolic disorders, vitamin deficiencies, toxic disorders, and a final chapter on various other brain conditions leading to psychiatric illness.

The book is of course not a handbook that tries to be as complete as possible. Also, all topics are seen with emphasis on psychiatric rather than neurological or other medical aspects; this gives it a special flavor not found in most discussions of these conditions. The selection of material is not in all cases entirely clear, but the omission of specific child psychiatric disorders, for example, is explained in the preface as due to limitation of the author's experience. The emphasis on brain disorder results in some surprising but logical consequences: alcoholic disorders, for instance, are partly discussed in the chapter on vitamin deficiencies and partly in the one on toxic disorders, but a discussion of alcohol addiction and its social implications, as well as one of opiate addiction is missing. There are no illustrations, and the reader is usually referred to other works for technical details.

Within these confines—a postgraduate textbook of the psychiatric aspects of brain dysfunction in the adult—this book is excellent. It has the advantages of a one-author book, such as uniformity of purpose and style. The style, in fact, is lucid, and the book is a pleasure to read. The topics are extensively discussed vis-à-vis clinical features, etiology, pathology, and treatment, with a review of important publications. One particularly strong point is the presentation of many short illustrative case histories cited from the world literature. In his foreword, Sir Denis Hill states that "there is no comparable book in the English-speaking world," and a review of available texts would tend to confirm that opinion. He adds that it "will no doubt become standard reading . . . for postgraduate students of psychiatry and neurology." One may certainly wish this to be so (this might perhaps also result in a lower price).

The main problem with this work does not concern the author but the fact that it has evidently taken several years to print it, a fact that is somewhat surprising in this age of computers and space travel. This has resulted in the neglect of some recent developments; for example, there is only a very rudimentary treatment of computerized X-ray tomography. However, the text is carefully edited, and there are few errors.

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Pediatric Psychopharmacology: The Use of Behavior Modifying Drugs in Children, *edited by John S. Werry, M.D. New York, N.Y., Brunner/Mazel, 1978, 399 pp., $19.50.*

The field of psychopharmacology in child psychiatry has begun to develop intensively in the last decade. The state of the art and science is now about where psychopharmacology for adult patients was about 10 years ago. This volume represents one of several recent studies. Dr. Werry has successfully pulled together some order out of the current chaos. He has coordinated the contribution of 13 contributors from 8 centers in the United States, New Zealand, and Great Britain.

Although many of the findings are now out of date, the overall coverage and the organization of the chapters of this book are commendable. In the first 5 chapters on basic principles, thoughtful contributions can be found on the biochemistry of the various compounds, pharmacological principles in children, research methodology, and some of the important ethical, social, and legal considerations in research and treatment. Dr. Werry puts the field into historical and cultural perspective in his introduction. His views on the scene in the United States from his vantage point in New Zealand are refreshingly direct.

The second part of the book contains chapters on individual drugs and their use in children 12 years old and younger: stimulant, antidepressant, antipsychotic, antiepileptic, antimanic, antianxiety, and hallucinogenic drugs, vitamins, and other drugs. The drug categories are borrowed from adult psychopharmacology only for the sake of convenience because the individual drugs are used for conditions in childhood that do not relate to the uses these adult-oriented categories imply. This issue is particularly well covered by Gittelman-Klein, Spitzer, and Cantwell in chapter five. The same chapter reviews the new diagnostic classifications in *DSM-III* and *ICD-9*.

An important message is advanced in the early chapters: investigation in the field of psychopharmacology for children should no longer be left to "amateurs." Several authors reiterate the need for careful, sophisticated, and rather complex research techniques. Cantwell points out that the best evidence so far supports drug use for target symptoms rather than for diagnostic entities. For example, imipramine is used extensively in enuresis and school phobia, lithium may be used to control hyperactivity or aggressiveness, and haloperidol has specific indications for the Tourette syndrome. About the only diagnostic entity for which medication is specific is the "attention deficit disorder with hyperactivity" (hyperkinetic syndrome).

Several of the authors touch on an explanation for the lag in development and the complications in applying psychopharmacological principles to child psychiatry. There is an understandable reluctance to give medication to young people. Perhaps even more important is the recognition that the developing child metabolizes and uses drugs differently at each age. Until very recently very few psychotropic medications have even been tested, much less approved for use in children.

The psychosocial aspects of medicating children are also touched on, such as the "overmedication" of children in certain school systems to control behavior, the use of medications in state hospitals, and the general tendency for adults to feel that they need to "do something" about childhood problems, sometimes at the expense of the child's real needs.

On the negative side the book is puzzling in that it aims at no specific audience. The editor says he hopes the text will be "comprehensible to any professional working or training to work with exceptional children." However, many sections are too advanced for neophytes. On the other hand, parts of some chapters are too superficial and brief for advanced workers or clinicians. In addition, clinical studies have already modified, negated, or replaced some of the findings reported.

The chapters on individual drugs provide a comprehensive baseline of knowledge. The editor has organized these well

so that each drug or drug group is reviewed in a systematic way under headings of chemical structure, pharmacology, clinical effects, side effects, drug interaction, clinical indications, "nonindications," and "contraindications," along with clinical administration and, when relevant, social and ethical issues. All in all, within its limitations the volume represents a good cross-section of a changing field.

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Psychiatric Diagnosis: Exploration of Biological Predictors, *edited by Hagop S. Akiskal, M.D., and William L. Webb, M.D. New York, N.Y., SP Medical & Scientific Books (Spectrum Publications), 1978, 490 pp., $25.00.*

Only a few years ago psychiatric diagnosis was considered the hobgoblin of small minds. Psychoanalysts complained that diagnoses oversimplified the complexity of the individual patient, the Menninger school complained that illnesses were unitary rather than multiple, and scientific empiricists complained that diagnoses were too unreliable for use in research or good clinical care. This volume, the result of a three-day international neuroscience symposium devoted to the subject of psychiatric diagnosis, indicates that the tide of pessimism has reversed and that nosology has today become the preoccupation of many very fine minds. The participants include many of the leaders of modern psychiatry. Sir Martin Roth, Samuel Guze, Robert Spitzer, Jean Endicott, and Eli Robins are the first five contributors, for example, and their company is equally distinguished. The various contributions indicate that not only has psychiatric diagnosis regained intellectual and clinical respectability, it may indeed be the primary base on which scientific psychiatry must be built.

The presentations deal with four large subjects: clinical and biological criteria for psychiatric diagnosis, genetic principles in the classification of psychiatric disorders, biochemical and pharmacological correlates, and neurophysiological and neuropsychological correlates. Because the presentations were written primarily by investigators who have done pioneering research in each of these areas, they provide a comprehensive survey of current knowledge in psychiatry that also seems fresh and exciting. Distinguished "experts" sometimes become careless or casual when asked to write a survey of their own and related work, but that is not the case with this group. Nearly all of the contributors have taken care to write clear and informative reviews of their particular areas.

The volume leads off with an impressive discussion of the entire field of psychiatric diagnosis by Sir Martin Roth. He touches succinctly and thoughtfully on such various subjects as mathematical and computerized approaches to diagnosis, categorical versus dimensional approaches, the relationship between etiological and symptomatological classifications, research versus clinical classifications, and the assessment of reliability and validity. Guze discusses the important contributions of the "St. Louis school" to the definition and validation of diagnostic categories, and Spitzer, Endicott, and Robins describe their successful efforts to improve the reliability of psychiatric diagnoses. Corbett reviews specific problems in the definition of affective and schizophrenic syndromes, and Akiskal and associates review the methodological problems inherent in developing a system that is both clinical and biological and then apply this methodology to the description of cyclothymic disorders.

Genetic and family studies have provided powerful input for the development of modern nosology. Their contribution is summarized in a second set of papers. Matthysse and Kidd, both basic behavioral geneticists, describe the two mathematical models currently used most frequently to formulate genetic hypotheses, the multifactorial model and the single major locus model. Rosenthal gives a lucid summary of his work with Kety and Wender, using the adopted offspring method to explore the possibility that schizophrenic disorders form a spectrum. Cadoret and Cloninger present data suggesting that alcoholism and affective disorder may also form a spectrum, as may hysteria and antisocial personality.

The final two sections cover topics of equal importance by equally distinguished investigators: Dunner and Fieve on the impact of lithium on diagnosis, Carlsson on the neuropharmacology of schizophrenia, Janowsky and Davis on physostigmine and methylphenidate challenge tests, Wyatt and associates on platelet monamine oxidase, Meltzer and associates on neuromuscular dysfunction, Goodwin and associates on pharmacological differentiation of affective disorders, Lang on the psychophysiology of anxiety, Hartmann on sleep patterns as biological predictors, Buchsbaum on average evoked potentials, Lehmann and Ban on design issues in experimental approaches to diagnosis, Tucker and Silberfarb on neurological dysfunction in schizophrenia, and Whybrow on neuropsychology.

As a diagnosis "maven" myself, I anticipated reading this book with a bit of boredom. Instead, I was pleasantly surprised and usually read with interest and even excitement. It discusses most major issues currently relevant to the development of diagnostic systems and explores the various biological and psychological correlates that may be used to validate them. In general, each chapter is a clear summary of current thinking in a particular area. To be sure, some important areas have been overlooked, such as social factors, personality factors, psychological testing, and neuroendocrinology. In general, the editors have opted for a more "biological" perspective. The book is an excellent one overall. I recommend it for academicians and clinicians alike—for anyone who would like a brief but comprehensive overview of changes in diagnostic practice and the scientific rationale behind them.

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War on the Mind: The Military Uses and Abuses of Psychology, *by Peter Watson. New York, N.Y., Basic Books, 1978, 522 pp., $20.00.*

This book is a definitive account of the development and use of psychology in the military. Peter Watson, who is both a psychologist and a journalist, skillfully explores an area about which most psychiatrists have a cursory knowledge at best. Watson's research uncovered 7,500 studies published in this subject area since the end of World War II (more than 1 a day). Of the 146 organizations involved in military psychology around the world, 130 are in the United States. Only 80 of these belong to the Armed Forces. The rest are private, university-based, or hospital-based institutes or think tanks.

The author reviews the original involvement of military psychology in matching soldiers with military machines, training military specialists, and selecting troops. He then reviews psychology's involvement with "paramilitary"

areas—counterinsurgency operations, motivational studies of guerillas, the sociological and psychological effects of economic underdevelopment, and human factors in clandestine organizations, to mention a few.

In his introduction the author raises a number of provocative issues. In the following five sections he deals with different aspects of military psychology: combat, stress, determinants of loyalty and treason, survival, and the psychology of counterinsurgency. In these five sections the author is dispassionate and objective. Although he acknowledges his own occasional antimilitary feelings, he strives to give a reasonable account of both the good and the bad, the constructive and provocative (or dangerous) studies in each category.

Watson discusses the concern that secrecy unavoidably leads to poor methodology as well as useless and/or unreliable results. Without scientific interchange among scientists and without scrutiny by professional colleagues, a study's results can be disastrous. Watson notes that President Johnson escalated the bombing in Viet Nam on the basis of a psychological study by a prestigious organization that predicted the Viet Cong would succumb to a small amount of additional force. Watson also discusses a new technology that would link a pilot's brain to a computer so that the pilot would be able to fire and guide a missile to a target by thought processes. Finally, he describes the problems that arise when we do not know how to use the information we develop.

In his conclusion Watson posits that the popular fears of "psywar" or "mind control" are exaggerated. The vast majority of esoteric psychological experiments fail; instead, scientists explore the control of human behavior in war. In a discussion of war psychology versus peace psychology, Watson points out how studies of leadership, weapon effect, risk-taking, and survival can all be used to strengthen our efforts for peace. He urges more collaboration between military and nonmilitary psychologists and a decrease in the amount of military secrecy.

Although nationalism dictates that a nation's resources be used for its own survival, Watson proposes an international association of military psychologists to foster openness and collaboration among scientists. Research in this area need not increase the likelihood of war nor be abusive, but open scrutiny on an international level is necessary to ensure results that benefit mankind.

In this fascinating but at times tedious volume, Peter Watson challenges our profession and our society to be aware of the potential of the various psychological sciences and to use them for the best interest of all nations. This is not a book that everyone need read. However, the introduction and the conclusions contain information that behavioral scientists should possess in order to understand the full implications of the work in which we are involved.

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The Endorphins. Advances in Biochemical Psychopharmacology, Vol. 18, *edited by Erminio Costa, M.D., and Marco Trabucchi, M.D. New York, N.Y., Raven Press, 1978, 366 pp., $28.00.*

This book contains the material presented in Brescia, Italy, on August 28–30, 1977, at a symposium on endorphins. It seems that the papers were printed just as they were received; when an editor or, more probably, the publisher, found a typo or a poorly chosen word and replaced it, the replacements were made with a different type than the original. Raven Press has, with only a few exceptions, avoided using camera-ready copy; it should continue to do so until it has mastered such techniques as correcting authors' manuscripts and placing tables and figures well (e.g., after, not before, their citation).

With regard to the contents of this volume, the selection of topics and authors is very good, as is the coverage of most aspects of endorphinology—from the original concept of the existence of endorphins to the work in progress in the summer of 1977. Some chapters contain sufficient details of the laboratory techniques to allow the reader to reproduce the studies (a chapter by the Johns Hopkins group even gives the name and address of suppliers of nonstandard items). Others only guide the reader into the rapidly exploding literature on the endorphins. The literature references are current, as they have to be in this new field.

In the preface the editors use a phrase that, at least to me, exemplifies a current problem in pharmacological semantics: "endogenous ligands . . . function as putative neurotransmitters." Nothing, but nothing, can function as a *putative* neurotransmitter. Although a few authors misuse the naked term "neurotransmitter," more and more are abusing the modified "putative neurotransmitter."

A chapter by Floyd Bloom and his colleagues at the Salk Institute on the cellular localization and electrophysiological and behavioral effects of β-endorphin exemplifies what is good and what is poor in this volume. The authors are leaders in the field, the references include many papers so recent that they were still in press when this book was published, and the figures are adequately printed so that the reader is able to discern the point at issue. However, typographical corrections are blatantly inserted in different type from the original, and 20 or so errors were not corrected. Putting aside these nit-picking points, the reader will find one of the clearest descriptions of the methodology used in immunocytochemical studies (except that the tense goes back and forth from past to present) and one of the most thorough listings of regional distribution of endorphins to be found.

After making allowances for the fact that there is no table of abbreviations even though authors use different abbreviations for the same compound and some authors do not define their abbreviations, and for the fact that some authors presume familiarity with the field and others assume only general knowledge, I have to recommend that anyone interested in endorphins obtain a copy of this book and have it available on his or her bookshelf because it represents the best available review.

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[1]This review was prepared by Dr. Usdin in his private capacity. No official support or endorsement by the National Institute of Mental Health is intended or should be inferred.

Explorations in Psychoanalysis, *by Ralph R. Greenson. New York, N.Y., International Universities Press, 1978, 560 pp., $22.50.*

Collections of the contributions of eminent psychiatric and psychoanalytic authors provide readers with a convenient way to systematically survey a coherent body of work or the evolution of an idea or point of view. *Explorations in Psy-*

choanalysis is an aptly titled sampling of the writings of an outstanding teacher and clinician covering more than 40 years of psychoanalytic investigation. The papers are offered in chronological sequence and are derived from extensive clinical experience with a wide variety of patients. Each illustrative vignette is a model of succinctness, vividness, and recognizability. Indeed, the first dozen papers read like the reports of a dedicated naturalist or anthropologist, an observer of the human scene recording his findings as he probes deeper and deeper into a new land.

Greenson's studies of "The Psychology of Apathy," "On Enthusiasm," "On Phobia, Anxiety and Depression," and "On Gambling" earn him recognition as the de Tocqueville of the realms of neurotic phenomenology. As a result, the reader is provided with a comprehensible and demystifying guide to the understanding of patients with related problems. In each instance Greenson adduces the relevant insights afforded by psychoanalytic psychology and psychopathology and details the characteristic appearance and behavior patterns of the types under observation.

Most of the papers deal with specific problems of technique. Greenson suggests that this volume be considered a companion to his 1967 textbook (1), but it is immediately clear that a great many of the clinical and theoretical topics discussed bear closely on every aspect of psychotherapeutic work. An appropriate emphasis is placed on transference and countertransference phenomena. The author documents the necessity for a continuous scrutiny of the way in which the therapist relates to his or her patient. He underscores the crucial verbal and nonverbal communications from the patient that arise in response to fluctuations in this interplay. The way in which neglect of any of the varied aspects of transference, subtly present in every clinical moment, can seriously jeopardize understanding and therapeutic effectiveness is brought home in many convincing demonstrations. After repeatedly stressing the ubiquitousness of resistances that complicate any therapeutic effort, Greenson draws together a host of pertinent insights in a discussion of "The Problem of Working Through."

It should be noted that Greenson places great emphasis on his concept of the "working alliance." This formulation, related to Zetzel's use of the term "therapeutic alliance" (2), has had a far-reaching impact on psychotherapists and students of the theory of therapy. Its appearance in this volume gives us an opportunity to examine the ideas involved in the context of Greenson's papers on technique.

It seems to me that the central argument and the clinical illustrations offered in support of the working alliance are not entirely convincing. There is considerable question as to the value of separating the concept of a working alliance from that of the transference neurosis. The disadvantage of treating the resistances encountered in a patient's dealings with the therapist and in the conduct of the therapy itself as special problems is that it can lead to the belief that they are derived from something other than the patient's active psychic conflicts and must be handled in a unique way. In many instances the idea that a favorable climate for therapeutic work must be fostered by the therapist's behaving in a more "human" manner promotes interventions and expectations that do not advance an adequate understanding of the clinical situation at hand.

Mention should be made of one of the highlights of this valuable volume, Greenson's review of the fourth volume of *The Writings of Anna Freud*. It is a brilliant expression of his appreciation of and admiration for the contributions of Anna Freud, whose interests, particularly as they are expressed in these writings, are in many cases paralleled by Greenson's own. He succeeds in demonstrating how her papers of the period 1945–1956 reflect and clarify many clinical and theoretical developments in the burgeoning field of psychoanalysis. Because Greenson's extraordinary talents as a teacher and clinician are evident throughout this work, the book can be heartily recommended to anyone interested in psychotherapy and human psychology.

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HERBERT F. WALDHORN, M.D.
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Principles and Practice of Child Psychiatry, *by Stella Chess, M.D., and Mahin Hassibi, M.D. New York N.Y., Plenum Press, 1978, 486 pp., $20.00.*

In the first chapter of *Pragmatism* (1) William James proposed the now well-known separation of "tough-minded" and "tender-minded" philosophers. He described the tough-minded thinkers as devotees of empiricism, who liked pluralistic classifications based on observation and remained skeptical of anything beyond factual data. In contrast, the tender-minded thinkers were adherents of rationalism, who used reason to build speculative systems of abstract, unifying principles. James's dichotomy of thinkers is not only applicable to philosophers, as James himself was aware, but may be extended to a variety of disciplines that are enriched by the counter-play of both orientations. Psychiatry may be seen as having its "tough" clinicians and "tender" theorists perhaps Kraepelin and Freud could serve as notable paradigms. The subspecialty of child psychiatry is no exception to this bifurcation of orientations. The works of Frederick Allen, Erik Erikson, and Anna Freud, which concern themselves with expounding a general philosophical position, are balanced by the contributions of Leo Kanner, Loretta Bender, and Michael Rutter, which concentrate on the documentation of clinical fact.

This new book by Chess and Hassibi may be viewed as firmly in the tough-minded heritage and as exemplifying this tradition at its best. The authors adopt the viewpoint of an unbiased clinician, sticking close to the observable data and refraining from espousing any theoretical prejudice. The volume is divided into four sections. The first section presents an introduction to the field by tracing the history of childhood, documenting the course of normal development, and summarizing the major theories of development. The writing is straightforward and clear, with ample references to important studies. The second section is concerned with general concepts of psychopathology in childhood, stressing the various etiological factors culminating in a multicausal model of illness that requires comprehensive assessment. The third and longest section deals with particular syndromes of child psychopathology. These chapters are mainly phenomenological, listing symptom complexes in textbook fashion. The major contributions are cited and theoretical views noted in an objective and, at times, appropriately critical manner. I especially appreciated the inclusion of chapters o

physically handicapped and gifted children, two potentially at-risk groups that have not received sufficient attention. The last section covers treatment and prevention. Some readers may find the space allotted to individual psychotherapy somewhat short and skimpy, but the relatively brief presentation of this therapeutic dimension may reflect the authors' concern with the larger perspective of promoting child mental health, including prevention and methods of service delivery.

Chess and Hassibi have managed to compile concisely and clearly an impressive amount of information. As they mention in their preface, they were aided in the formidable task of covering so vast a field by noted authorities who graciously read and commented on selected chapters, thus adding the benefit of their expertise to this work. Aside from the documentation of extensive material, the impressive aspect of this book is the respect the authors show for the complexity of child development and psychopathology. Throughout the work the reader senses the wisdom gained through extensive clinical experience, which teaches that each child is an individual. The modest tone of the authors is certainly to be applauded. Chess and Hassibi often remind the reader of how much we still do not know about children's psychological health and illness. However, much of what we do know can be found in this book.

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JULES R. BEMPORAD, M.D.
Boston, Mass.

Habilitation of the Handicapped: New Dimensions in Programs for the Developmentally Disabled, *by Marvin Rosen, Gerald R. Clark, and Marvin S. Kivitz. Baltimore, Md., University Park Press, 1977, 366 pp., $18.50.*

Although the title of this book uses the broader term "handicapped," its major thrust is the treatment and management of mentally retarded people. This topic is sequentially presented in a series of 21 chapters arranged into seven sections. Each of the section titles focuses on a key issue, such as the nature of the habilitation process, institutional versus community-based challenges, and the major components of modern programs that can alleviate social and emotional deficits.

The nature, goals, and problems of the habilitation process in mental retardation are clearly presented in the first two sections. A succinct overview follows of the contemporary trend of deinstitutionalization, with particular reference to its reality underpinnings. For example, the world literature concerning follow-up studies of formerly institutionalized retarded citizens clearly documents the key reasons for failure in community placement—social and emotional factors (not the type or level of mental retardation). In the following two sections the authors present their research findings on key aspects of these social and emotional factors: self-concept, helplessness, and the personality residuals of excessive acquiescence. Next, an excellent section focuses on how to implement some very innovative treatment methods and management techniques for alleviating these deficits. By summarizing the entire book, the last section clearly articulates the current movement away from the late intervention of intellectual and emotional deficits (i.e., rehabilitation) to the initiation of developmentally oriented programs that can foster optimal developmental goals (i.e., habilitation).

This book is an excellent review and a clear presentation of the available modern approaches to maximizing the developmental and psychosocial dimensions of the mentally retarded in the United States. Accordingly, I highly recommend it to colleagues who include the care and treatment of retarded citizens in their clinical, teaching, and research careers in psychiatry.

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International Review of Research in Mental Retardation, Vol. 9, *edited by Norman R. Ellis. New York, N.Y., Academic Press (Harcourt Brace Jovanovich), 1978, 298 pp., $23.00.*

This volume is the latest in a series directed to psychological investigations in the broad field of mental retardation. It covers information processing studies and their implications for the care of the retarded, with several sections on labeling, psychophysiology, and biological defects as well as a group of reports on services for the retarded; there is also a chapter on idiots savants. The authors are established workers in psychology and special education; all 15 of the contributions come from American authors, although the title implies an international overview. The school and community experiences in normalization are also from the United States.

The book begins with the accurate notation that the climate for research in retardation is currently poor and that federal spending and the national emphasis on "breakthroughs" have led to disappointment. It examines studies of deinstitutionalization, mainstreaming, and the problems of placing the retarded in schools and in the general community. The papers include classically academic psychological studies in the mental processes of the retarded, addressing many of the basic problems in conceptualizing memory and learning processes. They review current issues under investigation in universities in the United States: masking, iconic memory, visual persistence, sensory storage, and scanning.

The results are not exciting; the conclusions cluster around such statements as, "The retarded are characterized by a lag in the development of certain central processes, rather than a structural defect in their processing systems" (p. 15). Another author concludes that research has been "both provocative and disappointing" (p. 24).

There is little psychodynamic contribution to these studies, although they do reveal the state of academic research. Direct applications for professionals dealing with the retarded are hardly addressed. However, the review of alternatives to residential care are based on actual experience. They are easier for clinicians to appreciate and indicate how the rhetoric of "normalization" has been biologically naïve and socially illusory. The resistance of teachers, students, and school administrators to the integration of retarded children into their classes is poignantly documented and clearly delineated. There are helpful data about the many types of facilities for the retarded and the myriad ways in which the definition of group homes, semi-independent units, and protected environments will determine the funding and function of these facilities.

The work has sections of great interest to psychologists,

teachers, and community psychiatrists who deal with retarded populations and has value for considering the ways of getting psychiatric patients out of hospitals into community settings. It also provides a good "state of the art" cross-section of the psychological investigations in mental retardation. Overall, it can be viewed as a sound reference volume for specialists.

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Social Skills and Mental Health, *by Peter Trower, Bridget Bryant, and Michael Argyle, with John Marzillier. Pittsburgh, Pa., University of Pittsburgh Press, 1978, 301 pp., $12.95.*

This book is a volume in the Contemporary Community Health Series of the University of Pittsburgh Press. It was written by the research and project staff of the social skills research project of Littlemore Hospital, Oxford, England. The book consists mainly of a careful review of the literature on various interpersonal and social skill development techniques and a manual for assessing social deficits and training clients to improve these deficits.

A large number of techniques for improving social skills have been developed over the past decade. A number of these involve interpersonal skills—verbal and nonverbal communications, assertiveness training, and decision making. These skills are especially needed by individuals who are socially inadequate as a result of passive, inadequate, or asthenic personalities. *Social Skills and Mental Health* reviews the literature concerning the effectiveness of training programs to remedy these interpersonal skill defects and then outlines in manual form the program developed at Littlemore Hospital.

The review of the literature in the area of interpersonal behaviors is excellent, including listening, observing, speaking, asserting, planning, and how these can be taught. This area of skill training is important and must be given greater attention as we move to carry out more mental health services in the community; it will be especially needed for chronic mental patients returning to their communities after years of hospitalization. However, it appears that some of these techniques may not be as effective with chronic patients as they are with individuals with inadequate personalities who come to outpatient clinics. One wonders why these rehabilitative training programs are referred to as "therapy," but this is a fine point because they are important.

The second part of the book, the manual for assessing and teaching interpersonal behavorial skills, is very specific and graphic. It should be of great use to those who work in mental hospitals, halfway houses, social rehabilitation programs, and community mental health centers oriented to developing social skills.

HAROLD L. MCPHEETERS, M.D.
Atlanta, Ga.

Living in Prison: The Ecology of Survival, *by Hans Toch. New York, N.Y., Free Press (Macmillan Publishing Co.), 1978, 310 pp., $12.95.*

The subtitle of this book is appropriate. Anyone who has had the experience of working directly with inmates will find the concepts so vividly portrayed by the author and his coworkers familiar. Hans Toch has succeeded in communicating the stresses of prison life as seen by inmates in several well chosen institutions.

The greater part of the text is based on inmate interviews illustrating how the environment of the prison is perceived by the individual. From these interviews the major areas of concern are spelled out: safety, freedom, activity, privacy, structure, support, and feedback. After reading the inmate interviews, one certainly feels that the word "concern" is an understatement. The author views the importance of each of these concerns in a transactional framework; each depends on many variables but mainly on inmate personality and institutional setting.

The author shows that by listening to each inmate one can make better choices in the classification process. Ideally, each inmate would have extensive testing and interviews to evaluate his adaptive strengths and weaknesses to enable one to match him to the proper institutional environment. Admitting fiscal restraints, Toch and his team have developed a Prisoner Preference Profile to assist in the classification process. The last chapters of the book examine the validity of the Prisoner Preference Profile to the extent that the results of the inventory make sense in terms of whatever else is known about the inmate.

Age, ethnicity, marital status, and other demographic data correlated well with inmate concerns. For example, older inmates were less concerned with freedom than younger inmates. The comparison of each of the demographic variables with each of the inmate concerns seems to validate the instrument for use as a time-saving device in the classification process.

The inmate interviews excerpted in the text are certainly valuable in the sense that they serve to describe and explain the development of the Prisoner Preference Profile. They are just as valuable in giving the reader a sense of the tremendous impact of the prison environment on incarcerated individuals. The section on stress and its amelioration and especially the chapters on "Niches in Prisons" and "Living in Protection" should be read by anyone who works in the correctional system and may be called on to make decisions relating to the health and well-being of inmates. One has to know the right questions to ask inmates with symptoms of anxiety, depression, or other indicators of defensive structure. In many cases the answers are found by searching out with the inmate his concerns about the prison environment. The author has given us an appreciation for the real feelings of prisoners and their attempts to adapt to and change their prison environment.

DAVID L. HEDBERG, M.D.
Hartford, Conn.

Consciousness and Self-Regulation: Advances in Research and Theory, Vol. 2, *edited by Gary E. Schwartz and David Shapiro. New York, N.Y., Plenum Press, 1978, 428 pp., $22.50.*

Reviews of basic research ordinarily permit only limitec extrapolation to the clinical setting. *Consciousness and Self-Regulation, Vol. 2,* however, contains in several of its 1(chapters a surprising amount of information relevant to psychiatric practice.

Pope and Singer's chapter, "Stream of Consciousness,' for example, reviews research in attribution theory, attention-focusing, and memory selectivity. These areas, impor-

tant for psychotherapists who would consider themselves scientists as well as artists, are embarrassingly unfamiliar to most psychiatrists. Pope and Singer reject oversimplified explanations of cognition, including the psychoanalytic denigration of primary process as immature thinking.

Although the concept of repression has generally not fared well in the hands of researchers, Sackeim and Gur do moderately well by it. They methodically forge a case in support of self-deception as a genuine phenomenon. In the key experiment, subjects who often misidentified their own voices on audiotape also evidenced autonomic changes that implied accurate self-recognition.

After demonstrating that the cognitive factors affecting eating behavior are manifold, intricate, and interactive, Rodin emphasizes the fragility of self-control in individuals with weight problems. The fundamental difficulty is presumed to be either constitutional or developmental. Fat people—even formerly fat people—show heightened sensitivity to external cues, including nonfood cues; however, they seem impaired in their ability to generate images entirely from imagination. Some of Rodin's data appear compatible with the fat-cell theory of hunger, which is not discussed.

Stroebel and Glueck allege that patients with autonomic hyperactivity lost most of their anxiety symptoms during an eight-week course of twice-daily meditation. The authors suggest that specific psychosomatic symptoms may respond better to biofeedback than to meditation. A striking finding is the poor compliance by psychiatric inpatients with Wolpe/Jacobson or Benson relaxation training compared with the good compliance accorded meditation. The latter apparently was less tiresome to the subjects. Stroebel and Glueck's enthusiastic claims, however, are not in tune with the three related chapters in this book.

Widespread research interest was kindled by early biofeedback reports from Miller and DiCara, who studied operant conditioning of autonomic responses of curarized animals, and Kamiya, who studied voluntary enhancement of alpha brainwaves and consequently of pleasant relaxation. A decade's work has brought forth a considerable body of data and some promising therapeutic applications. Ironically, however, the original studies have been seriously questioned. Roberts' chapter in the present volume ruefully concludes that "the phenomena reported in these experiments are unlikely to have been a product of learning. . . . The power of operant conditioning as a technique for treating visceral pathology appears to have been overstated in the past."

Orne and Wilson demolish essentially all of the popular claims about alpha biofeedback training for the achievement of health or bliss: "Once novelty and visuomotor effects are eliminated, alpha augmentation may be the product of relaxation in one individual and of hyperarousal in another, while a third may show little relationship between subjective state and alpha density." Orne's exciting talent for always asking yet another discerning question is fully evident in this chapter, a masterpiece of research detective work.

Lynch and Schuri reply to the question, Can individuals learn to modify the automatic regulation of the peripheral vascular system? Their answer: yes, sometimes, to some extent, and especially if the subjects are children; but not by means of biofeedback or direct conditioning. Classical conditioning may alter peripheral vascular response indirectly through the agency of an antecedent "central" reaction. Other influences could be focal isometric exercise, subject/experimenter interaction, and certain cognitions.

Ádám, one of the book's two eminent contributors from behind the Iron Curtain, gives new meaning to "making the unconscious conscious." Human subjects stimulated in the duodenum or on the cervix uteri showed EEG changes but felt nothing at a certain voltage. As verbal feedback was provided, the subjects began to perceive the visceral stimuli more and more readily. One wonders if this work could have clinical application in such problems as enuresis, encopresis, and anorgasmia.

Luria, the Soviet scientist to whom this volume is posthumously dedicated, provides a broad neuropsychological review and update. He depicts human consciousness as a brain network coordinated by language and continuously developing in response to life experiences. This conception of consciousness renders untenable birth trauma, primal scream theories, and the literal interpretation of such phenomena as hypnotic age regression and multiple personality.

Finally, the chapter by McGuigan succeeds in establishing that nonoral motor responses are regularly associated with speech and thought. Whether these motor responses are fundamental or even important to speech and thought is something else again.

Most psychiatrist readers would welcome more help from the editors. In future volumes Schwartz and Shapiro might consider attempting an integration or at least an explanation of conflicting viewpoints.

JOSEPH S. SILVERMAN, M.D.
Altoona, Pa.

Family Therapy in Clinical Practice, *by Murray Bowen, M.D. New York, N.Y., Jason Aronson, 1978, 547 pp., $25.00.*

Murray Bowen, one of the foremost pioneers in family therapy, has significantly increased the therapist's theoretical and clinical range. Because for him psychoanalysis and current practice were not resting places but stepping stones to general systems theory, the meaning and scope of the therapist's involvement have been greatly extended. As a consequence, this book, a collection of Bowen's most important papers published since 1957 representing the evolution of his family systems theory, will be of serious interest to all mental health professionals.

Bowen was not content with the opportunities for brilliant intuitions and philosophical operations that are so often the delight and comfort of the therapist's task. Nor would he accept the givens of his time as a place to start. His work demonstrates an original thinker's slow, methodical accumulation of data in clinical practice, research, and teaching, hewing as close to a scientific model as possible. Observations of results led to such changes as seemed useful. This is the essence of modern "operation control."

For example, the hospitalization of the family of a schizophrenic patient as well as the patient in a research study conducted during 1954–1959 at the National Institute of Mental Health led to clear and amazing observations about the relationship of the "patient's" behavior to what happened in the family and the hospital staff. Heretofore unavailable data and modifications became available for testing. The results were a family concept of schizophrenia, the development of a method of family therapy, and extension of the family relationship patterns seen in schizophrenia to families with less severe forms of emotional illness.

A serendipitous control study became available before Bowen actually realized it was such. In 1967 he discovered that psychiatric residents he saw only once a month informally, who were encouraged to visit their relatives and establish a person-to-person relationship but not get drawn in-

to their emotional system, made greater progress in working out problems in their nuclear families than residents he saw weekly in formal family therapy with their spouses. This discovery was a turning point in Bowen's professional development. The implications of this finding, if further validated, offer hope for mental health and preventive medicine in a society in which the progress of technology has tended to separate families and weaken roots.

One semantic consideration and a caveat should be mentioned. Labeling is often a problem in new work. Combinations of three relationship names, e.g., "father-mother-child," can denote the interface of many different systems configurations. When such a "triangle" is discussed, a modifier is needed to make clear that the three people do not relate in only one way. For example, one could speak of progressive triangles or regressive triangles. The caveat has to do with Bowen's discounting of group therapy. As often happens when a very experienced practitioner of one method regards unfavorably another method with which he or she has less experience, Bowen fails to recognize the applicability of group therapy to family systems. He makes the point that he does family therapy with one person but then inconsistently refers to the therapy group as a "number of individuals" rather than regarding it more fittingly as a "number of family representatives." In so doing, he eliminates the possibility of helping his "low-scale" families (low on the differentiation-of-self spectrum) to whom he offers so little hope. In such cases, when the resources of one therapist may be overwhelmed, a therapy group or a multiple therapist team (e.g., Whittaker and associates) offers the input of several family systems. This is not only a quantitative but a qualitative addition.

This criticism does not take away from the magnitude of Bowen's contribution. In my opinion, not every practitioner has the therapeutic personality to use Bowen's method successfully. To remain "detriangled" in the therapeutic system, special training is usually necessary, as well as the actual experience of differentiating from one's own family. However, the mental hygienic possibilities as Bowen's method becomes part of the culture (as Freud's work has) are very great. It could be a revolutionary contribution.

DAVID MENDELL, M.D.
Houston, Tex.

The Language of Change: Elements of Therapeutic Communication, *by Paul Watzlawick. New York, N.Y., Basic Books, 1978, 166 pp., $11.00.*

Dr. Watzlawick promises a challenging and innovative discussion of the use of language in psychotherapy. He attempts to synthesize research data on brain lateralization and to present an approach to the use of this information for the psychotherapist. Unfortunately, the promise is not fulfilled.

Throughout the book there is an intermingling of philosophical concepts and literary references to add emphasis and make distinctions in areas that are less clear than the author proposes. Watzlawick draws sharp distinctions between right and left brain functioning on the basis of recent as well as classical studies. Although research in the last decade has produced considerable support for the lateralization of brain functioning, clinical implications are as yet far from accurately charted. Right and left brain differences clearly exist, but it appears that in intact humans sharp divisions are not as well-defined as the author proposes. Integrated functioning and synthesis occurs, but special emphasis is evident in data on asymmetric activation in normal individuals, handedness, and sex differences in brain organization. Preliminary data also suggest the possibility of lateralization in schizophrenia and affective disorders. However, this is not clearly established, nor are the implications understood.

The author's purpose in providing evidence for the separation of right and left hemisphere functions is to explore the implications for therapy. He proposes a language that "blocks the left hemisphere" and "speaks" to the right. In this endeavor he uses clinical material largely drawn from the work of Dr. Milton Erikson, which does not provide a definitive model. The examples range between those which are useful and interesting to those which lack sophistication and depth and are at times manipulative and disrespectful to patients. In any event, they are not astonishing revelations. Watzlawick often sets up straw men to topple. He does not consider newer developments in theory and technique, including those from psychoanalysis and behavior modification. The value of the text resides in its rich store of literary examples and some interesting clinical material, but it is hardly the unique and revolutionary approach promised at the outset.

CAROL C. NADELSON, M.D.
Boston, Mass.

The Private Worlds of Dying Children, *by Myra Bluebond-Langner. Princeton, N.J., Princeton University Press, 1978, 275 pp., $12.50.*

The social scientist can make important contributions to pediatric oncological care. Bluebond-Langner does that in her anthropological study of the child's view of a pediatric hospital oncology ward and clinic. To present the child's view is always refreshing and, even if the children are dying of leukemia, their ways of adapting to, denying, subverting, ignoring the hospital's social structure, and otherwise trying to maintain their real values in the face of death are so authentic that they give zest and dignity to a world that is catastrophic for the children and their families. The author's device of a play to convey the drama of a child's world is a sparkling beginning for the book.

The perspective of the social scientist allows the author both an armor and a vulnerability that a "real" member of the health team would never know. Bluebond-Langner's vulnerability comes from the fact that she was not a member of a "helping profession" in this hospital setting. She was not there to help but to learn, understand, and to do no harm. In fact, she says that the clinical workers showed unvarnished hostility toward her when one patient relapsed. At times, she seemed to be in the way, at times too peripheral, but she succeeded in observing 32 patients between ages 3 and 9 years. The things she found out can only aid the humane clinical worker.

The armor protecting the author could be envied by pediatricians, one suspects: she had the freedom to move forward only when she had the fully informed consent of the child. Indeed, she did not intrude when the child did not welcome her; she could leave as soon as a parent came to be with the child. At all other times, however, she was an empathetic presence, remaining long hours with the children to provide companionship and understanding. To be able to talk with children under a contract that one will not be intrusive would

be a desirable Utopia for clinicians on a leukemia ward. The author's implicit message is that if clinicians desired it, they could take on more of the noninterventive outlook of the anthropologist. I agree; children respond honestly to honest inquiry about their private worlds. Even dying children are responsive:

> I will act as an observer. I will talk with the child only when he speaks with me. At no time will I introduce the subject of his illness, or ask questions about his condition. I am interested in what is on the child's mind and what he chooses to express. I am interested in his means of communication and how he chooses to use them with regard to the variety of plans and purposes a child has. (p. 49)

The preceding quotation might have been taken from an informal statement by Jean Piaget or David M. Levy, but Bluebond-Langner gives no sign that she has experienced the slightest breeze from Piaget on the matter of a child's self-concept or problem-solving skills. Piaget would have made very apt background reading and would have altered and added credibility to some of Bluebond-Langner's generalizations. Her generalizations of what the child knows are skewed in favor of the cognition of a 9-year-old. That bias—almost a fatal blunder—pervades chapters three through seven. Therefore, what is supposed to be original is sometimes based on unfamiliarity with other scientists who have reported their work with children in the non-social sciences literature.

Whenever adults undertake to colonize the head of a child, they portray the child as a budding young criminal living according to a pleasure principle; at the same time the adults curb the child from obtaining gratifications until he or she is older. "Later" is a strong word for adults who socialize children. The child must wait, taking on a perspective of preparing for pleasures later on. The leukemic children presented in this book have no future pleasures. They know it, and they try to make do. One closes this interesting, tender book with a wish that the adults could do their jobs as well as the children do their dying.

PAUL L. ADAMS, M.D.
Louisville, Ky.

Minimal Brain Dysfunction: Fact or Fiction. Advances in Biological Psychiatry, Vol. 1, *edited by A.F. Kalverboer, H.M. van Praag, and J. Mendlewicz. Basel, Switzerland, S. Karger, 1978, 107 pp., $25.50 (paper).*

A review of a book of collected papers follows a fairly well agreed on format. The reviewer notes that the papers are of mixed quality, making any generalization difficult. No reviewer has—to the best of my knowledge—applied what might be called the "medical rule of thirds." As we all remember, a handy mnemonic for most treatments is that one-third of the patients improve, one-third get worse, and one-third remain the same. By analogy, one-third of the articles in collected volumes generally seem to be enlightening, one-third uninspired, and one-third toxic to the reader's intellectual organization.

The novice who wishes to learn something about minimal brain dysfunction need not read this book because a number of broader summaries are available elsewhere. To those of us obsessives who like to keep up this review produces a sigh of relief because it is one book in the torrent that need not be read. The significance of this book is that, finally, our European colleagues have taken notice of the fact that minimal brain dysfunction, which has been the object of so much attention in American child psychiatry, *may* be a "real" syndrome. This is an advance, illustrated by an epidemiological study conducted by the productive and creative English psychiatrist Michael Rutter, who found one hyperactive child on the Isle of Wight.

A number of the contributors to this book feel uncomfortable with a minimal brain dysfunction syndrome because patients given this diagnosis need not have identical characteristics. This is hardly unique to minimal brain dysfunction; it is characteristic of other syndromes accepted on the Continent, such as the schizophrenias and affective disorders. There are a few other interesting errors in logic, such as the idea that the use of one name implies a unitary etiology. Otherwise, the issues discussed in the book are no longer discussed in American academic circles; these include the relationship between demonstrable brain damage and behavioral deviations and the relationship between neurological findings and behavioral difficulties. The paper of most interest is that by Grahame-Smith, who succinctly reviews the current animal models for the syndrome.

The fact that this book has appeared in Europe is indicative of a narrowing of the child psychiatric cultural lag that has existed between the United States and Western Europe. To those of us who believe that in minimal brain dysfunction we are dealing with a phenomenologically distinct and important syndrome characterizing many disturbed children and some adults, this cannot but be welcome. As behaviorists have observed, even critical attention is more positively reinforcing than complete neglect. Less cynically, I hope this book signifies that some of our colleagues in Western Europe are beginning to consider and deal with this important and common, if misnamed, syndrome.

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Books Received

This listing acknowledges the receipt of recent books. Books of particular interest to the readers of this journal will be reviewed as space permits, and copies of the reviews will be sent to the publishers. Books cannot be returned to the publishers.

The Survival of Domination: Inferiorization and Everyday Life, *by Barry D. Adam. New York, N.Y., Elsevier, 1978, 171 pp., $15.00.*

Mental Capacity: Medical and Legal Aspects of the Aging, *by J. Brooke Aker, Arthur C. Walsh, M.D., and James R. Beam. Colorado Springs, Colo., Shepard's (New York, N.Y., McGraw-Hill Book Co., distributor), 1977, 366 pp., no price listed.*

Medical Examination Review Book, Vol. 7A: Textbook Study Guide of Psychiatry, 2nd ed., *by James R. Allen, M.D., and Barbara Ann Allen, M.P.H., Ph.D., A.C.S.W. Garden City, N.Y., Medical Examination Publishing Co., 1978, 144 pp., $8.50 (spiral-bound).*

Transmethylations and the Central Nervous System, *edited by V.M. Andreoli, A. Agnoli, and C. Fazio. New York, N.Y., Springer-Verlag, 1978, 182 pp., $29.00.*

Severe and Mild Depression: The Psychotherapeutic Approach, *by Silvano Arieti, M.D., and Jules Bemporad, M.D. New York, N.Y., Basic Books, 1978, 441 pp., $20.00.*

Psychological Statistics: A Case Approach, *by Carl Auerback and Joseph L. Zinnes. Philadelphia, Pa., J.B. Lippincott Co. (New York, N.Y., Harper & Row, distributor), 1978, 427 pp., $13.95.*

Self-Mastery Through Self-Hypnosis, *by Roger Bernhardt and David Martin. New York, N.Y., New American Library, 1978, 174 pp., $1.95 (paper).*

Art of the Huichol Indians, *edited by Kathleen Berrin. New York, N.Y., Harry N. Abrams, 1978, 212 pp., $9.95 (paper).*

Liquor and Poverty: Skid Row As a Human Condition, *by Leonard U. Blumberg, Thomas E. Shipley, Jr., and Stephen F. Barsky. New Brunswick, N.J., Rutgers Center of Alcohol Studies Publications Division, 1978, 262 pp., $14.00.*

Social Conflict and Mental Health Services, *by Robert D. Borgman, Ph.D. Springfield, Ill., Charles C Thomas, 1978, 333 pp., $24.50; $18.50 (paper).*

Handicapped Children: Strategies for Improving Services, *by Garry D. Brewer and James S. Kakalik. New York, N.Y., McGraw-Hill Book Co., 1979, 596 pp., $19.95.*

Aspects of Community Psychiatry: Review and Preview. POCA Perspectives 7, *edited by Josef M. Divic, M.D., and Michael Dinoff, Ph.D. University, Ala., University of Alabama Press, 1978, 174 pp., $12.75.*

Behavior Therapy: Scientific, Philosophical, and Moral Foundations, *by Edward Erwin. New York, N.Y., Cambridge University Press, 1978, 250 pp., $16.95.*

Hypnosis at Its Bicentennial: Selected Papers, *edited by Fred H. Frankel, M.B.Ch.B., D.P.M., and Harold S. Zamansky, Ph.D. New York, N.Y., Plenum Press, 1978, 301 pp., $27.50.*

Stress and Physical Health: A Bibliography, *compiled by Robert Friis. Irvine, Calif., Human Behavior Research Group, 1978, 35 pp., $5.95 (spiral-bound).*

The Boys and Girls Book About One-Parent Families, *by Richard A. Gardner, M.D.; illustrated by Alfred Lowenheim. New York, N.Y., G.P. Putnam's Sons, 1978, 236 pp., $8.95.*

Handbook of Psychotherapy and Behavior Change: An Empirical Analysis, 2nd ed., *edited by Sol L. Garfield and Allen E. Bergin. New York, N.Y., John Wiley & Sons, 1978, 966 pp., no price listed.*

Feelings: Our Vital Signs, *by Willard Gaylin, M.D. New York, N.Y., Harper & Row, 1979, 242 pp., $10.00.*

How to Find Out in Psychiatry: A Guide to Sources of Mental Health Information, *by Bette Greenberg. New York, N.Y., Pergamon Press, 1978, unnumbered pages, $12.00.*

Behavior and Neurology of Lizards: An Interdisciplinary Colloquium, *edited by Neil Greenberg and Paul D. MacLean. Rockville, Md., National Institute of Mental Health, 1978, 339 pp., no price listed (paper).*

Self-Involvement in the Middle East Conflict, *by the Group for the Advancement of Psychiatry Committee on International Relations. New York, N.Y., GAP, 1978, 205 pp., $6.00 (paper).*

Massachusetts General Hospital Handbook of General Hospital Psychiatry, *edited by Thomas P. Hackett, M.D., and Ned H. Cassem, M.D. St. Louis, C.V. Mosby Co., 1978, 575 pp., $14.95 (paper).*

The Essential Montessori, *by Elizabeth G. Hainstock. New York, N.Y., New American Library, 1978, 145 pp., $1.95 (paper).*

Autobiography of Dying, *by Archie J. Hanlan; edited by Muriel E. Nelson. Garden City, N.Y., Doubleday & Co., 1979, 193 pp., $8.95.*

The Sleeping Pill, *by Ernest Hartmann, M.D. New Haven, Conn., Yale University Press, 1978, 303 pp., $12.50.*

The Empty Face, *by Katharina Havekamp. New York, N.Y., Richard Marek Publishers, 1978, 220 pp., $8.95.*

Adults and Their Parents in Family Therapy: A New Direction in Treatment, *by Lee Headley. New York, N.Y., Plenum Press, 1977, 190 pp., $14.95.*

International Dental Care Delivery Systems: Issues in Dental Health Policies, *edited by John I. Ingle and Patricia Blair. Cambridge, Mass., Ballinger Publishing Co. (J.B. Lippincott Co.), 1978, 251 pp., $16.50.*

Family Policy: Government and Families in Fourteen Countries, *edited by Sheila B. Kamerman and Alfred J. Kahn. New York, N.Y., Columbia University Press, 1978, 515 pp., $25.00; $10.00 (paper).*

Psychotherapeutics in Medicine, *edited by Toksoz B. Karasu, M.D., and Robert I. Steinmuller, M.D., with Betty Meltzer, M.A. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1978, 338 pp., $24.50.*

Why Not Say It Clearly: A Guide to Scientific Writing, *by Lester S. King, M.D. Boston, Mass., Little, Brown and Co., 1978, 183 pp., $5.95 (paper).*

Mental Health Advocacy: An Emerging Force in Consumers' Rights, *edited by Louis E. Kopolow, M.D., and Helene Bloom. Rockville, Md., National Institute of Mental Health, 1977, 101 pp., no price listed (paper).*

Self-Administration of Abused Substances: Methods for Study. NIDA Research Monograph 20, *edited by Norman A. Krasnegor, Ph.D. Rockville, Md., National Institute on Drug Abuse, 1978, 246 pp., single copies free.*

Existential Social Work, *by Donald F. Krill. New York, N.Y., Free Press (Macmillan Publishing Co.), 1978, 205 pp., $13.95.*

Psychiatric Aspects of Opiate Dependence, *by Albert A. Kurland West Palm Beach, Fla., CRC Press, 1978, 257 pp., $59.95.*

The Culture of Narcissism: American Life in an Age of Diminishing Expectations, *by Christopher Lasch. New York, N.Y., W.W. Norton & Co., 1978, 260 pp., $11.95.*

Childhood and Cultural Despair: A Theme and Variations in Seventeenth-Century Literature, *by Leah Sinanoglou Marcus. Pittsburgh, Pa., University of Pittsburgh Press, 1978, 295 pp., $11.95.*

Drug Intervention Index 1978/79, *by Eric W. Martin, Ph.C., M.S., Ph.D. Philadelphia, Pa., J.B. Lippincott Co., 1978, 312 pp., $10.95 (paper).*

Childhood Psychopharmacology: Current Concepts, *by J. Mendlewicz and H.M. van Praag. Basel, Switzerland, S. Karger, 1978, 96 pp., $25.50 (paper).*

Dreaming and Memory: A New Information-Processing Model, *by Stanley R. Palombo, M.D. New York, N.Y., Basic Books, 1978, 227 pp., $15.00.*

Predicting Dangerousness: The Social Construction of Psychiatric Reality, *by Stephen J. Pfohl. Lexington, Mass., Lexington Books (D.C. Heath and Co.), 1978, 241 pp., $19.50.*

Group Psychotherapy: Theory and Practice, 2nd ed., *by Hugh Mullan and Max Rosenbaum. New York, N.Y., Free Press (Macmillan Publishing Co.), 1978, 409 pp., $17.95.*

Sex Education for the Health Professional: A Curriculum Guide, *edited by Norman Rosenzweig, M.D., and F. Paul Pearsall, Ph.D. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1978, 327 pp., no price listed.*

"I Want to Change, but I Don't Know How," *by Tom Rusk, M.D., and Randy Read, M.D. Santa Barbara, Calif., Ross-Erikson (Blue Pacific Books), 1978, 336 pp., $5.95 (paper).*

The Child and Death, *edited by Olle Jane Z. Sahler, M.D. St. Louis, Mo., C.V. Mosby Co., 1978, 288 pp., $9.95 (paper).*

The Personal Sphere Model, *by Raoul A. Schmiedeck, M.D., Ph.D. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1978, 199 pp., $18.50.*

The Mentally Ill in Community-Based Sheltered Care: A Study of Community Care and Social Integration, *by Steven P. Segal and Uri Aviram. New York, N.Y., Wiley-Interscience (John Wiley & Sons), 1978, 312 pp., no price listed.*

The Power of Human Imagination: New Methods in Psychotherapy, *edited by Jerome L. Singer and Kenneth S. Pope. New York, N.Y., Plenum Press, 1978, 394 pp., $25.00.*

A Piaget Primer: How a Child Thinks, *by Dorothy G. Singer and Tracey A. Revenson. New York, N.Y., International Universities Press, 1978, 143 pp., $15.00; Plume Book (New American Library), 1978, 143 pp., $3.95 (paper).*

The Death of the Asylum: A Critical Study of State Hospital Management, Services, and Care, *by John A. Talbott, M.D. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1978, 174 pp., $22.50.*

There Is a Rainbow Behind Every Dark Cloud. *Tiburon, Calif., Center for Attitudinal Healing, 1978, 96 pp., $5.95 (paper).*

Psychology of Crime and Criminal Justice, *edited by Hans Toch. New York, N.Y., Holt, Rinehart and Winston, 1979, 487 pp., $14.95.*

Awakening Intuition, *by Frances E. Vaughan. Garden City, N.Y., Anchor Books (Anchor Press/Doubleday), 1979, 218 pp., $3.50 (paper).*

The Hyperactive Child and the Learning Disabled Child: A Handbook for Parents, *by Paul H. Wender, M.D., and Esther H. Wender, M.D. New York, N.Y., Crown Publishers, 1978, 134 pp., $6.95.*

The Icarus Complex: Studies of an Alleged Relationship Between Fascination for Fire, Enuresis, High Ambition, and Ascensionism, *by Nils Wiklund. Lund, Sweden, Bokforlaget Doxa, 1978, 253 pp., 80 Swiss krona (paper).*

Cognitive Styles in Personal and Cultural Adaptation, *by Herman A. Witkin. Worcester, Mass., Clark University Press, 1978, 68 pp., no price listed (paper).*

Mental Retardation and Developmental Disabilities: An Annual Review, Vol. 10, *edited by Joseph Wortis, M.D. New York, N.Y., Brunner/Mazel, 1978, 258 pp., $17.50.*

Getting Together: A Guide to Sexual Enrichment for Couples, *by Drs. Leon and Shirley Zussman, with Jeremy Brecher. New York, N.Y., William Morrow and Co., 1979, 235 pp., $7.95.*

OFFICIAL ACTIONS

1979 Annual Meeting: Registration and Related Information

The 132nd annual meeting of the American Psychiatric Association will be held in Chicago, Ill., May 12–18, 1979; the theme of this meeting is "The Scientific and Humanistic Future of Psychiatry." The information here is what was available as of mid-January 1979. The official program book containing the scientific, business, and social program will be distributed in Chicago at the time of registration. (The preliminary scientific program is printed in the March 2 issue of Psychiatric News.)

REGISTRATION

All registration will take place in the Conrad Hilton Hotel. The registration desk will be open:

Saturday, May 12—1 p.m. to 5 p.m.
Sunday through Thursday, May 13–17—8 a.m. to 5 p.m.
Friday, May 18—8 a.m. to 10 a.m.

A registration fee of $15 per day, or $60 for all seven days, is required from all nonmembers with the exception of 1) invited nonmember program participants and their immediate families, 2) APA members and their immediate family members living in the same household. Associate members, members-in-training, and their immediate family members living in the same household, and 3) foreign visitors outside of the U.S. and Canada. The fee covers admission to the sessions and includes a badge and a copy of the official program. Badges are required for all sessions (including the opening session) and for the exhibit area. However, a badge does not cover admission to the business session of the Association, which is open only to voting members.

A $20 registration fee is required of military personnel on active duty.

A $10 registration fee is required of the spouses and dependents of all nonmember registrants (excluding invited nonmember program participants).

A $15 registration fee is required of medical students, interns, residents, chaplains, nursing students, and students in the mental health professions. Students must present a letter from their school or training facility instructor certifying their status.

The *Scientific Proceedings in Summary Form* for the 1979 annual meeting will be available throughout the week in the Conrad Hilton Hotel.

KEY LOCATIONS

All APA staff offices will be located on the fifth floor of the Conrad Hilton Hotel:

Administrative Office	Rooms 418, 419
Meetings Management	Continental Room, Main Lobby
Program Committee	Room 415
Journal Office	Room 412
Press Offices	Rooms 413, 414
Local Arrangements and Spouse Hospitality Center	Normandie Lounge
Registration	North Hall, Lower Level
Information and Message Center	Lower Level Lobby
Membership Resource Center	East Hall, Lower Level

INFORMATION AND MESSAGE CENTER

This center will be located in the Conrad Hilton Hotel and will be manned during registration hours. Self-service message racks will be available for pick-up, deposit, and exchange of messages at all times, even when the message center is closed. Extremely urgent messages will receive the personal attention of APA staff members. All registrants are requested to check the message center periodically. There will also be satellite information centers in the Marriott, Palmer House, and Hyatt Regency Hotels.

JOURNAL OFFICE

The office of the *American Journal of Psychiatry* will be in Room 412 of the Conrad Hilton Hotel. The office will be open from 8:00 a.m. to 5:00 p.m., Monday through Thursday, and on Friday until 12:00 noon. Staff will be available to discuss any questions that authors who are presenting papers at the meeting may have. They particularly welcome the opportunity to confer with authors about tables and figures to be included in the papers.

The *Journal* has first refusal rights for all numbered papers. Permission must be secured from the Editor, John C. Nemiah, M.D., before a paper may be submitted elsewhere. The *Journal* will accept for review only papers that are in final form and conform to the requirements listed in "Information for Contributors." The *Journal* does not hold first refusal rights for new research papers and papers presented at morning workshops and evening panels; however, authors may submit them for publication if they wish.

It is the policy of the *Journal* to publish only original material that has not been published elsewhere in any form and that is not being considered for publication elsewhere.

CONVOCATION

The convocation will take place Monday, May 14, at 7:30 p.m. in the International Ballroom of the Conrad Hilton Hotel. The William C. Menninger Memorial Lecture will be given at the convocation by Dr. Robert Jastrow.

SPOUSE HOSPITALITY CENTER

The Local Arrangements Committee will host a Spouse Hospitality Lounge in the Normandie Lounge of the Conrad Hilton Hotel. Information on all leisure-time activities will be available Sunday through Friday in this area. All tours will depart from the 8th Street entrance of the Conrad Hilton Hotel.

PLACEMENT SERVICE

There will be a register available in the Membership Resource Center for prospective employers and for those seeking positions.

SHUTTLE BUS SERVICE

Bus service will be available among the hotels. Route maps, hours of operation, and fare information will be available at the Information and Message Center.

ARTS AND CULTURE

A special performance of the award-winning musical "Annie" will be presented at the Arie Crown Theater on Tuesday, May 15, at 8:00 p.m. Complimentary reserved tickets will be available for APA members (2 per registrant) at a special ticket booth on a first-come first-serve basis. Complimentary shuttle bus service will be provided to and from the theater. A symposium on "Annie" will be held on Wednesday, May 16, from 2:00 p.m. to 5:00 p.m. in the Red Lacquer Room of the Palmer House.

CONTINUING EDUCATION CREDIT

The official annual meeting program, with a few exceptions, meets the criteria for continuing medical education Category I credit. One hour of credit may be claimed for each hour of participation. As an organization accredited for continuing medical education activities, the American Psychiatric Association certifies that the continuing medical education activities designated Category I meet the criteria for Category I for the Physician's Recognition Award of the American Medical Association on an hour-for-hour basis. Continuing medical education sessions are open to all meeting registrants. Check the tentative program schedule in the February 2 issue of *Psychiatric News* for sessions that offer Category II credit.

All program activities are designated either Category I or Category II; these include courses, symposia, paper sessions, special lectures, video, films, panels, and issue workshops. Seven two-day courses on Saturday, May 12, and Sunday, May 13, and five symposia on Sunday, May 13, may also be taken for additional Category I credit. Half-day courses on Tuesday and Friday will be held from 10:15 a.m. to 2:15 p.m. and on Monday from 2:00 p.m. to 6:00 p.m. Full-day courses will be offered on Wednesday and Thursday from 10:15 a.m. to 5:15 p.m.; one full-day course on Wednesday will be offered from 10:15 a.m. to 6:15 p.m. All full-day courses have a 1-hour break for lunch. Preregistration coupons for courses (March 23 deadline) and complete course descriptions can be found in various January-February-March issues of *Psychiatric News*.

MORNING WORKSHOPS AND EVENING PANELS

Issue workshops will be held on Tuesday, Wednesday, and Thursday from 10:30 a.m. to 12:00 noon. All issue workshops offer Category II credit with the exception of those starred in the official program, which offer Category I credit. Evening panels will be held on Tuesday, Wednesday, and Thursday from 8:00 p.m. to 10:00 p.m. All evening panels offer Category I credit. All workshops and panels will be held at various hotels.

VIDEOTAPE PROGRAM

Videotape sessions will be shown on wide-screen closed-circuit color TV in the Conrad Hilton, Grand Ballroom, 2nd floor, from 2:00 p.m. to 5:00 p.m. on Monday, Wednesday, and Thursday, and from 8:00 a.m. to 9:45 a.m. on Tuesday and Friday.

FILMS

All films will be shown in the Palmer House, State Ballroom, 4th floor. Noon film features can be seen Monday through Friday and evening film features can be seen Tuesday and Wednesday at 8:00 p.m.

SPECIAL LECTURES

APA will sponsor 30 special lectures this year. Among the named lectures will be the Adolf Meyer Lecture (Tuesday, 1:30 p.m. to 3:00 p.m.), the Benjamin Rush Lecture (Wednesday, 10:30 a.m. to 12:00 noon), the Manfred S. Guttmacher Lecture (Wednesday, 10:30 a.m. to 12:00 noon), the Solomon Carter Fuller Lecture (Wednesday, 2:00 p.m. to 3:30 p.m.), the Seymour Vestermark Lecture (Wednesday, 3:45 p.m. to 5:00 p.m.), the Simon Bolivar Lecture (Thursday, 10:30 a.m. to 12:00 noon), and the APA Founders Award Lecture (Thursday, 10:30 to 12:00 noon). Among the guest lecturers will be the Reverend Jesse L. Jackson (Monday, 2:00 p.m. to 5:00 p.m.), Professor Archibald Cox (Monday, 3:45 p.m. to 5:00 p.m.), Congressman Paul Rogers of Florida (Tuesday, 10:30 a.m. to 12:00 noon). Included in the 30 lectures will be an APA Lecture Series, featuring 13 distinguished psychiatrists and nonpsychiatrist physicians.

NEW RESEARCH

These sessions will be held on Tuesday through Friday from 8:00 a.m. to 9:45 a.m. in the Hyatt Regency, Ballrooms A, B, and C. All 12 sessions will offer Category II credit. Program leaflets containing all details will be available at the registration desk.

EXHIBITS

Scientific and technical exhibits will be on display in the Conrad Hilton Hotel Monday through Wednesday, 9:00 a.m. to 4:30 p.m., and Thursday, 9 a.m. to 2 p.m.

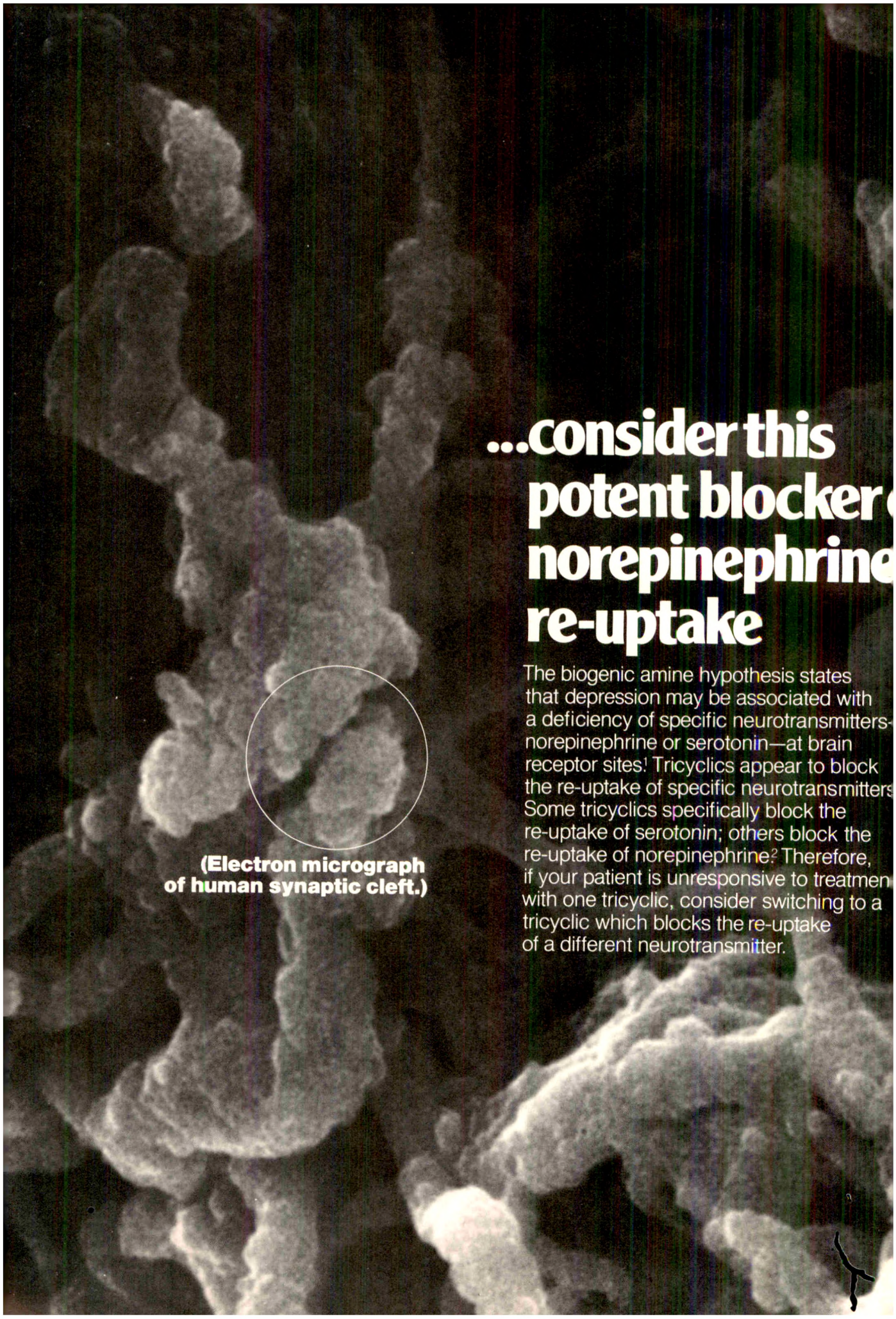
LUNCHEON IN HONOR OF FAMILIES OF APA OFFICERS

A luncheon will be held in honor of the families of APA officers on Tuesday, May 15, from 11:30 a.m. to 1:30 p.m. at the Chicago Public Library and Cultural Center. Tickets for the luncheon are $10 per person and may be purchased at the Leisure Time Activities Ticket Sales Desk.

APA DINNER DANCE

A dinner dance featuring two bands, one before and on during dinner, will be held on Wednesday, May 16, at 7:3 p.m. in the Grand Ballroom of the Conrad Hilton Hote Tickets are $25 per couple and may be purchased at the Le sure Time Activities Ticket Sales Desk or in advance throug a leisure time activities coupon published in a brochur mailed to each member.


When your patient is clinically depressed...
"...everything I saw was negative."



...consider this potent blocker of norepinephrine re-uptake

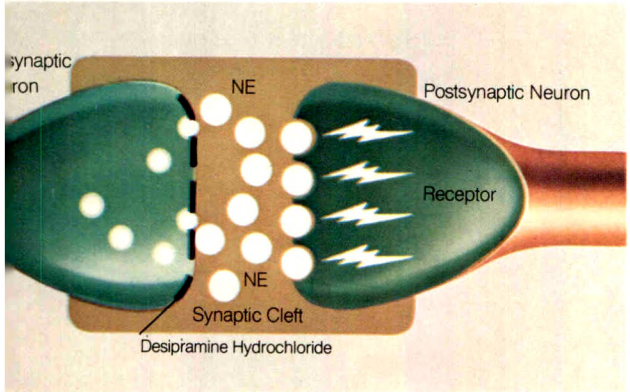
The biogenic amine hypothesis states that depression may be associated with a deficiency of specific neurotransmitters-norepinephrine or serotonin—at brain receptor sites[1] Tricyclics appear to block the re-uptake of specific neurotransmitters. Some tricyclics specifically block the re-uptake of serotonin; others block the re-uptake of norepinephrine.[2] Therefore, if your patient is unresponsive to treatment with one tricyclic, consider switching to a tricyclic which blocks the re-uptake of a different neurotransmitter.

(Electron micrograph of human synaptic cleft.)

Norpramin®
desipramine hydrochloride (tablets NF)
5, 50, 75, 100, 150 mg.

tent blocker norepinephrine re-uptake

cording to current theory[1,3] some yclic antidepressants may fail in certain es of depression because they are not ficiently potent blockers of norepi- hrine re-uptake. Laboratory studies ve shown that desipramine is the most ent inhibitor of norepinephrine uptake.[4] Norpramin may work in some ents when other tricyclic antidepres- ts do not.



onsider it now, onsider it next.

Less anticholinergic activity

(See Warnings section.)

Studies in animals[5] and in normal human subjects[6,7] have shown that desipramine has significantly less anticholinergic activity than amitriptyline or doxepin. The single bond side chain is associated with lower anticholinergic activity than these other leading antidepressants. This may mean:

- less dry mouth
- less blurred vision
- less urinary retention

Helps patients remain calm but active

Norpramin does not usually inhibit normal activity although patients should be cautioned as to driving or operating machinery if drowsiness occurs.

In addition...

Begins to improve sleep patterns within one week in some patients[8]

- Norpramin helps relieve the sleep disturbances that often accompany depression.
- As the depression is relieved, the difficulty in falling asleep, restlessness, and early morning awakening diminish.

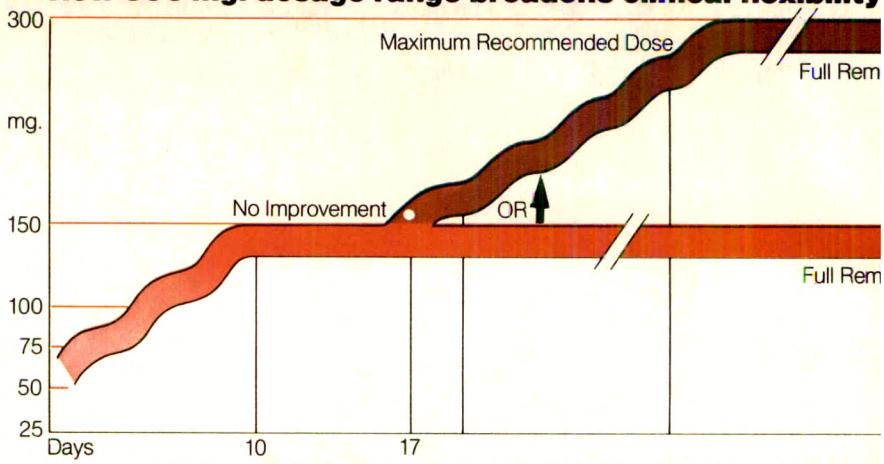
References:

1. Maas, J.W.: Biogenic amines and depression. Biochemical and pharmacological separation of two types of depression. Arch. Gen. Psychiat. 32:1357-1361, 1975. **2.** Mendels, J., Stern, S., and Frazer, A.: Dis. Nerv. Syst. 37:3, 1976. **3.** Davis, J.M.: Central biogenic amines and theories of depression and mania. In, Phenomenology and Treatment of Depression, W.E. Fann, I. Karacan, A.D. Pokorny, and R.L. Williams, Ed., New York, Spectrum Publications, Inc., 1977, pp. 17-32. **4.** Ross, S.B. and Renyi, A.L.: Tricyclic antidepressant agents. 1. Comparison of the inhibition of the uptake of ^{3}H-noradrenaline and ^{14}C-5-hydroxytryptamine in slices and crude synaptosome preparations of the midbrain-hypothalamus region of the rat brain. Acta Pharmacol. (Kobenhavn) 36:382-394, 1975. **5.** Snyder, S.H. and Yamamura, H.I.: Antidepressants and the muscarinic acetylcholine receptor. Arch. Gen. Psychiat. 34:236-239, 1977. **6.** Blackwell, B., Stefopoulos, A., Enders, P., Kuzma, R., and Adolphe, A.: The anticholinergic activity of two tricyclic antidepressants. Amer. J. Psychiat., 135:722-724, June 1978. **7.** Peterson, G.R., Hostetler, R.M., Blackwell, B., Kuzma, R., and Adolphe, A.B.: Effects of the tricyclic antidepressants desipramine and doxepin on anticholinergic and CNS activity in non-depressed volunteers. Neuroscience Abstr. 3:447, 1977. **8.** Zung, W.W.K.: The pharmacology of disordered sleep. A laboratory approach. J.A.M.A. 211:1532-1534, 1970.

(For prescribing information see next page.)

Merrell

Now 300 mg. dosage range broadens clinical flexibility



A maximum dosage of 300 mg. a day, as a single or divided dose, may now be prescribe Norpramin. Since inadequate dosage is thought to be one of the major causes of treatme failure, the new therapeutic range should prove clinically helpful. Plasma desipramine measurement would constitute the optimal guide to dosage monitoring.

Norpramin®
(desipramine hydrochloride tablets NF)

AVAILABLE ONLY ON PRESCRIPTION

Brief Summary

MECHANISM OF ACTION: Available evidence suggests that many depressions have a biochemical basis in the form of a relative deficiency of neurotransmitters such as norepinephrine and serotonin. Norepinephrine deficiency may be associated with relatively low urinary 3-methoxy-4-hydroxyphenyl glycol (MHPG) levels, while serotonin deficiencies may be associated with low spinal fluid levels of 5-hydroxyindolacetic acid.

While the precise mechanism of action of the tricyclic antidepressants is unknown, a leading theory suggests that they restore normal levels of neurotransmitters by blocking the re-uptake of these substances from the synapse in the central nervous system.

Evidence indicates that the secondary amine tricyclic antidepressants, including Norpramin, may have greater activity in blocking the re-uptake of norepinephrine. Tertiary amine tricyclic antidepressants, such as amitriptyline, may have greater effect on serotonin re-uptake.

Norpramin (desipramine hydrochloride) is not a monoamine oxidase (MAO) inhibitor and does not act primarily as a central nervous system stimulant. It has been found in some studies to have a more rapid onset of action than imipramine. Earliest therapeutic effects may occasionally be seen in 2 to 5 days, but full treatment benefit usually requires 2 to 3 weeks to obtain.

INDICATIONS: Norpramin (desipramine hydrochloride) is indicated for relief of symptoms in various depressive syndromes, especially endogenous depression.

CONTRAINDICATIONS: Desipramine hydrochloride should not be given in conjunction with, or within 2 weeks of, treatment with an MAO inhibitor drug; hyperpyretic crises, severe convulsions, and death have occurred in patients taking MAO inhibitors and tricyclic antidepressants. When Norpramin (desipramine hydrochloride) is substituted for an MAO inhibitor, at least 2 weeks should elapse between treatments. Norpramin should then be started cautiously and should be increased gradually.

The drug is contraindicated in the acute recovery period following myocardial infarction. It should not be used in those who have shown prior hypersensitivity to the drug. Cross sensitivity between this and other dibenzazepines is a possibility.

WARNINGS: 1. Extreme caution should be used when this drug is given in the following situations: a. In patients with cardiovascular disease, because of the possibility of conduction defects, arrhythmias, tachycardias, strokes, and acute myocardial infarction. b. In patients with a history of urinary retention or glaucoma, because of the anticholinergic properties of the drug. c. In patients with thyroid disease or those taking thyroid medication, because of the possibility of cardiovascular toxicity, including arrhythmias. d. In patients with a history of seizure disorder, because this drug has been shown to lower the seizure threshold. **2.** This drug is capable of blocking the antihypertensive effect of guanethidine and similarly acting compounds. **3.** USE IN PREGNANCY: Safe use of desipramine hydrochloride during pregnancy and lactation has not been established; therefore, if it is to be given to pregnant patients, nursing mothers, or women of childbearing potential, the possible benefits must be weighed against the possible hazards to mother and child. Animal reproductive studies have been inconclusive. **4.** USE IN CHILDREN: Norpramin (desipramine hydrochloride) is not recommended for use in children since safety and effectiveness in the pediatric age group have not been established. **5.** The patient should be cautioned that this drug may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery. **6.** In patients who may use alcohol excessively, it should be borne in mind that the potentiation may increase the danger inherent in any suicide attempt or overdosage.

PRECAUTIONS: 1. It is important that this drug be dispensed in the least possible quantities to depressed outpatients, since suicide has been accomplished with this class of drug. Ordinary prudence requires that children not have access to this drug or to potent drugs of any kind; if possible this drug should be dispensed in containers with child-resistant safety closures. Storage of this drug in the home must be supervised responsibly. **2.** If serious adverse effects occur, dosage should be reduced or treatment should be altered. **3.** Norpramin (desipramine hydrochloride) therapy in patients with manic-depressive illness may induce a hypomanic state after the depressive phase terminates. **4.** The drug may cause exacerbation of psychosis in schizophrenic patients. **5.** Close supervision and careful adjustment of dosage are required when this drug is given concomitantly with anticholinergic or sympathomimetic drugs. **6.** Patients should be warned that while taking this drug their response to alcoholic beverages may be exaggerated. **7.** Clinical experience in the concurrent administration of ECT and antidepressant drugs is limited. Thus, if such treatment is essential, the possibility of increased risk relative to benefits should be considered. **8.** The sedative effects of Norpramin and benzodiazepines (*e.g.*, chlordiazepoxide or diazepam) are additive. Both the sedative and anticholinergic effects of the major tranquilizers are additive to those of Norpramin. **9.** This drug should be discontinued as soon as possible prior to elective surgery because of the possible cardiovascular effects. Hypertensive episodes have been observed during surgery in patients taking desipramine hydrochloride. **10.** Both elevation and lowering of blood sugar levels have been reported. **11.** Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during therapy; the drug should be discontinued if there is evidence of pathologic neutrophil depression.

ADVERSE REACTIONS: Note: Included in the following listing are a few adverse reactions that have not been reported with this specific drug. However, the pharmacologic similarities among the tricyclic antidepressant drugs require that each of the reactions be considered when Norpramin (desipramine hydrochloride) is given.

Cardiovascular: hypotension, hypertension, tachycardia, palpitation, arrhythmias, heart block, myocardial infarction, stroke.

Psychiatric: confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia and nightmares; hypomania; exacerbation of psychosis.

Neurologic: numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures; alteration in EEG patterns; tinnitus.

Anticholinergic: dry mouth, and rarely associated sublingual adenitis; blurred vision, disturbance of accommodation, mydriasis, increased intraocular pressure; constipation, paralytic ileus; urinary retention, delayed micturition, dilatation of urinary tract.

Allergic: skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight), edema (of face and tongue or general), drug fever, cross sensitivity with other tricyclic drugs.

Hematologic: bone marrow depressions including agranulocytosis, eosinophilia, purpura, thrombocytopenia.

Gastrointestinal: anorexia, nausea and vomiting, epigastric distress, peculiar taste, abdominal cramps, diarrhea, stomatitis, black tongue.

Endocrine: gynecomastia in the male, breast enla ment and galactorrhea in the female; increased decreased libido, impotence, testicular swelling; el tion or depression of blood sugar levels.

Other: jaundice (simulating obstructive), altered l function; weight gain or loss; perspiration, flush urinary frequency, nocturia; parotid swelling; dro ness, dizziness, weakness and fatigue, headache; pecia.

Withdrawal Symptoms: Though not indicative of ad tion, abrupt cessation of treatment after prolor therapy may produce nausea, headache, and mala

DOSAGE AND ADMINISTRATION: Not recommended for in children. Lower dosages are recommended for elc patients and adolescents. Lower dosages are recommended for outpatients compared to hospital patients, who are closely supervised. Dosage shoul initiated at a low level and increased accordin clinical response and any evidence of intolera Following remission, maintenance medication ma required for a period of time and should be at the lo dose that will maintain remission.

Usual Adult Dose: The usual adult dose is 100 to 20 per day. In more severely ill patients, dosage ma further increased gradually to 300 mg./day if neces Dosages above 300 mg./day are not recommended

Dosage should be initiated at a lower level increased according to tolerance and clinical resp

Treatment of patients requiring as much as 300 should generally be initiated in hospitals, where re visits by the physician, skilled nursing care, and quent electrocardiograms (ECG's) are available.

The best available evidence of impending to from very high doses of Norpramin is prolongation QRS or QT intervals on the ECG. Prolongation of th interval is also significant, but less closely corre with plasma levels. Clinical symptoms of intolera especially drowsiness, dizziness, and postural hyp sion, should also alert the physician to the nee reduction in dosage. Plasma desipramine measure would constitute the optimal guide to dosage mo ing.

Initial therapy may be administered in divided or a single daily dose.

Maintenance therapy may be given on a once- schedule for patient convenience and compliance.

Adolescent and Geriatric Dose: The usual adolescen geriatric dose is 25 to 100 mg. daily.

Dosage should be initiated at a lower leve increased according to tolerance and clinical resp to a usual maximum of 100 mg. daily. In more sever patients, dosage may be further increased to 150 day. Doses above 150 mg./day are not recommend these age groups.

Initial therapy may be administered in divided or a single daily dose.

Maintenance therapy may be given on a once- schedule for patient convenience and compliance.

OVERDOSAGE: See prescribing information for a di sion of symptoms and treatment of overdose.

Product Information as of April, 1978

Merrell

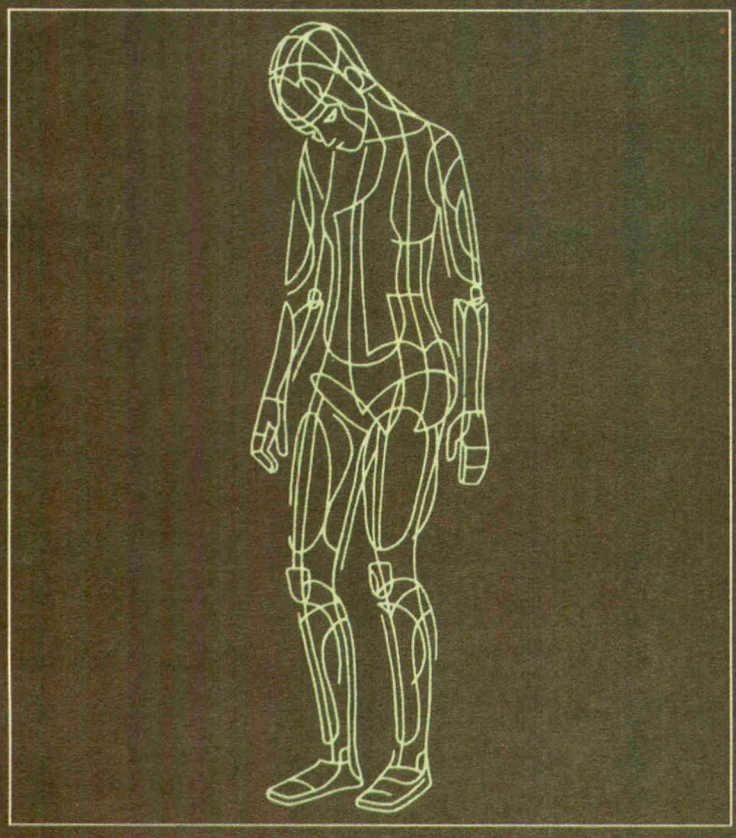
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Provides the well established antidepressant effect of amitriptyline
with the proven antianxiety effect of Librium® (chlordiazepoxide HCl)

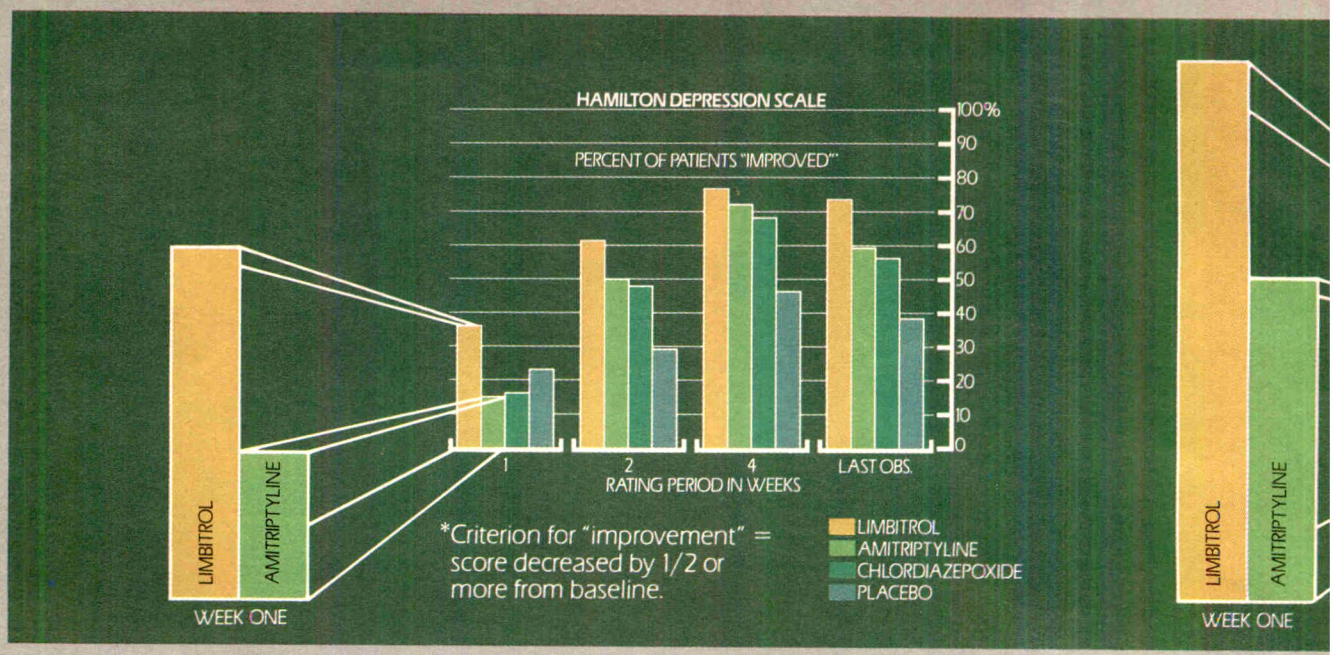


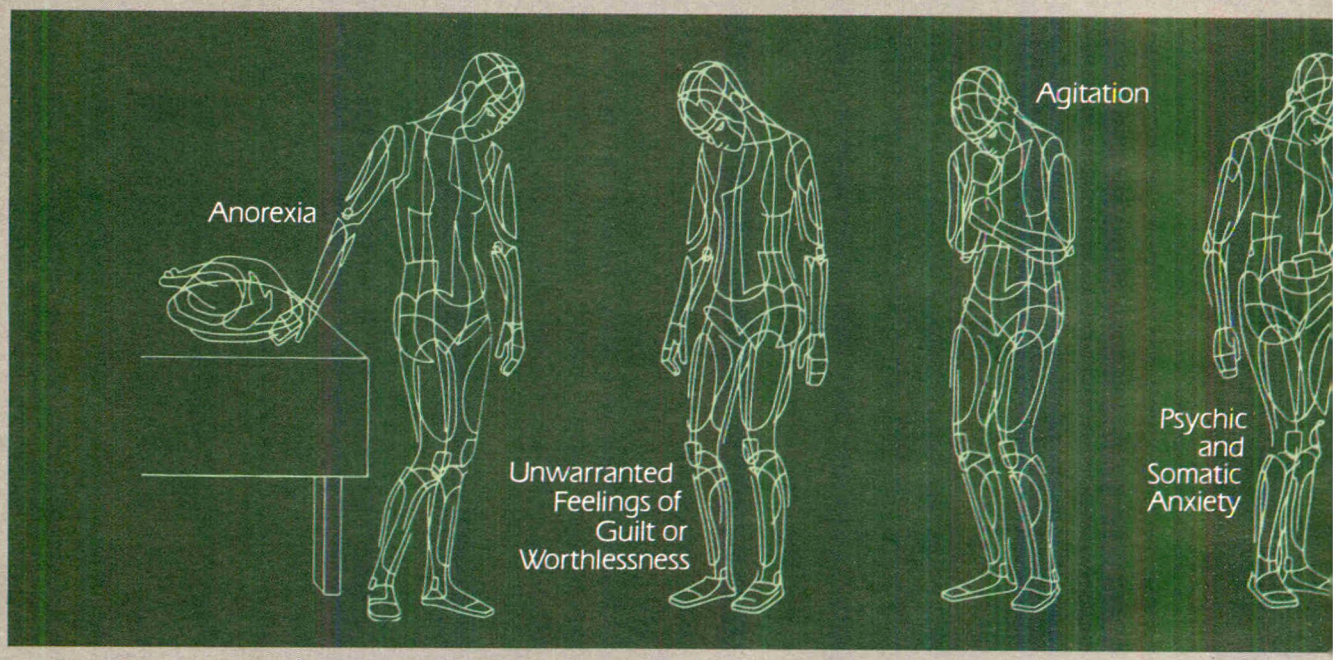
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Limbitrol achieved these superior results...

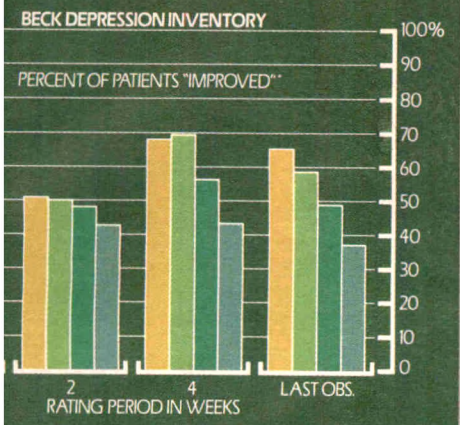
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*Criterion for "improvement" = score decreased by 1/2 or more from baseline.
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AMITRIPTYLINE
CHLORDIAZEPOXIDE
PLACEBO
LIMBITROL
AMITRIPTYLINE
WEEK ONE


Anorexia
Unwarranted Feelings of Guilt or Worthlessness
Agitation
Psychic and Somatic Anxiety





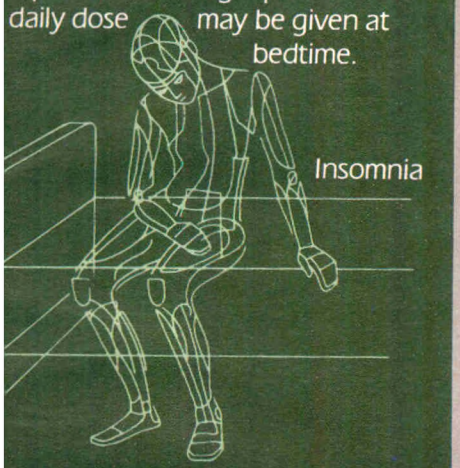
ion for "improvement" =
decreased by 1/3 or
from baseline.

Greater patient improvement

Within the first week—

twice as many patients achieved significant relief—sooner than with amitriptyline alone

omnia, a major target symptom, can
relieved sooner by taking advantage
he sedative effect of Limbitrol. For
he patients, the larger part or all of
daily dose may be given at bedtime.



Better relief of symptoms

Within the first week—

symptom severity was reduced to a greater extent than with amitriptyline alone

As measured by the Hamilton Depression Scale

- insomnia
- psychic anxiety
- somatic anxiety
- agitation
- suicidal ideation
- worthlessness

As measured by the Beck Depression Inventory

- pessimism
- dissatisfaction
- guilt
- social withdrawal
- anorexia

imbitrol®

ets 10-25 each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt)

ets 5-12.5 each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)

rapid relief of
derate to severe
pression and anxiety

Greater patient acceptance

- Despite a higher incidence of drowsiness, there was a lower dropout rate due to side effects with Limbitrol than with amitriptyline

(Limbitrol 4%, amitriptyline 13%, chlordiazepoxide 1%)

- The dropout rate due to therapeutic failure was lower with Limbitrol than with amitriptyline

(Limbitrol 2%, amitriptyline 6%, chlordiazepoxide 12%)

Please see summary of complete product information on last page of this advertisement.

ROCHE

How to Initiate and Maintain Therapy

Select dosage strength appropriate for each patient

- Limbitrol 10-25 is recommended for most patients
- Limbitrol 5-12.5 may be indicated for those who do not tolerate higher doses and for elderly patients

Specify daily dosage based on symptom severity

- An initial dosage of three tablets is recommended
- Dosage may be increased to six tablets or decreased to two tablets daily as necessary
- Once a satisfactory response is obtained, evaluate response, and if necessary, reduce dosage to smallest amount needed to maintain remission

Utilize dosage options to best accommodate individual patient needs

- T.I.D. or Q.I.D., familiar regimens best suited for patients who tolerate medication without undue drowsiness
- Two tablets one hour before bedtime and one tablet midday may minimize daytime drowsiness and help relieve a common target symptom—insomnia
- Entire dosage h.s. to take maximum advantage of the sedative effect

How to make each patient an informed patient

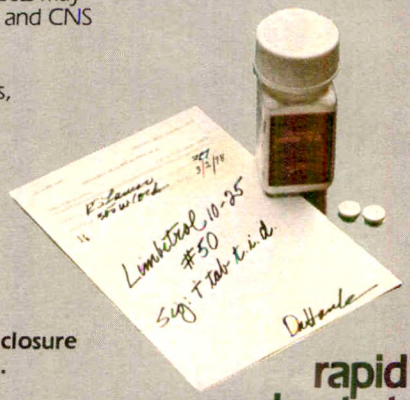
1. Discuss with patients the probability that they will experience drowsiness, especially during the first week.
2. Reassure your patients that drowsiness is one indication that the medication is working and that it may help alleviate their insomnia.
3. Encourage patients to report if drowsiness becomes troublesome so that, if necessary, dosage schedule can be adjusted or the symptom treated.
4. Caution patients about the combined effects with alcohol or other CNS depressants. Let them know that the additive effects may produce a harmful level of sedation and CNS depression.
5. Caution patients about activities requiring complete mental alertness, such as operating machinery or driving a car.
6. Warn pregnant patients and patients of childbearing age that the safety of Limbitrol in pregnancy has not yet been established.

Please see complete product disclosure for other pertinent information.

Limbitrol should not be used under t following circumstances

1. Hypersensitivity to benzodiazepines or tricyclic antidepressants.
2. Should not be given with an MAO inhibitor. To replace an MAO inhibitor with Limbitrol, discontinue MAO inhibitor for minimum of 14 days before cautiously ini ing Limbitrol therapy.
3. During the acute recovery phase follo ing myocardial infarction.

Limbitro



F
rapid relief
moderate to seve
depression and anxie

LIMBITROL® TABLETS
Tranquilizer–Antidepressant

Before prescribing, please consult complete product information, a summary of which follows:
Indications: Relief of moderate to severe depression associated with moderate to severe anxiety.
Contraindications: Known hypersensitivity to benzodiazepines or tricyclic antidepressants. Do not use with monoamine oxidase (MAO) inhibitors or within 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use; then initiate cautiously, gradually increasing dosage until optimal response is achieved. Contraindicated during acute recovery phase following myocardial infarction.
Warnings: Use with great care in patients with history of urinary retention or angle-closure glaucoma. Severe constipation may occur in patients taking tricyclic antidepressants and anticholinergic-type drugs. Closely supervise cardiovascular patients. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, especially high doses. Myocardial infarction and stroke reported with use of this class of drugs.)

***Usage in Pregnancy:* Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Since physical and psychological dependence to chlordiazepoxide have been reported rarely, use caution in administering Limbitrol to addiction-prone individuals or those who might increase dosage; withdrawal symptoms following discontinuation of either component alone have been reported (nausea, headache and malaise for amitriptyline; symptoms [including convulsions] similar to those of barbiturate withdrawal for chlordiazepoxide).
Precautions: Use with caution in patients with a history of seizures, in hyperthyroid patients or those on thyroid medication, and in patients with impaired renal or hepatic function. Because of the possibility of suicide in depressed patients, do not permit easy access to large quantities in these patients. Caution patients about possible combined effects with alcohol and other CNS depressants and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Periodic liver function tests and blood counts are recommended during prolonged treatment. Amitriptyline component may block action of guanethidine or similar antihypertensives. Concomitant use with other psychotropic drugs has not been evaluated; sedative effects may be additive. Discontinue several days before surgery. Limit concomitant administration of ECT to essential treatment. See Warnings for precautions about pregnancy. Limbitrol should not be taken during the nursing period. Not recommended in children under 12.
In the elderly and debilitated, limit to smallest effective dosage to preclude ataxia, oversedation, confusion or anticholinergic effects.
Adverse Reactions: Most frequently reported are those associated with either component alone: drowsiness, dry mouth, constipation, blurred vision, dizziness and bloating. Less frequently occurring reactions include vivid dreams, impotence, tremor, confusion and nasal congestion. Many depressive symptoms including anorexia, fatigue, weakness, restlessness and lethargy have been reported as side effects of both Limbitrol and amitriptyline. Granulocytopenia, jaundice and hepatic dysfunction have been observed rarely.
This list includes adverse reactions not reported with Limbitrol but requiring consideration because they have been reported with one or both components or closely related drugs.
Cardiovascular: Hypotension, hypertension, tachycardia, palpitations, myocardial infarction, arrhythmias, heart block, stroke.
Psychiatric: Euphoria, apprehension, poor concentration, delusions, hallucinations, hypomania and increased or decreased libido.
Neurologic: Incoordination, ataxia, numbness, tingling and paresthesias of the extremities, extrapyramidal symptoms, syncope, changes in EEG patterns.
Anticholinergics: Disturbance of accommodation, paralytic ileus, urinary retention, dilatation of urin tract.
Allergic: Skin rash, urticaria, photosensitization, edema of face and tongue, pruritus.
Hematologic: Bone marrow depression includin agranulocytosis, eosinophilia, purpura, thrombocytopenia.
Gastrointestinal: Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, black tongue.
Endocrine: Testicular swelling and gynecomasti the male, breast enlargement, galactorrhea and minor menstrual irregularities in the female and elevation and lowering of blood sugar levels.
Other: Headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, jaund alopecia, parotid swelling.
Overdosage: Immediately hospitalize patient suspected of having taken an overdose. Treatm symptomatic and supportive. I.V. administration to 3 mg physostigmine salicylate has been repo to reverse the symptoms of amitriptyline poisoni See complete product information for manifesta and treatment.
Dosage: Individualize according to symptom se and patient response. Reduce to smallest effec dosage when satisfactory response is obtained. Larger portion of daily dose may be taken at be Single h.s. dose may suffice for some patients. dosages are recommended for the elderly.
Limbitrol 10-25, initial dosage of three to four tab daily in divided doses, increased up to six table decreased to two tablets daily as required. Limb 5-12.5, initial dosage of three to four tablets dail divided doses, for patients who do not tolerate doses.
Supplied: Limbitrol 10-25 tablets each contain mg chlordiazepoxide and 25 mg amitriptyline (a hydrochloride salt) and Limbitrol 5-12.5 tablets containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)—bottle 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 50.

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Announcing a Special Issue of **The American Journal of Psychiatry** *on*

DEPRESSION and ALCOHOLISM

This mid-April special issue, included in regular subscriptions to the *American Journal of Psychiatry*, focuses on two topics of intense concern to the psychiatric profession: depression and alcoholism.

Containing 30 original articles, it covers the spectrum of diagnostic considerations, treatment, management and complications, etiology and phenomenology. The emphasis throughout is clinical. While several articles explore new work on alcoholism and on the relationship between depression and alcoholism, current studies on depression dominate this valuable collection.

Partial List of Contributors

David Avery, M.D., Karen Beckman, Robert H. Belmaker, M.D., Walter A. Brown, M.D., Jesse O. Cavenar, Jr., M.D., Alberto DiMascio, Ph.D.,* David L. Dunner, M.D., Larry Ereshefsky, Pharm.D., Ronald R. Fieve, M.D., Eric W. Fine, M.D., Evelyn L. Goldberg, Sc.D., John E. Hamm, M.D., John E. Helzer, M.D., Shirley Y. Hill, Ph.D., Martin H. Keeler, M.D., Gerald L. Klerman, M.D., James H. Kocsis, M.D., Eleftherios Lykouras, M.D., Anthony J. Marsella, Ph.D., Roy J. Mathew, M.D., D.P.M., Demmie Mayfield, M.D., Jerome A. Motto, M.D., Michael R. O'Leary, Ph.D., Irving Philips, M.D., Brigitte A. Prusoff, M.P.H., Timothy M. Rivinus, M.D., Marc A. Schuckit, M.D., Fred A. Steinberg, Ph.D., George J. Warheit, Ph.D., Myrna M. Weissman, Ph.D., Steven J. Wolin, M.D.

*deceased

Partial List of Topics

Diagnosis

Depression simulating organic brain disease — diagnosis and lithium treatment of manic-depressive disorder in the retarded — delineation of an MMPI symptom pattern unique to lithium responders

Treatment, Management

Depression treated with imipramine and ECT — possible cardiovascular effect of lithium — lithium maintenance: factors affecting outcome — efficacy of drugs and psychotherapy in the treatment of acute depressive episodes

Etiology, Phenomenology

Life events at the onset of bipolar affective illness — childhood depression: interpersonal interactions and depressive phenomena — drug abuse among alcoholic women — psychiatric problems in women during alcoholic detoxification — life events, coping, stress, and depressive symptomatology

A special Guest Editorial by Morris A. Lipton, Ph.D., M.D., provides a unifying perspective to this special issue.

Extra copies of the April 1979 Special Issue (the 2nd April issue) will be available at $2.25 (domestic), $2.90 (foreign). *Payment must be included with order.* Order from:

American Psychiatric Association, Circulation Department, 1700 18th Street, NW, Washington, DC 20009

Must the MBD child take a controlled drug in school?



Not if he's taking Cylert® (pemoline) C IV

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Here's how Cylert eliminates this problem:

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- Avoids ups and downs of drug action brought about by multiple daily dosage
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*Copy of the Cylert Monograph available to Physicians on written request.

When not to use medication

Cylert should not be used for (and will not be effective in) simple cases of overactivity in school-age children.

Neither should it be used in the child who exhibits symptoms secondary to environmental factors and/or primary psychiatric disorders, including psychosis.

The physician should rely on a complete history of the child and a thorough description of symptoms from both parents and teacher before postulating a diagnosis of MBD.

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(pemoline)

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Indications: *MINIMAL BRAIN DYSFUNCTION IN CHILDREN*—as adjunctive therapy to other remedial measures (psychological, educational, social).

Special Diagnostic Considerations: The cause of minimal brain dysfunction (MBD) is unknown. Diagnosis of MBD involves the use of medical, psychological, educational, and social tools, since no single diagnostic test is adequate.

MBD is characterized by chronic moderate to severe hyperactivity, short attention span, distractibility, emotional lability, and impulsivity. Nonlocalizing (soft) neurological signs, learning disability, and abnormal EEG may or may not be present. The diagnosis of MBD must be based upon a complete history and evaluation of the child and not solely on the presence of one or more of these characteristics.

Drug treatment is not indicated for all children with MBD. In the primary therapy of MBD, appropriate educational placement is essential and psychosocial intervention is generally necessary. When these measures alone are insufficient, the decision to prescribe stimulant medication will depend upon the physician's assessment of the chronicity and severity of the child's symptoms. Stimulants are not intended for use in the child who exhibits symptoms secondary to environmental factors and/or primary psychiatric disorders, including psychosis.

Contraindications: Cylert (pemoline) is contraindicated in patients with known hypersensitivity or idiosyncrasy to the drug. (See ADVERSE REACTIONS.)

Warnings: Cylert is not recommended for children less than 6 years of age since its safety and efficacy in this age group have not been established.

Sufficient data on the safety and the efficacy of the long-term use of Cylert in children with minimal brain dysfunction are not yet available.

A temporary suppression of the predicted growth rate (weight and/or height gain) has been reported for children receiving long-term stimulant therapy. A definite causal relationship between stimulant drugs and this finding has not been established.

Precautions: Liver function tests should be performed periodically during therapy with Cylert. The drug should be discontinued if abnormalities are revealed and confirmed by follow-up tests. (See ADVERSE REACTIONS regarding reports of abnormal liver function tests and jaundice.)

Cylert should be administered with caution to patients with significantly impaired hepatic or renal function.

The interaction of Cylert with other drugs has not been studied in humans. Patients who are receiving Cylert concurrently with other drugs, especially drugs with CNS activity, should be monitored carefully.

Cylert failed to demonstrate a potential for self-administration in primates. However, the pharmacologic similarity of pemoline to other psychostimulants with known dependence liability suggests that psychological and/or physical dependence might also occur with Cylert. There have been isolated reports of transient psychotic symptoms occurring in adults following the long-term misuse of excessive oral doses of pemoline. Cylert should be given with caution to emotionally unstable patients who may increase the dosage on their own initiative.

Usage during Pregnancy and Lactation: The safety of Cylert (pemoline) for use during pregnancy and lactation has not been established.

Fertility, reproduction, and teratology studies were conducted in laboratory animals. Pemoline, in doses of 18.75 or 37.5 mg./kg./day, had no effect on the fertility of male or female rats. The drug, when given to pregnant rats (from gestation day 15 through weaning) and to rabbits (from gestation days 6-18) at these same dosage levels, produced no teratogenic or embryotoxic effects, and had no effect on the viability of the young at birth. However, increased incidences of stillbirths and cannibalization were observed when pemoline was given to rats at these dosage levels, beginning 14 days prior to conception.

Adverse Reactions: Insomnia is the most frequently reported side effect of Cylert; it usually occurs early in therapy, prior to an optimum therapeutic response. In the majority of cases it is transient in nature or responds to a reduction in dosage.

Anorexia with weight loss may occur during the first weeks of therapy. In the majority of cases it is transient in nature; weight gain usually resumes within three to six months.

Stomach ache, skin rashes, increased irritability, mild depression, nausea, dizziness, headache, drowsiness, and hallucinations have been reported.

Elevations of SGOT, SGPT, and serum LDH have occurred in patients taking Cylert, usually after several months of therapy. These effects appear to be reversible upon withdrawal of the drug, and are thought to be manifestations of a delayed hypersensitivity reaction. There have also been a few reports of jaundice occurring in patients taking Cylert; a causal relationship between the drug and this clinical finding has not been established.

There have been reports of dyskinetic movements of the lips, face, and extremities occurring with the use of Cylert. Convulsive seizures have also been reported. A definite causal relationship between Cylert and these reactions has not been established.

Mild adverse reactions appearing early during the course of treatment with Cylert often remit with continuing therapy. If adverse reactions are of a significant or protracted nature, dosage should be reduced or the drug discontinued.

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FAMILY THERAPY AND EVALUATION THROUGH ART *by* **Hanna Yaxa Kwiatkowska,** *The George Washington Univ., Washington, D. C. Foreword by* **Lyman C. Wynne.** This book describes the special kind of communication provided by the graphic and plastic media, and how it can and is being used with families for therapy, evaluation and research. Details of the family art session are presented, followed by discussions of family art therapy as it relates to adjunctive verbal therapy and to hysterical and schizophrenic patients. The procedures of family art evaluation are then meticulously examined. *'78, 304 pp., 125 il. (12 in color), 7 tables, $21.75*

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SOCIAL CONFLICT AND MENTAL HEALTH SERVICES *by* **Robert D. Borgman,** *Univ. of Southern Mississippi, Hattiesburg.* The author integrates social conflict theory with phenomenological, developmental, and problem-solving techniques to provide a singularly effective framework for understanding and intervening in the lives of troubled and troublesome individuals and families. Social conflict theory is also related to the problems of mental health organizations and professional relationships. A sampling of other topics includes mental patienthood, suicide and homicide, sexual oppression, school aversion, social conflicts of adolescence, and problems of the elderly. *'79, 356 pp., cloth-$24.50, paper-$18.50*

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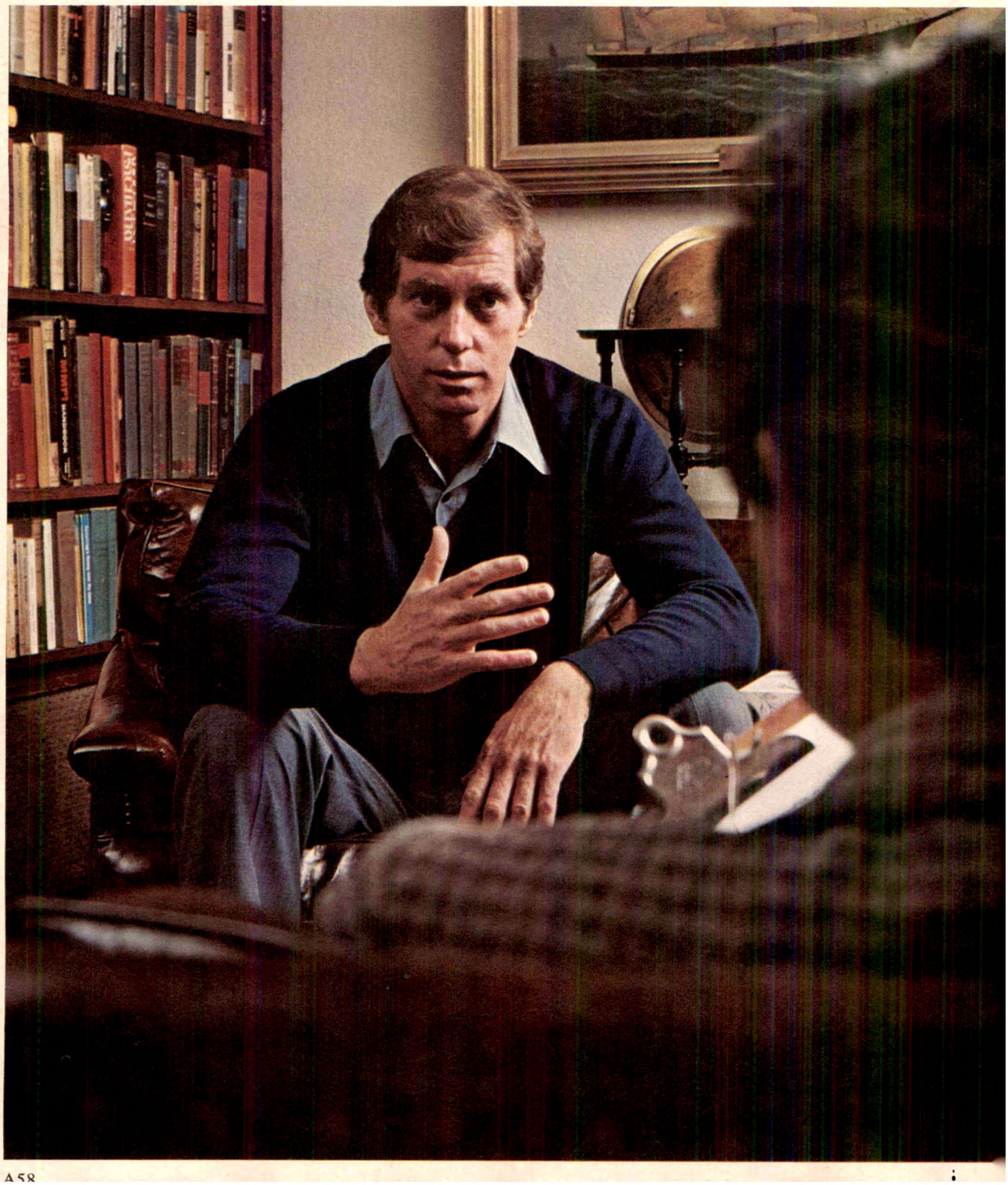
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MSD
MERCK SHARP & DOHME

Contraindications: Known hypersensitivity. Should not be given concomitantly with a monoamine oxidase inhibitor since hyperpyretic crises, severe convulsions, and deaths have occurred. When used to replace a monoamine oxidase inhibitor, allow a minimum of 14 days to elapse before initiating therapy with amitriptyline HCl. Initiate dosage of amitriptyline HCl cautiously with gradual increase in dosage until optimum response is achieved. Not recommended during the acute recovery phase following myocardial infarction.

Warnings: May block the antihypertensive action of guanethidine or similarly acting compounds. Should be used with caution in patients with a history of seizures or a history of urinary retention, or with angle-closure glaucoma or increased intraocular pressure; in patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely; arrhythmias, sinus tachycardia and prolongation of the conduction time have been reported, particularly with high doses; myocardial infarction and stroke have been reported with drugs of this class. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. May impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle. In patients who use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage. Safe use during pregnancy and lactation has not been established; in pregnant patients, nursing mothers, or women who may become pregnant, weigh possible benefits against possible hazards to mother and child. Not recommended for patients under 12 years of age.

Precautions: Schizophrenic patients may develop increased symptoms of psychosis; patients with paranoid symptomatology may have an exaggeration of such symptoms; manic depressive patients may experience a shift to the manic phase. In these circumstances, the dose of amitriptyline HCl may be reduced or a major tranquilizer, such as perphenazine, may be administered concurrently.

When given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required; paralytic ileus may occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs. Use cautiously in patients receiving large doses of ethchlorvynol, since transient delirium has been reported on concurrent administration. May enhance the response to alcohol and the effects of barbiturates and other CNS depressants. The possibility of suicide in depressed patients remains until significant remission occurs. Potentially suicidal patients should not have access to large quantities of this drug. Prescriptions should be written for the smallest amount feasible. Concurrent electroshock therapy may increase the hazards associated with such therapy; such treatment should be limited to patients for whom it is essential. When possible, discontinue the drug several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported. Use with caution in patients with impaired liver function.

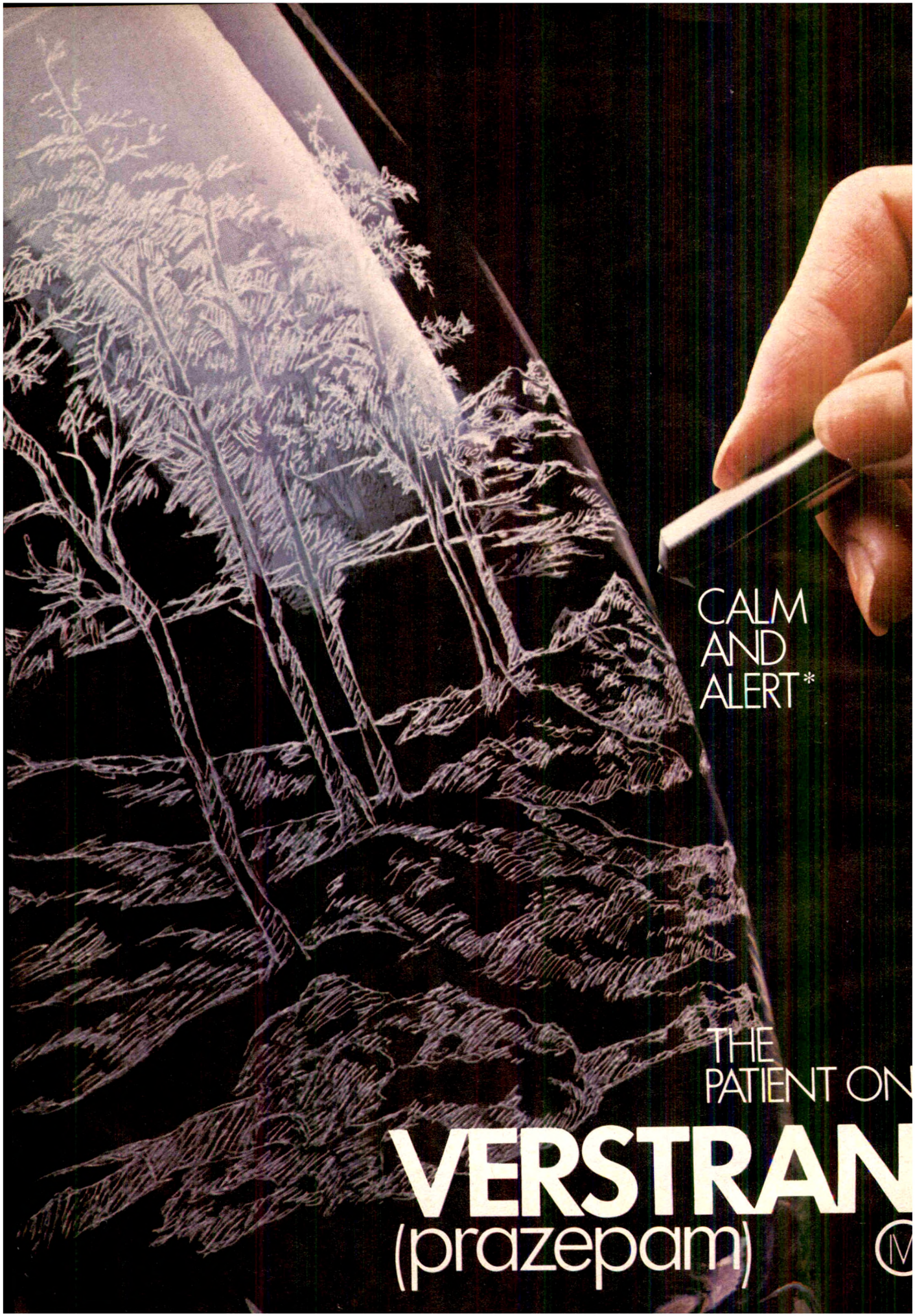
Adverse Reactions: *Note:* Included in this listing are a few adverse reactions not reported with this specific drug. However, pharmacological similarities among the tricyclic antidepressant drugs require that each reaction be considered when amitriptyline is administered. *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, increased intraocular pressure, constipation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, leukopenia, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, black tongue, rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache, and malaise; these are not indicative of addiction.

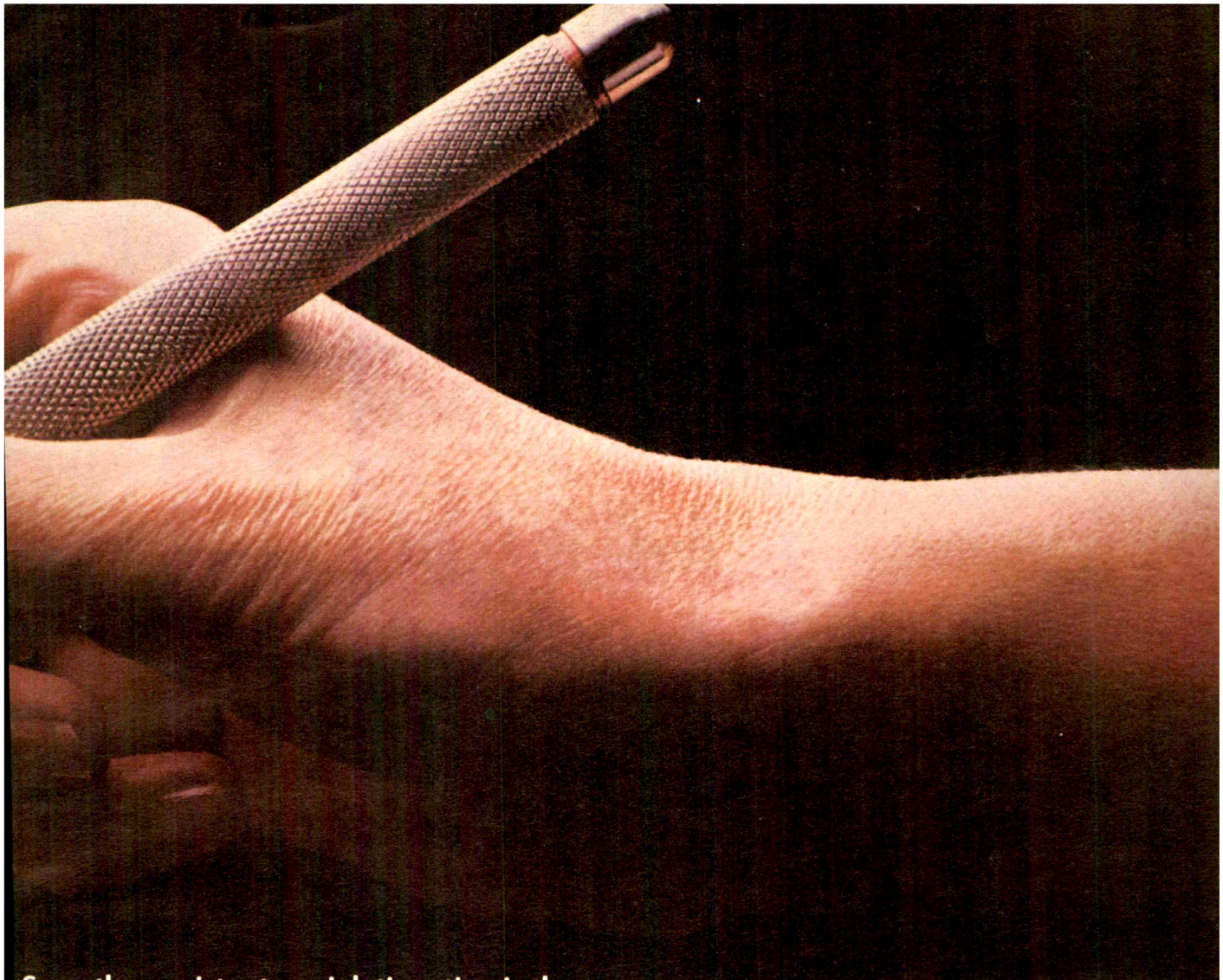
Overdosage: Hospitalize as soon as possible all patients suspected of having taken an overdose. Treatment is symptomatic and supportive. In addition, the intravenous administration of 1 to 3 mg physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage should be repeated as required, particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine.

How Supplied: Tablets containing 10 mg and 25 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100, 1000, and 5000; tablets containing 50 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100 and 1000; tablets containing 75 mg and 100 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100; tablets containing 150 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 30 and 100; for intramuscular use, in 10-ml vials containing per ml: 10 mg amitriptyline HCl, 44 mg dextrose, 1.5 mg methylparaben and 0.2 mg propylparaben as preservatives, and water for injection q.s. 1 ml.

For more detailed information, consult your MSD representative or see full prescribing information. Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa. 19486

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Smooth, consistent anxiolytic action is the reason.

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- Therapeutic response achieved with first dose
- Verstran—a suitable antianxiety alternative to diazepam

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- Other side effects, if present, are minimal†
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- No accumulation after steady state is reached

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- The simplicity of a single-strength 10-mg scored tablet, for easy titration and improved patient compliance
- The flexibility of *once daily*§ dosage
- The option of a *b.i.d.* dosage regimen, gradually adjustable within a range of 20 to 60 mg/day



*As with all CNS-acting agents, patients should be cautioned against driving, drinking, or engaging in hazardous activities.

†Data on file, Warner/Chilcott Medical Department.

‡See package insert.

§At bedtime.

Before prescribing, see complete product information on the following page.

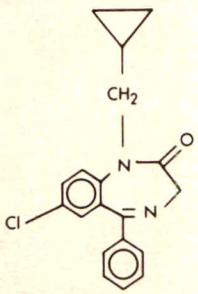
TO ENHANCE PATIENT RESPONSIVENESS CONSIDER

VERSTRAN (prazepam) Ⓒ IV

VERSTRAN® (prazepam) Ⓒ IV

Caution: Federal law prohibits dispensing without prescription.

Description: Verstran (prazepam), a benzodiazepine derivative, is identified chemically as 7-chloro-1-(cyclopropylmethyl)-1,3-dihydro-5-phenyl-2H-1,4-benzodiazepin-2-one. The molecular weight is 324.8 and the structural formula is as follows:



Clinical Pharmacology: Studies in normal subjects have shown that Verstran (prazepam) has depressant effects on the central nervous system. Oral administration of single doses as high as 60 mg and divided doses up to 100 mg t.i.d. (300 mg total daily dose) were without toxic effects.

Single oral doses of Verstran in normal subjects produced peak blood levels at 6 hours postadministration, with significant amounts still present after 48 hours. Verstran was slowly absorbed over a prolonged period, rather constant blood levels were maintained, and excretion was prolonged. The mean half-life of prazepam measured in subjects given 10 mg t.i.d. for one week was 63 (±15 s.d.) hours before and 70 (±10 s.d.) hours after multiple dosing — a nonsignificant difference. Human metabolism studies showed that prior to elimination from the body, prazepam is metabolized in large part to 3-hydroxyprazepam and oxazepam.

Indications: Verstran (prazepam) is indicated for the symptomatic relief of anxiety associated with anxiety neurosis, in other psychoneuroses in which anxiety symptoms are prominent features, and as an adjunct in disease states in which anxiety is manifested.

The effectiveness of Verstran (prazepam) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should reassess periodically the usefulness of the drug for the individual patient.

Contraindications: Verstran (prazepam) is contraindicated in patients with a known sensitivity to the drug and in those with acute narrow angle glaucoma.

Warning: Verstran (prazepam) is not recommended in psychotic states and in those psychiatric disorders in which anxiety is not a prominent feature.

Patients taking Verstran should be cautioned against engaging in hazardous occupations requiring mental alertness, such as operating dangerous machinery, including motor vehicles.

Because Verstran has a central nervous system depressant effect, patients should be advised against the simultaneous use of other CNS-depressant drugs, including phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants. The effects of alcohol may also be increased.

Physical and Psychological Dependence: Withdrawal symptoms similar in character to those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepine drugs. These symptoms include convulsions, tremor, abdominal and muscle cramps, vomiting and sweating. Addiction-prone individuals, such as drug addicts and alcoholics, should be under careful surveillance when receiving benzodiazepines because of the predisposition of such patients to habituation and dependence.

Precautions:

Use in Pregnancy and Lactation:

An increased risk of congenital malformations associated with the use of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) during the first trimester of pregnancy has been suggested in several studies. Verstran (prazepam) a benzodiazepine derivative, has not been studied adequately to determine whether it, too, may be associated with an increased risk of fetal abnormality. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physician about the desirability of discontinuing the drug. In view of their molecular size, Verstran and its metabolites are probably excreted in human milk. Therefore, this drug should not be given to nursing mothers.

In those patients in whom a degree of depression accompanies the anxiety, suicidal tendencies may be present, and protective measures may be required. The least amount of drug that is feasible should be available to the patient at any one time.

Patients taking Verstran for prolonged periods should have blood counts and liver function tests periodically. The usual precautions in treating patients with impaired renal or hepatic function should also be observed. Hepatomegaly and cholestasis were observed in chronic toxicity studies in rats and dogs.

In elderly or debilitated patients, the initial dose should be small, and increments should be made gradually, in accordance with the response of the patient, to preclude ataxia or excessive sedation.

Pediatric Use: Safety and effectiveness in patients below the age of 18 have not been established.

Adverse Reactions: The side effects most frequently reported during double-blind placebo-controlled trials employing a typical 30 mg divided total daily dose and the percent incidence in the Verstran (prazepam) group were: fatigue (11.6%), dizziness (8.7%), weakness (7.7%), drowsiness (6.8%), lightheadedness (6.8%), and ataxia (5.0%). Less frequently reported were: headache, confusion, tremor, vivid dreams, slurred speech, palpitation, stimulation, dry mouth, diaphoresis, and various gastrointestinal complaints. Other side effects included: pruritus, transient skin rashes, swelling of feet, joint pains, various genitourinary complaints, blurred vision, and syncope. Single nightly dose, controlled trials of variable dosages showed a dose-related incidence of these same side effects. Transient and reversible aberrations of liver function tests have been reported, as have slight decreases in blood pressure and increases in body weight.

These findings are characteristic of benzodiazepine drugs.

Overdosage: As in the management of overdosage with any drug, it should be borne in mind that multiple agents may have been taken.

If vomiting has not occurred spontaneously, it should be induced. Immediate gastric lavage is also recommended. General supportive care, including frequent monitoring of the vital signs and close observation of the patient, is indicated. Hypotension though unlikely, may be controlled with Levophed® (levarterenol bitartrate) or Aramine® (metaraminol bitartrate). Caffeine and Sodium Benzoate Injection USP, may be used to counteract central nervous system depressant effects.

Dosage and Administration: Verstran (prazepam) is administered orally in divided doses. The usual daily dose is 30 mg. The dose should be adjusted gradually within the range of 20 to 60 mg daily in accordance with the response of the patient. In elderly or debilitated patients it is advisable to initiate treatment at a divided daily dose of 10 mg to 15 mg. (See Precautions.)

Prazepam may also be administered as a single dose daily at bedtime. The recommended starting nightly dose is 20 mg. The response of the patient to several days' treatment will permit the physician to adjust the dose upward or, occasionally, downward to maximize antianxiety effect with a minimum of daytime drowsiness. The optimum dosage will usually range from 20 to 40 mg.

Drug Interactions: If Verstran (prazepam) is to be combined with other drugs acting on the central nervous system, careful consideration should be given to the pharmacology of the agents to be employed. The actions of the benzodiazepines may be potentiated by barbiturates, narcotics, phenothiazines, monoamine oxidase inhibitors or other antidepressants.

If Verstran is used to treat anxiety associated with somatic disease states, careful attention must be paid to possible drug interaction with concomitant medication.

How Supplied: Verstran (prazepam) 10 mg light blue scored tablets in bottles of 100 (N 0047-0276-51) and 1000 (N 0047-0276-60).

STORE BETWEEN 59°–86° F (15°–30° C).

VE-GP-8

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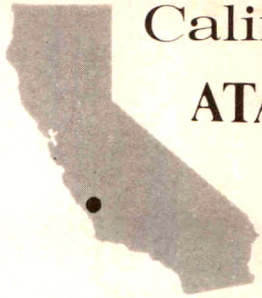
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You know what to expect from antidepressant therapy;



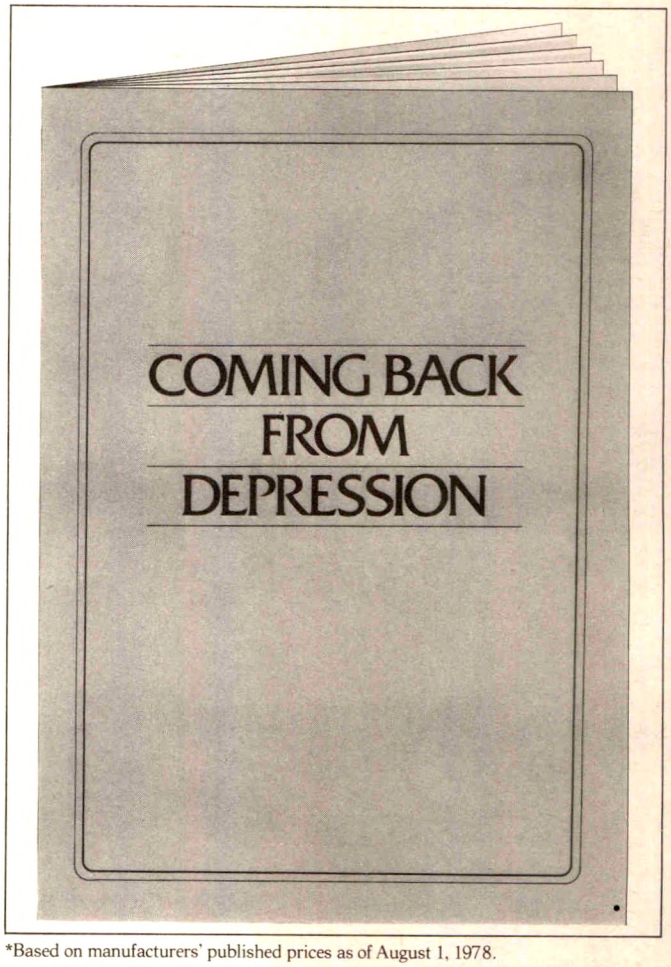
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*Based on manufacturers' published prices as of August 1, 1978.

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Amitriptyline Hydrochloride Tablets USP

lowers the cost, not the quality, of antidepressant therapy

Please see following page for brief summary.

SQUIBB®

Amitid™ (Amitriptyline Hydrochloride Tablets USP)

DESCRIPTION: Amitriptyline hydrochloride is a dibenzocycloheptadiene derivative available for oral administration as FILMLOK® tablets (FILMLOK is a Squibb trademark for veneer-coated tablets).

INDICATIONS: For relief of symptoms of depression alone or accompanied by anxiety. Endogenous depression is more likely to be alleviated than are other depressive states.

CONTRAINDICATIONS: Contraindicated in patients with a history of hypersensitivity to amitriptyline. Do not administer concomitantly with a monamine oxidase (MAO) inhibitor since hyperpyretic crises, severe convulsions, and deaths occurred when tricyclic antidepressants and MAO inhibitors were administered simultaneously. When an MAO inhibitor is to be replaced by amitriptyline, allow 14 days to elapse after discontinuation of the former and then initiate amitriptyline cautiously and gradually increase dosage until optimum response is achieved. Amitriptyline is not recommended for use during the acute recovery phase following myocardial infarction.

WARNINGS: Amitriptyline may block antihypertensive action of guanethidine or similarly acting drugs. Use with caution in patients with history of seizures and (because of atropine-like action of amitriptyline) in patients with history of narrow-angle glaucoma (even average doses may precipitate an attack), increased intraocular pressure, or urinary retention. Closely watch patients with cardiovascular disorders since, in addition to having caused myocardial infarction and stroke, tricyclic antidepressants (including amitriptyline) particularly with high dosage have been reported to produce arrhythmias, sinus tachycardia, and prolongation of conduction time. Close supervision is required when amitriptyline is given to hyperthyroid patients or those on thyroid medication. Amitriptyline may impair mental and/or physical abilities required for performance of hazardous tasks such as operating machinery or driving a motor vehicle. Bear in mind that in patients who may use alcohol excessively the potentiation may increase the danger inherent in any suicide attempt or overdosage.

Usage in Pregnancy: Safe use during pregnancy and lactation has not been established; therefore, in administering the drug to pregnant patients, nursing mothers, or women who may become pregnant, weigh the possible benefits against the possible hazards to the mother and child. Animal reproduction studies have been inconclusive, and clinical experience has been limited.

Usage in Children: At the present time, not recommended for patients under 12 years of age because of lack of experience with use in children.

PRECAUTIONS: Schizophrenic patients may develop increased symptoms of psychosis; patients with paranoid symptomatology may have an exaggeration of such symptoms; manic depressive patients may experience a shift to the manic phase. In these circumstances the dose of amitriptyline may be reduced or a major tranquilizer may be administered concurrently.

Closely supervise and carefully adjust dosage in concomitant use with anticholinergic or sympathomimetic drugs, including combination of epinephrine and local anesthetics. Paralytic ileus may occur with concomitant use of tricyclic antidepressants and anticholinergic-type drugs. Caution is advised if used concurrently with large doses of ethchlorvynol since transient delirium has been reported when one gram of that drug and 75 to 150 mg of amitriptyline HCl were administered. Amitriptyline may enhance response to alcohol and the effects of barbiturates and other CNS depressants. The possibility of suicide in depressed patients remains during treatment and until significant remission occurs. Potentially suicidal patients should not have easy access to large quantities of the drug. Prescriptions should be written for the smallest amount feasible. Limit concurrent administration of this drug and electroshock therapy to patients for whom it is essential since the hazards associated with such therapy may be increased. Discontinue this drug, when possible, several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported. Use amitriptyline with caution in presence of impaired liver function.

ADVERSE REACTIONS: NOTE: A few of the adverse reactions listed below have not been reported with this specific drug, but each of the reactions should be considered when administering amitriptyline because of pharmacological similarities among tricyclic antidepressants.

Cardiovascular: Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, and stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness; tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; and syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, increased intraocular pressure, constipation, paralytic ileus, urinary retention, and dilatation of the urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, and edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, leukopenia, eosinophilia, purpura, and thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, and black tongue. Rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, and elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, and alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache, and malaise. These are not indicative of addiction.

For full prescribing information, consult package insert.

HOW SUPPLIED: Available for oral administration in tablets providing 10, 25, 50, 75, and 100 mg amitriptyline hydrochloride. The 10, 25, and 50 mg tablets are available in bottles of 100 and 1000. The 75 and 100 mg tablets are available in bottles of 100.

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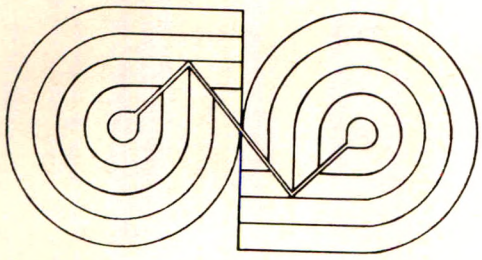
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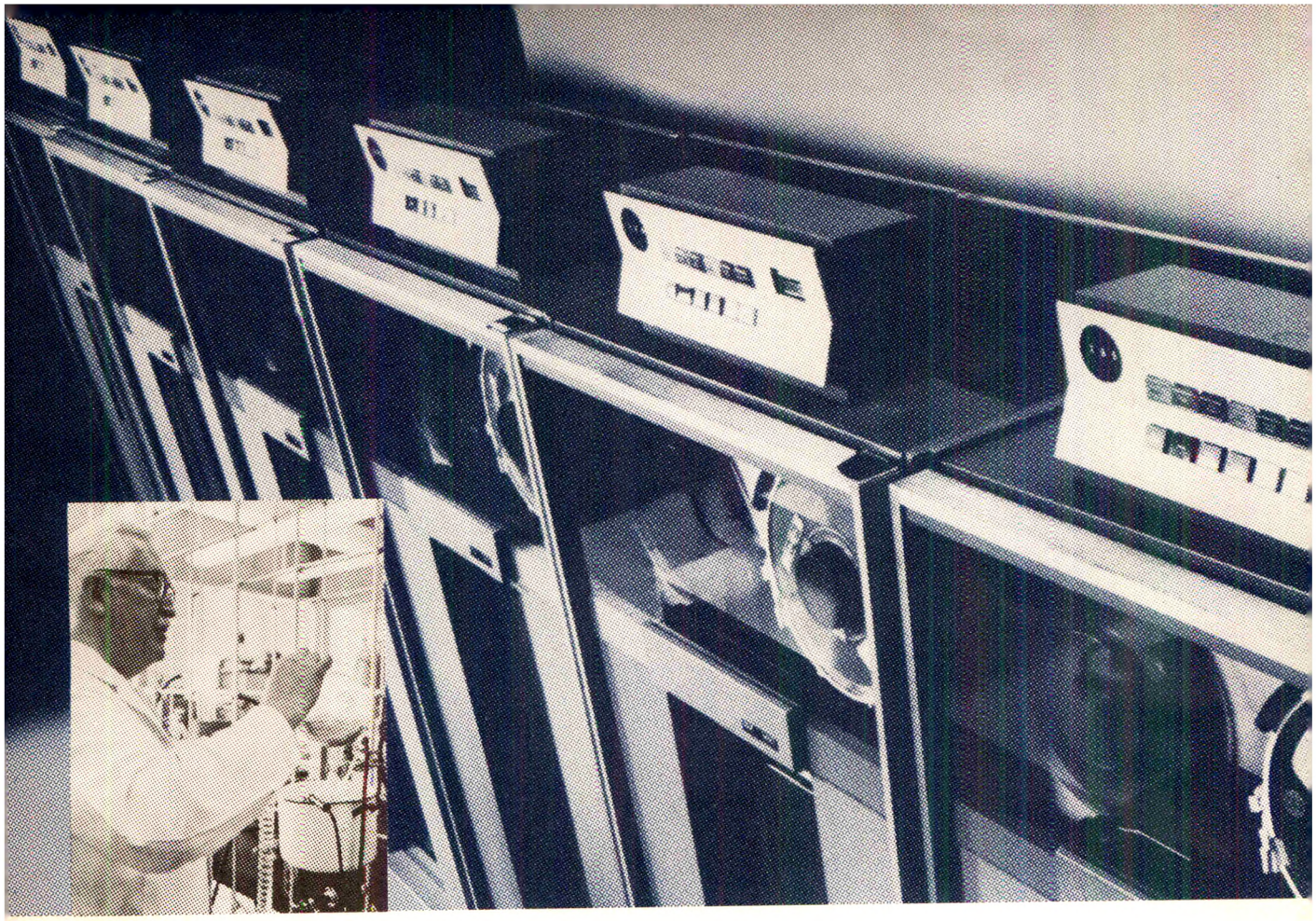
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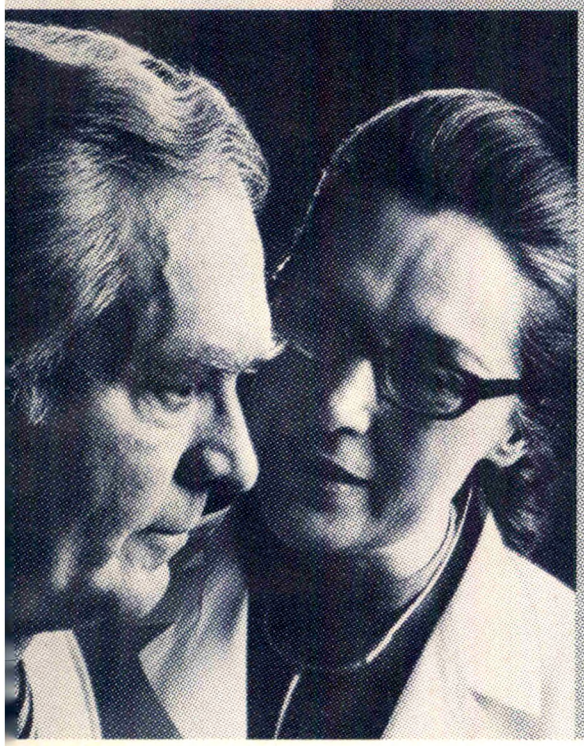
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chlordiazepoxide HCl/Roche



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Please see next page for summary of product information.

Librium® 5mg, 10mg, 25mg capsules
chlordiazepoxide HCl/Roche

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Relief of anxiety and tension occurring alone or accompanying various disease states. Efficacy beyond four months not established by systematic clinical studies. Periodic reassessment of therapy recommended.

Contraindications: Patients with known hypersensitivity to the drug.

Warnings: Warn patients that mental and/or physical abilities required for tasks such as driving or operating machinery may be impaired, as may be mental alertness in children, and that concomitant use with alcohol or CNS depressants may have an additive effect. Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported.

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (*e.g.*, excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.

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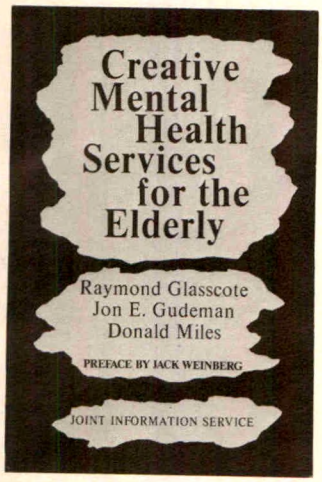
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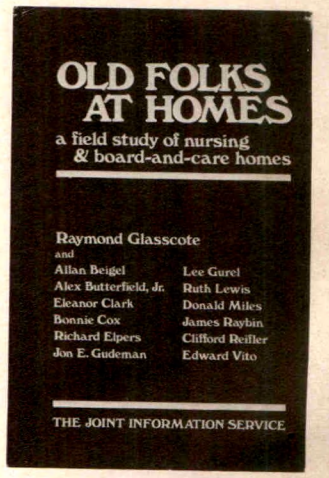
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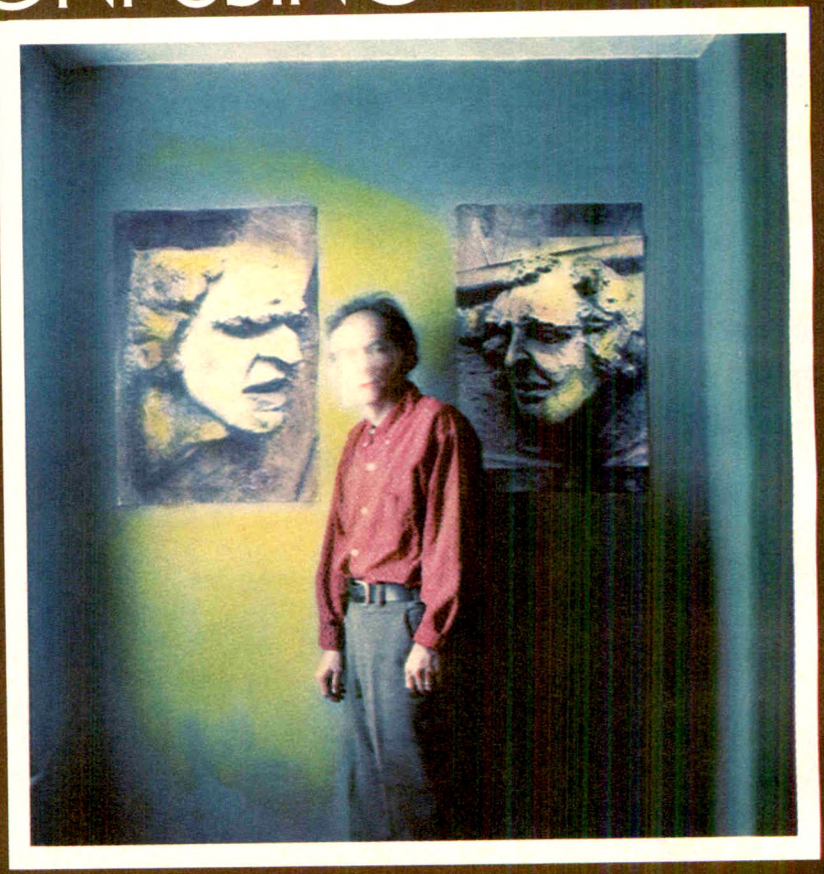
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Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

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'Stelazine' has not been shown effective in the management of behavioral complications in patients with mental retardation.

Final classification of the less-than-effective indications requires further investigation.

Contraindications: Comatose or greatly depressed states due to C.N.S. depressants; blood dyscrasias; bone marrow depression; liver damage.

Warnings: Generally avoid using in patients hypersensitive (e.g., blood dyscrasias, jaundice) to any phenothiazine. Caution patients about activities requiring alertness (e.g., operating vehicles or machinery), especially during the first few days' therapy.

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Precautions: Use cautiously in angina. Avoid high doses and parenteral administration when cardiovascular system is impaired. Antiemetic effect may mask signs of toxic drug overdosage or physical disorders. Additive effect is possible with other C.N.S. depressants. Prolonged administration of high doses may result in cumulative effects with severe C.N.S. or vasomotor symptoms. If retinal changes occur, discontinue drug. Agranulocytosis, thrombocytopenia, pancytopenia, anemia, cholestatic jaundice, liver damage have been reported.

Patients on long-term therapy, especially high doses, should be evaluated periodically for possible adjustment or discontinuance of drug therapy.

Adverse Reactions: Drowsiness, dizziness, skin reactions, rash, dry mouth, insomnia, amenorrhea, fatigue, muscular weakness, anorexia, lactation, blurred vision. Neuromuscular (extrapyramidal) reactions: motor restlessness, dystonias, pseudo-parkinsonism, persistent tardive dyskinesia.

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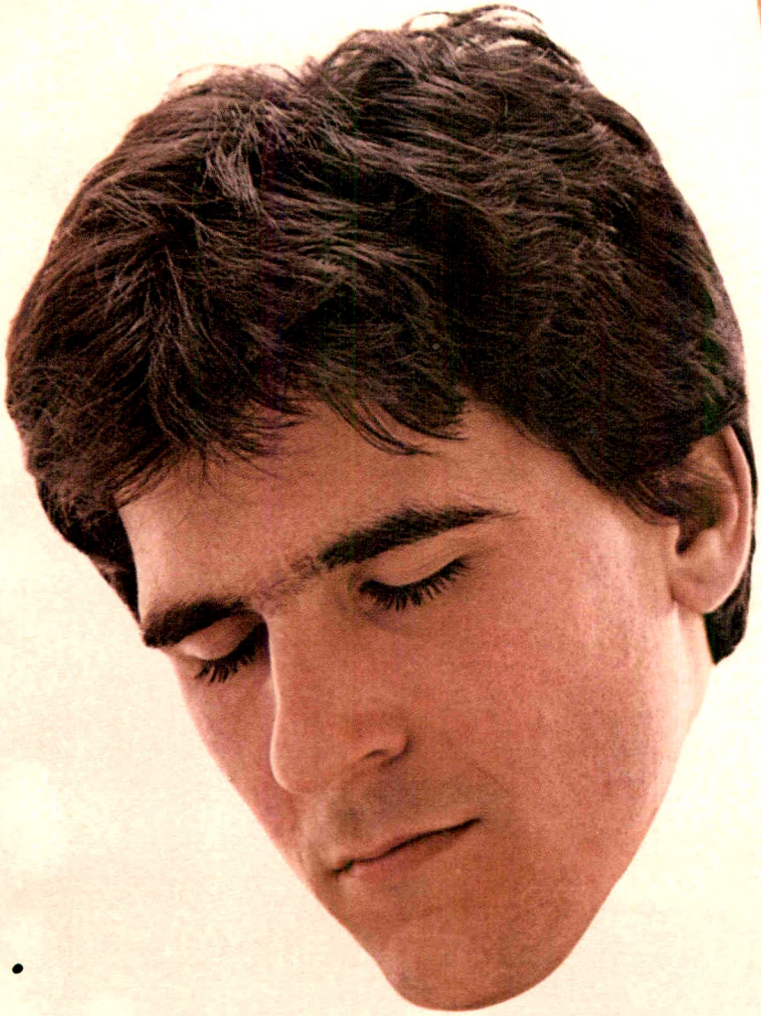
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Help for the apathetic/ withdrawn geriatric*

For the aged person suffering from withdrawn or apathetic senile behavior, Ritalin can often help relieve bothersome symptoms. In one double-blind study[1] of withdrawn, apathetic geriatric patients, Ritalin elicited notable improvement in competence, interest and retardation. While no side effects were reported in this study, blood pressure and pulse variations were noted in both groups but were not considered of exceptional magnitude.





Help for narcoleptic patients

The narcoleptic patient can fall asleep anytime...anyplace... and the consequences can be grave, or at the least, embarrassing. Although there is no known cure for narcolepsy, Ritalin acts promptly to counteract sleepiness and is the medication most frequently used by cited investigators.[2,3] Occasional problems of tolerance may be managed by complete withdrawal of Ritalin during vacation or holidays, then reinstating the drug at lower dosage.[3]

Ritalin has been evaluated as possibly effective in these indications.

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Help for patients with mild depression*

As Ritalin acts promptly to relieve symptoms of mild depression, mood and outlook usually improve, and the patient can cope again. Generally, one prescription is sufficient. Ritalin is contraindicated in severe depression.

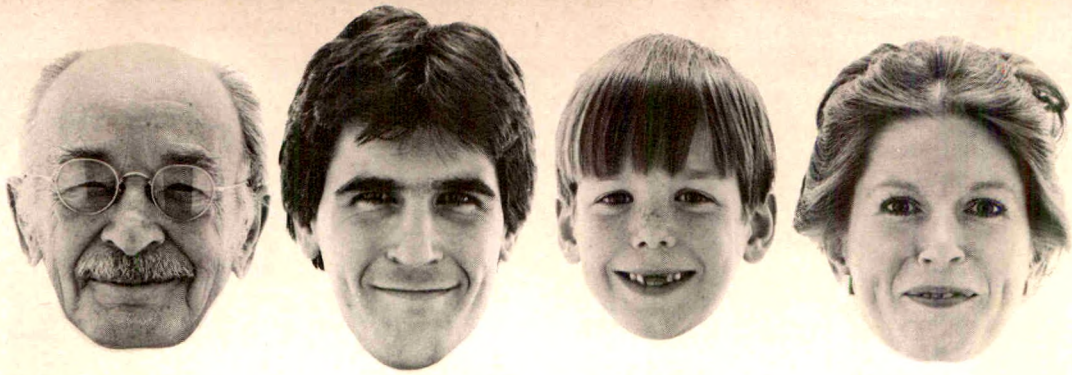


Help for the MBD child

For the child with Minimal Brain Dysfunction (MBD), adjunctive Ritalin is often highly effective when medication is needed to help reduce symptoms such as distractibility,[4] disorganized behavior,[4] and hyperactivity.[4] MBD children responding to Ritalin usually exhibit better classroom performance and improved interpersonal relationships. Moreover, as MBD symptoms are curbed, the affected child often responds more positively to nonpharmacologic modalities.[5] Ritalin should be periodically discontinued to assess the child's condition. Improvement may be sustained when the drug is temporarily or permanently discontinued.

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TABLETS

INDICATIONS
Based on a review of this drug by the National Academy of Sciences-National Research Council and/or other information, FDA has classified the indications as follows:
Effective: Minimal Brain Dysfunction in Children—as adjunctive therapy to other remedial measures (psychological, educational, social)
Special Diagnostic Considerations
Specific etiology of Minimal Brain Dysfunction (MBD) is unknown, and there is no single diagnostic test. Adequate diagnosis requires the use not only of medical but of special psychological, educational, and social resources.
Characteristics commonly reported include: chronic history of short attention span, distractibility, emotional lability, impulsivity, and moderate to severe hyperactivity; minor neurological signs and abnormal EEG. Learning may or may not be impaired. The diagnosis of MBD must be based upon a complete history and evaluation of the child and not solely on the presence of one or more of these characteristics.
Drug treatment is not indicated for all children with MBD. Stimulants are not intended for use in the child who exhibits symptoms secondary to environmental factors and/or primary psychiatric disorders, including psychosis.
Appropriate educational placement is essential and psychosocial intervention is generally necessary. When remedial measures alone are insufficient, the decision to prescribe stimulant medication will depend upon the physician's assessment of the chronicity and severity of the child's symptoms.
Effective: Narcolepsy
"Possibly" effective: Mild Depression; Apathetic or Withdrawn Senile Behavior
Final classification of the less-than-effective indications requires further investigation.

CONTRAINDICATIONS
Marked anxiety, tension, and agitation are contraindications to Ritalin, since the drug may aggravate these symptoms. Ritalin is contraindicated also in patients known to be hypersensitive to the drug and in patients with glaucoma.

WARNINGS
Ritalin should not be used in children under six years, since safety and efficacy in this age group have not been established.
Sufficient data on safety and efficacy of long-term use of Ritalin in children with minimal brain dysfunction are not yet available. Although a causal relationship has not been established, suppression of growth (*ie*, weight gain, and/or height) has been reported with the long-term use of stimulants in children. Therefore, patients requiring long-term therapy should be carefully monitored.
Ritalin should not be used for severe depression of either exogenous or endogenous origin.
Ritalin should not be used for the prevention or treatment of normal fatigue states.
There is some clinical evidence that Ritalin may lower the convulsive threshold in patients with prior history of seizures, with prior EEG abnormalities in absence of seizures, and, very rarely, in absence of history of seizures and no prior EEG evidence of seizures. Safe concomitant use of anticonvulsants and Ritalin has not been established. In the presence of seizures, the drug should be discontinued.
Use cautiously in patients with hypertension. Blood pressure should be monitored at appropriate intervals in all patients taking Ritalin, especially those with hypertension.
Symptoms of visual disturbances have been encountered in rare cases. Difficulties with accommodation and blurring of vision have been reported.

Drug Interactions
Ritalin may decrease the hypotensive effect of guanethidine. Use cautiously with pressor agents and MAO inhibitors.
Human pharmacologic studies have shown that Ritalin may inhibit the metabolism of coumarin anticoagulants, anticonvulsants (phenobarbital, diphenylhydantoin, primidone), phenylbutazone, and tricyclic antidepressants (imipramine, desipramine). Downward dosage adjustments of these drugs may be required when given concomitantly with Ritalin.

Usage in Pregnancy
Adequate animal reproduction studies to establish safe use of Ritalin during pregnancy have not been conducted. Therefore, until more information is available, Ritalin should not be prescribed for women of childbearing age unless, in the opinion of the physician, the potential benefits outweigh the possible risks.

Drug Dependence
Ritalin should be given cautiously to emotionally unstable patients, such as those with a history of drug dependence or alcoholism, because such patients may increase dosage on their own initiative.
Chronically abusive use can lead to marked tolerance and psychic dependence with varying degrees of abnormal behavior. Frank psychotic episodes can occur, especially with parenteral abuse. Careful supervision is required during drug withdrawal, since severe depression as well as the effects of chronic overactivity can be unmasked. Long-term follow-up may be required because of the patient's basic personality disturbances.

PRECAUTIONS
Patients with an element of agitation may react adversely; discontinue therapy if necessary.
Periodic CBC, differential, and platelet counts are advised during prolonged therapy.

ADVERSE REACTIONS
Nervousness and insomnia are the most common adverse reactions but are usually controlled by reducing dosage and omitting the drug in the afternoon or evening. Other reactions include hypersensitivity (including skin rash, urticaria, fever, arthralgia, exfoliative dermatitis, erythema multiforme with histopathological findings of necrotizing vasculitis, and thrombocytopenic purpura); anorexia; nausea; dizziness; palpitations; headache; dyskinesia; drowsiness; blood pressure and pulse changes, both up and down; tachycardia; angina; cardiac arrhythmia; abdominal pain; weight loss during prolonged therapy. Toxic psychosis has been reported. Although a definite causal relationship has not been established, the following have been reported in patients taking this drug: leukopenia and/or anemia; few instances of scalp hair loss.
In children, loss of appetite, abdominal pain, weight loss during prolonged therapy, insomnia, and tachycardia may occur more frequently; however, any of the other adverse reactions listed above may also occur.

DOSAGE AND ADMINISTRATION
Adults
Administer orally in divided doses 2 or 3 times daily, preferably 30 to 45 minutes before meals. Dosage will depend upon indication and individual response.
Average dosage is 20 to 30 mg daily. Some patients may require 40 to 60 mg daily. In others, 10 to 15 mg daily will be adequate. Patients who are unable to sleep if medication is taken late in the day should take the last dose before 6 p.m.
Children with Minimal Brain Dysfunction
(6 years and over)
Start with small doses (*eg*, 5 mg before breakfast and lunch) with gradual increments of 5 to 10 mg weekly. Daily dosage above 60 mg is not recommended. If improvement is not observed after appropriate dosage adjustment over a one-month period, the drug should be discontinued.
If paradoxical aggravation of symptoms or other adverse effects occur, reduce dosage, or, if necessary, discontinue the drug.
Ritalin should be periodically discontinued to assess the child's condition. Improvement may be sustained when the drug is either temporarily or permanently discontinued.
Drug treatment should not and need not be indefinite and usually may be discontinued after puberty.

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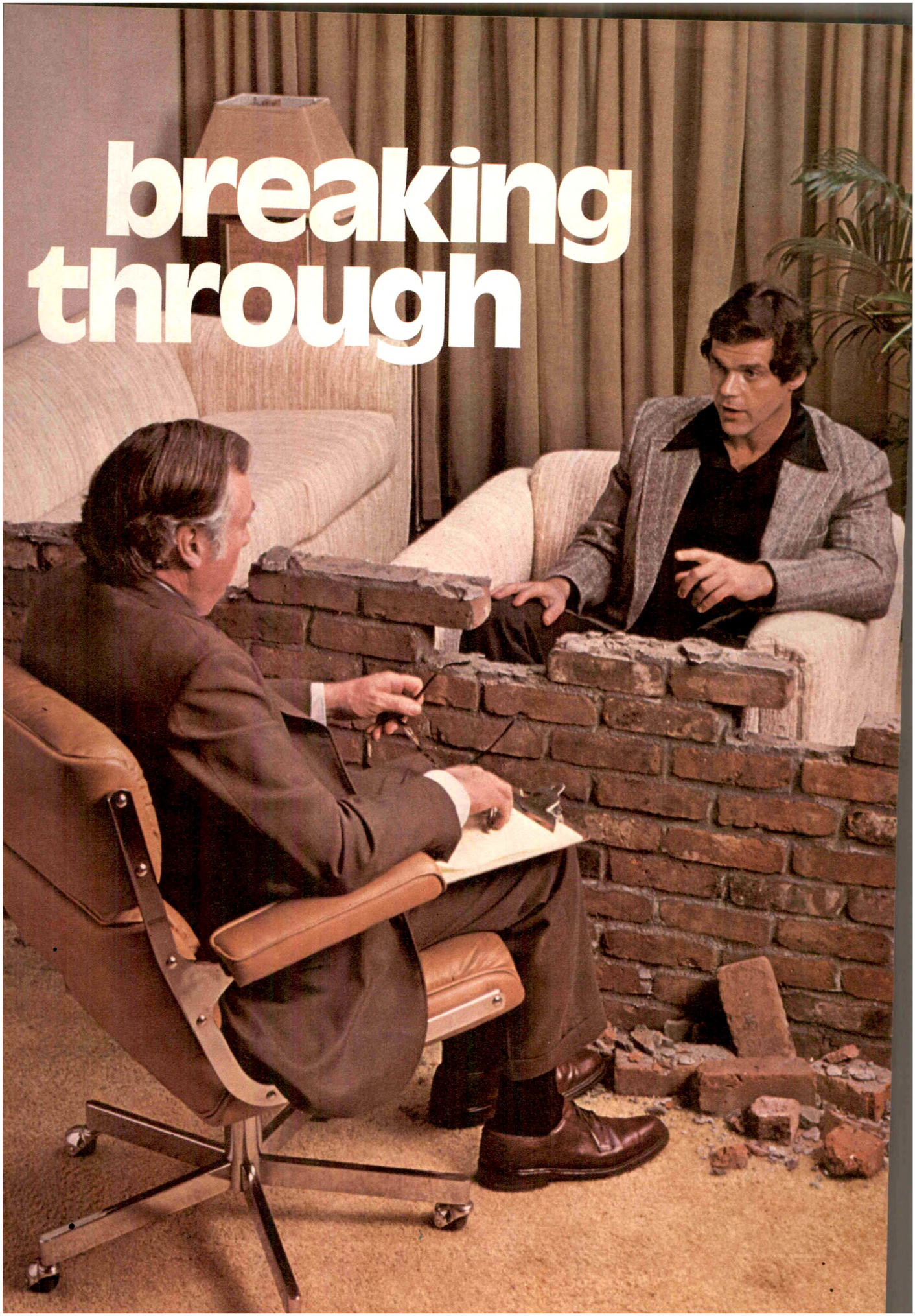
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CONTRAINDICATIONS: Central nervous system depression from drugs (barbiturates, alcohol, narcotics, analgesics, antihistamines); evidence of bone marrow depression; known hypersensitivity to phenothiazines or amitriptyline. Should not be given concomitantly with a monoamine oxidase inhibitor since hyperpyretic crises, severe convulsions, and deaths have occurred from such combinations. When used to replace a monoamine oxidase inhibitor, allow a minimum of 14 days to elapse before initiating therapy with TRIAVIL. Therapy should then be initiated cautiously with gradual increase in dosage until optimum response is achieved. Not recommended for use during acute recovery phase following myocardial infarction.

WARNINGS: TRIAVIL should not be given concomitantly with guanethidine or similarly acting compounds since TRIAVIL may block the antihypertensive action of such compounds. Use cautiously in patients with history of urinary retention, angle-closure glaucoma, increased intraocular pressure, or convulsive disorders. Dosage of anticonvulsive agents may have to be increased. In patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely. Tricyclic antidepressants, including amitriptyline HCl, have been reported to produce arrhythmias, sinus tachycardia, and prolongation of conduction time, particularly in high doses. Myocardial infarction and stroke have been reported with tricyclic antidepressant drugs. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. May impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle. In patients who use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage. Not recommended in children or during pregnancy.

PRECAUTIONS: Suicide is a possibility in depressed patients and may remain until significant remission occurs. Such patients should not have access to large quantities of this drug.

Perphenazine: Should not be used indiscriminately. Use with caution in patients who have previously exhibited severe adverse reactions to other phenothiazines. Likelihood of some untoward actions is greater with high doses. Closely supervise with any dosage. The antiemetic effect of perphenazine may obscure signs of toxicity due to overdosage of other drugs or make more difficult the diagnosis of disorders such as brain tumor or intestinal obstruction. A significant, not otherwise explained, rise in body temperature may suggest individual intolerance to perphenazine, in which case discontinue.

If hypotension develops, epinephrine should not be employed, as its action is blocked and partially reversed by perphenazine. Phenothiazines may potentiate the action of central nervous system depressants (opiates, analgesics, antihistamines, barbiturates, alcohol) and atropine. In concurrent therapy with any of these, TRIAVIL should be given in reduced dosage. May also potentiate the action of heat and phosphorous insecticides. There is sufficient experimental evidence to conclude that chronic administration of antipsychotic drugs which increase prolactin secretion has the potential to induce mammary neoplasms in rodents under the appropriate conditions. There are recognized differences in the physiological role of prolactin between rodents and humans. Since there are, at present, no adequate epidemiological studies, the relevance to human mammary cancer risk from prolonged exposure to perphenazine and other antipsychotic drugs is not known.

Amitriptyline: In manic-depressive psychosis, depressed patients may experience a shift toward the manic phase if they are treated with an antidepressant. Patients with paranoid symptomatology may have an exaggeration of such symptoms. The tranquilizing effect of TRIAVIL seems to reduce the likelihood of this effect. When amitriptyline HCl is given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required. Paralytic ileus may occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs.

Caution is advised if patients receive large doses of ethchlorvynol concurrently. Transient delirium has been reported in patients who were treated with 1 g of ethchlorvynol and 75-150 mg of amitriptyline HCl.

Amitriptyline HCl may enhance the response to alcohol and the effects of barbiturates and other CNS depressants.

Concurrent administration of amitriptyline HCl and electroshock therapy may increase the hazards associated with such therapy. Such treatment should be limited to patients for whom it is essential. Discontinue several days before elective surgery if possible. Elevation and lowering of blood sugar levels have both been reported. Use with caution in patients with impaired liver function.

ADVERSE REACTIONS: Similar to those reported with either constituent alone.
Perphenazine: Extrapyramidal symptoms (opisthotonus, oculogyric crisis, hyperreflexia, dystonia, akathisia, acute dyskinesia, ataxia, parkinsonism) have been reported and can usually be controlled by the concomitant use of effective antiparkinsonian drugs and/or by reduction in dosage, but sometimes persist after discontinuation of the phenothiazine.

Tardive dyskinesia may appear in some patients on long-term therapy or may occur after drug therapy with phenothiazines and related agents has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. Symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw. Involuntary movements of the extremities sometimes occur. There is no known treatment for tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms. It is advised that all antipsychotic agents be discontinued if the above symptoms appear. If treatment is reinstituted, or dosage of the particular drug increased, or another drug substituted, the syndrome may be masked. Fine vermicular movements of the tongue may be an early sign of the syndrome. The full-blown syndrome may not develop if medication is stopped when lingual vermiculation appears.

Other side effects are skin disorders (photosensitivity, itching, erythema urticaria, eczema, up to exfoliative dermatitis); other allergic reactions (asthma laryngeal edema, angioneurotic edema, anaphylactoid reactions); peripheral edema; reversed epinephrine effect; hyperglycemia; endocrine disturbances (lactation, galactorrhea, gynecomastia, disturbances of menstrual cycle); altered cerebrospinal fluid proteins; paradoxical excitement; hypertension, hypotension, tachycardia, and ECG abnormalities (quinidine-like effect); reactivation of psychotic processes; catatonic-like states; autonomic reactions, such as dry mouth or salivation, headache, anorexia, nausea, vomiting, constipation, obstipation, urinary frequency or incontinence, blurred vision, nasal congestion, and a change in pulse rate; other adverse reactions reported with various phenothiazine compounds, but not with perphenazine, include grand mal convulsions, cerebral edema, polyphagia, pigmentary retinopathy, photophobia, skin pigmentation, and failure of ejaculation.

The phenothiazine compounds have produced blood dyscrasias (pancytopenia, thrombocytopenic purpura, leukopenia, agranulocytosis, eosinophilia) and liver damage (jaundice, biliary stasis).

Pigmentation of the cornea and lens has been reported to occur after long-term administration of some phenothiazines. Although it has not been reported in patients receiving TRIAVIL, the possibility that it might occur should be considered.

Hypnotic effects, lassitude, muscle weakness, and mild insomnia have also been reported.

Amitriptyline: Note: Listing includes a few reactions not reported for this drug, but which have occurred with other pharmacologically similar tricyclic antidepressant drugs and must be considered when amitriptyline is administered. *Cardiovascular:* Hypotension; hypertension; tachycardia; palpitation; myocardial infarction; arrhythmias; heart block; stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth; blurred vision; disturbance of accommodation; increased intraocular pressure; constipation; paralytic ileus; urinary retention; dilatation of urinary tract. *Allergic:* Skin rash; urticaria; photosensitization; edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis; leukopenia; eosinophilia; purpura; thrombocytopenia. *Gastrointestinal:* Nausea; epigastric distress; vomiting; anorexia; stomatitis; peculiar taste; diarrhea; parotid swelling; black tongue. Rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido; elevated or lowered blood sugar levels. *Other:* Dizziness, weakness; fatigue; headache; weight gain or loss; increased perspiration; urinary frequency; mydriasis; drowsiness; alopecia. *Withdrawal Symptoms:* Abrupt cessation after prolonged administration may produce nausea, headache, and malaise. These are not indicative of addiction.

OVERDOSAGE: All patients suspected of having taken an overdosage should be admitted to a hospital as soon as possible. Treatment is symptomatic and supportive. However, the intravenous administration of 1–3 mg of physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage of physostigmine should be repeated as required particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine. On this basis, in severe overdosage with perphenazine-amitriptyline combinations, symptomatic treatment of central anticholinergic effects with physostigmine salicylate should be considered.

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rescribing Information for

Lidone™ (molindone hydrochloride)

ESCRIPTION — LIDONE (molindone hydrochlo-e) is a dihydroindolone compound which is not ucturally related to the phenothiazines, the tyrophenones or the thioxanthenes.
LIDONE is 3-ethyl-6, 7-dihydro-2-methyl-5-(mor-olinomethyl) indol-4(5H)-one hydrochloride. It a white crystalline powder, freely soluble in ter and alcohol and has a molecular weight of 2.67.

CTIONS — LIDONE (molindone hydrochloride) s a pharmacological profile in laboratory mals which predominantly resembles that of jor tranquilizers causing reduction of spon-eous locomotion and aggressiveness, suppres-n of a conditional response and antagonism of bizarre stereotyped behavior and hyperactivity uced by amphetamines. In addition, LIDONE an-onizes the depression caused by the tranquiliz-agent tetrabenazine.
n human clinical studies tranquilization is ieved in the absence of muscle relaxing or in-rdinating effects. Based on EEG studies, ONE exerts its effect on the ascending reticular ivating system.
Human metabolic studies show LIDONE (molin-e hydrochloride) to be rapidly absorbed and tabolized when given orally. Unmetabolized g reached a peak blood level at 1.5 hours. Phar-cological effect from a single oral dose persists 24 to 36 hours. There are 36 recognized tabolites with less than 2 to 3% unmetabolized ONE being excreted in urine and feces.

DICATIONS — LIDONE (molindone hydrochlo-e) is indicated in the management of the nifestations of schizophrenia.

NTRAINDICATIONS — LIDONE (molindone rochloride) is contraindicated in severe central vous system depression (alcohol, barbiturates, cotics, etc.) or comatose states, and in patients h known hypersensitivity to the drug.

RNINGS

ge in Pregnancy: Studies in pregnant pa-ts have not been carried out. Reproduction dies have been performed in the following mals:

gnant Rats Oral Dose —
mg/kg/day — 10 days, no adverse effect
mg/kg/day — 10 days, no adverse effect

gnant Mice Oral Dose —
mg/kg/day — 10 days, slight increase resorp-s
mg/kg/day — 10 days, slight increase resorp-s

gnant Rabbits Oral Dose —
g/kg/day — 12 days, no adverse effect
ng/kg/day — 12 days, no adverse effect
ng/kg/day — 12 days, no adverse effect

mal reproductive studies have not demon-ted a teratogenic potential. The anticipated efits must be weighed against the unknown s to the fetus if used in pregnant patients.

rsing Mothers: Data are not available on the tent of LIDONE (molindone hydrochloride) in milk of nursing mothers.

ge in Children: Use of LIDONE (molindone rochloride) in children below the age of twelve rs is not recommended because safe and effec-conditions for its usage have not been estab-ed.

ECAUTIONS — Some patients receiving ONE (molindone hydrochloride) may note vsiness initially and they should be advised nst activities requiring mental alertness until r response to the drug has been established.
creased activity has been noted in patients iving LIDONE. Caution should be exercised re increased activity may be harmful.
DONE does not lower the seizure threshold in rimental animals to the degree noted with e sedating antipsychotic drugs. However, con-ive seizures have been reported in a few ances.
DONE has an antiemetic effect in animals. A lar effect may occur in humans and may ure signs of intestinal obstruction or brain or.

ADVERSE REACTIONS

CNS Effects — The most frequently occurring effect is initial drowsiness that generally subsides with continued usage of the drug or lowering of the dose.

Noted less frequently were depression, hyperactivity and euphoria.

Neurological Extrapyramidal Reactions — Extrapyramidal reactions noted below may occur in susceptible individuals and are usually reversible with appropriate management.

Akathisia — Motor restlessness may occur early.

Parkinson Syndrome — Akinesia, characterized by rigidity, immobility and reduction of voluntary movements and tremor, have been observed. Occurrence is less frequent than akathisia.

Dystonic Syndrome — Prolonged abnormal contractions of muscle groups occur infrequently. These symptoms may be managed by the addition of a synthetic antiparkinson agent (other than L-dopa), small doses of sedative drugs, and/or reduction in dosage.

Autonomic Nervous System — Occasionally blurring of vision, tachycardia, nausea, dry mouth and salivation have been reported. Urinary retention and constipation may occur particularly if anticholinergic drugs are used to treat extrapyramidal symptoms.

Hematological — There have been rare reports of leucopenia and leucocytosis. If such reactions occur, treatment with LIDONE may continue if clinical symptoms are absent. Alterations of blood glucose, liver function tests, B.U.N., and red blood cells have not been considered clinically significant.

Metabolic and Endocrine Effects — Alteration of thyroid function has not been significant. Amenorrhea has been reported infrequently. Resumption of menses in previously amenorrheic women has been reported. Initially heavy menses may occur. Lactation associated with LIDONE therapy has not been reported. Increase in libido has been noted in some patients. Impotence has not been reported. Although both weight gain and weight loss have been in the direction of normal or ideal weight, excessive weight gain has not occurred with LIDONE.

Cardiovascular — Rare, transient, non-specific T wave changes have been reported on E.K.G. Association with a clinical syndrome has not been established. Rarely has significant hypotension been reported.

Ophthalmological — Lens opacities and pigmentary retinopathy have not been reported where patients have received LIDONE (molindone hydrochloride). In some patients, phenothiazine induced lenticular opacities have resolved following discontinuation of the phenothiazine while continuing therapy with LIDONE.

Skin — Early, non-specific skin rash, probably of allergic origin, has occasionally been reported. Skin pigmentation has not been seen with LIDONE usage alone.

LIDONE (molindone hydrochloride) has certain pharmacological similarities to other antipsychotic agents. Because adverse reactions are often extensions of the pharmacological activity of a drug, all of the known pharmacological effects associated with other antipsychotic drugs should be kept in mind when LIDONE is used. Upon abrupt withdrawal after prolonged high dosage an abstinence syndrome has not been noted.

Tardive Dyskinesia — Although rarely reported with LIDONE (molindone hydrochloride) symptoms were reversible upon discontinuation of therapy.

Tardive dyskinesia associated with other agents has appeared in some patients on long-term therapy and has also appeared after drug therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). There may be involuntary movements of extremities.

There is no known effective treatment of tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear. Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked. It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time the syndrome may not develop.

DOSAGE AND ADMINISTRATION — Initial and maintenance doses of LIDONE (molindone hydrochloride) should be individualized.

Initial Dosage Schedule

The usual starting dosage is 50 to 75 mg/day.
- — Increase to 100 mg/day in three or four days.
- — Based on severity of symptomatology, dosage may be titrated up or down depending on individual patient response.
- — An increase to 225 mg/day may be required in patients with severe symptomatology.
- — Some chronic, treatment-resistant patients may require up to 400 mg/day; (however, the long-term safety of 400 mg/day has not been established).

Elderly or debilitated patients should be started on lower dosage.

Maintenance Dosage Schedule

1. Mild — 5 mg to 15 mg three or four times a day.
2. Moderate — 10 mg to 25 mg three or four times a day.
3. Severe — 225 mg/day may be required.

Dosage may be administered once a day.

DRUG INTERACTIONS — Potentiation of drugs administered concurrently with LIDONE (molindone hydrochloride) has not been reported. Additionally, animal studies have not shown increased toxicity when LIDONE is given concurrently with representative members of three classes of drugs (i.e., barbiturates, chloral hydrate and antiparkinson drugs).

MANAGEMENT OF OVERDOSAGE — Symptomatic, supportive therapy should be the rule.

Gastric lavage is indicated for the reduction of absorption of LIDONE (molindone hydrochloride) which is freely soluble in water.

Since the adsorption of LIDONE (molindone hydrochloride) by activated charcoal has not been determined, the use of this antidote must be considered of theoretical value.

Emesis in a comatose patient is contraindicated. Additionally, while the emetic effect of apomorphine is blocked by LIDONE in animals, this blocking effect has not been determined in humans.

A significant increase in the rate of removal of unmetabolized LIDONE from the body by forced diuresis, peritoneal or renal dialysis would not be expected. (Only 2% of a single ingested dose of LIDONE is excreted unmetabolized in the urine.) However, poor response of the patient may justify use of these procedures.

While the use of laxatives or enemas might be based on general principles, the amount of unmetabolized LIDONE in feces is less than 1%. Extrapyramidal symptoms have responded to the use of diphenhydramine (Benadryl*) and the synthetic anticholinergic antiparkinson agents (i.e., Artane*, Cogentin*, Akineton*).

HOW SUPPLIED — LIDONE (molindone hydrochloride) capsules are supplied in bottles of 100 in the following dosage strengths and colors:

5 mg (**NDC** 0074-5542-13) blue and cream
10 mg (**NDC** 0074-5543-13) red and cream
25 mg (**NDC** 0074-5544-13) brown and cream

TM — Trademark
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*Artane — Trademark, Lederle Laboratories
*Cogentin — Trademark, Merck Sharp & Dohme
*Akineton — Trademark, Knoll Pharmaceutical Co.

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Significant gains are reported in control of hallucinations, conceptual disorganization, hostility, unusual thought content, emotional withdrawal.

Yet no reports of persistent tardive dyskinesia, excessive weight gain, impotence, lens opacities, skin pigmentation; and only rare cases of significant hypotension. Other side effects are similar to comparable agents.

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Vol. 136, No. 4A April 1979

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THE AMERICAN JOURNAL OF PSYCHIATRY

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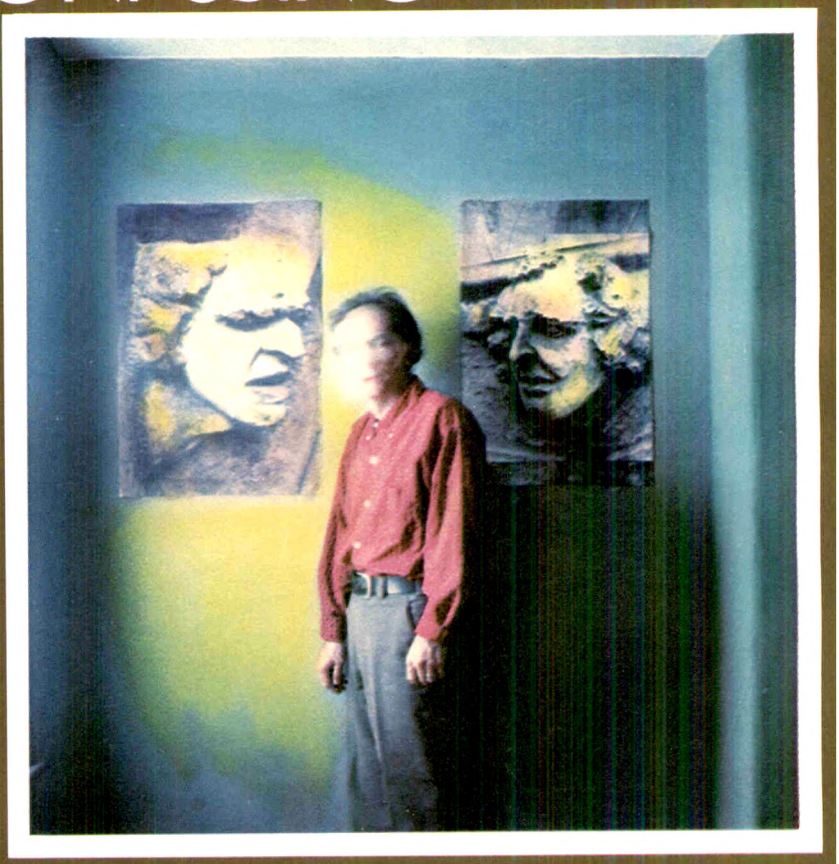
Platelet Monoamine Oxidase Activity in Schizophrenia: A Review of the Data

By Richard Jed Wyatt, Steven G. Potkin, and Dennis L. Murphy

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ADRIFT IN A SEA OF CONFUSING VOICES



'STELAZINE' HELPS FIND THE WAY BACK

The schizophrenic patient is often surrounded by confusing voices, bewildering thoughts, fright and anxiety. For such patients, 'Stelazine' provides effective, unsurpassed control of these and other psychotic symptoms.

Also important, convenient b.i.d. 'Stelazine' usually does not cause excessive sedation, which means that 'Stelazine' works with and not against your therapeutic program.

After 18 years of extensive research and clinical experience, no other antipsychotic agent has demonstrated significantly greater overall effectiveness and significantly fewer side effects than 'Stelazine'. These facts make 'Stelazine' a first choice for therapy.

Before prescribing, see complete prescribing information in SK&F literature or PDR. The following is a brief summary.

Indications

Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

Effective: For the management of the manifestations of psychotic disorders.

Possibly effective: To control excessive anxiety, tension and agitation as seen in neuroses or associated with somatic conditions.

'Stelazine' has not been shown effective in the management of behavioral complications in patients with mental retardation.

Final classification of the less-than-effective indications requires further investigation.

Contraindications: Comatose or greatly depressed states due to C.N.S. depressants; blood dyscrasias; bone marrow depression; liver damage.

Warnings: Generally avoid using in patients hypersensitive (e.g., blood dyscrasias, jaundice) to any phenothiazine. Caution patients about activities requiring alertness (e.g., operating vehicles or machinery), especially during the first few days' therapy.

Use in pregnancy only when necessary for patient's welfare.

Precautions: Use cautiously in angina. Avoid high doses and parenteral administration when cardiovascular system is impaired. Antiemetic effect may mask signs of toxic drug overdosage or physical disorders. Additive effect is possible with other C.N.S. depressants. Prolonged administration of high doses may result in cumulative effects with severe C.N.S. or vasomotor symptoms. If retinal changes occur, discontinue drug. Agranulocytosis, thrombocytopenia, pancytopenia, anemia, cholestatic jaundice, liver damage have been reported.

Patients on long-term therapy, especially high doses, should be evaluated periodically for possible adjustment or discontinuance of drug therapy.

Adverse Reactions: Drowsiness, dizziness, skin reactions, rash, dry mouth, insomnia, amenorrhea, fatigue, muscular weakness, anorexia, lactation, blurred vision. Neuromuscular (extrapyramidal) reactions: motor restlessness, dystonias, pseudo-parkinsonism, persistent tardive dyskinesia.

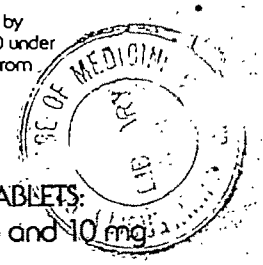
Other adverse reactions reported with Stelazine (trifluoperazine HCl, SK&F) or other phenothiazines: Some adverse effects are more frequent or intense in specific disorders (e.g., mitral insufficiency or pheochromocytoma).

Grand mal convulsions; altered cerebrospinal fluid proteins; cerebral edema; prolongation and intensification of the action of C.N.S. depressants, atropine, heat, and organophosphorus insecticides; nasal congestion, headache, nausea, constipation, obstipation, adynamic ileus, inhibition of ejaculation; reactivation of psychotic processes, catatonic-like states; hypotension (sometimes fatal); cardiac arrest; leukopenia, eosinophilia, pancytopenia, agranulocytosis, thrombocytopenic purpura; jaundice, biliary stasis; menstrual irregularities, galactorrhea, gynecomastia, false positive pregnancy tests; photosensitivity, itching, erythema, urticaria, eczema up to exfoliative dermatitis; asthma, laryngeal edema, angioneurotic edema, anaphylactoid reactions; peripheral edema; reversed epinephrine effect; hyperpyrexia; a systemic lupus erythematosus-like syndrome; pigmentary retinopathy; with prolonged administration of substantial doses, skin pigmentation, epithelial keratopathy, and lenticular and corneal deposits. EKG changes have been reported, but relationship to myocardial damage is not confirmed. Discontinue long-term, high-dose therapy gradually. NOTE: Sudden death in patients taking phenothiazines (apparently due to cardiac arrest or asphyxia due to failure of cough reflex) has been reported, but no causal relationship has been established.

Supplied: Tablets, 1 mg., 2 mg., 5 mg. and 10 mg., in bottles of 100; in Single Unit Packages of 100 (intended for institutional use only); Injection, 2 mg./mL; and Concentrate (intended for institutional use only), 10 mg./ml.

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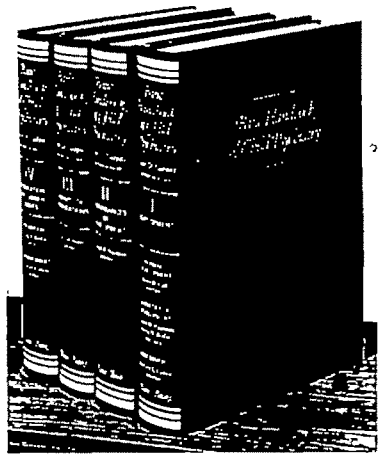
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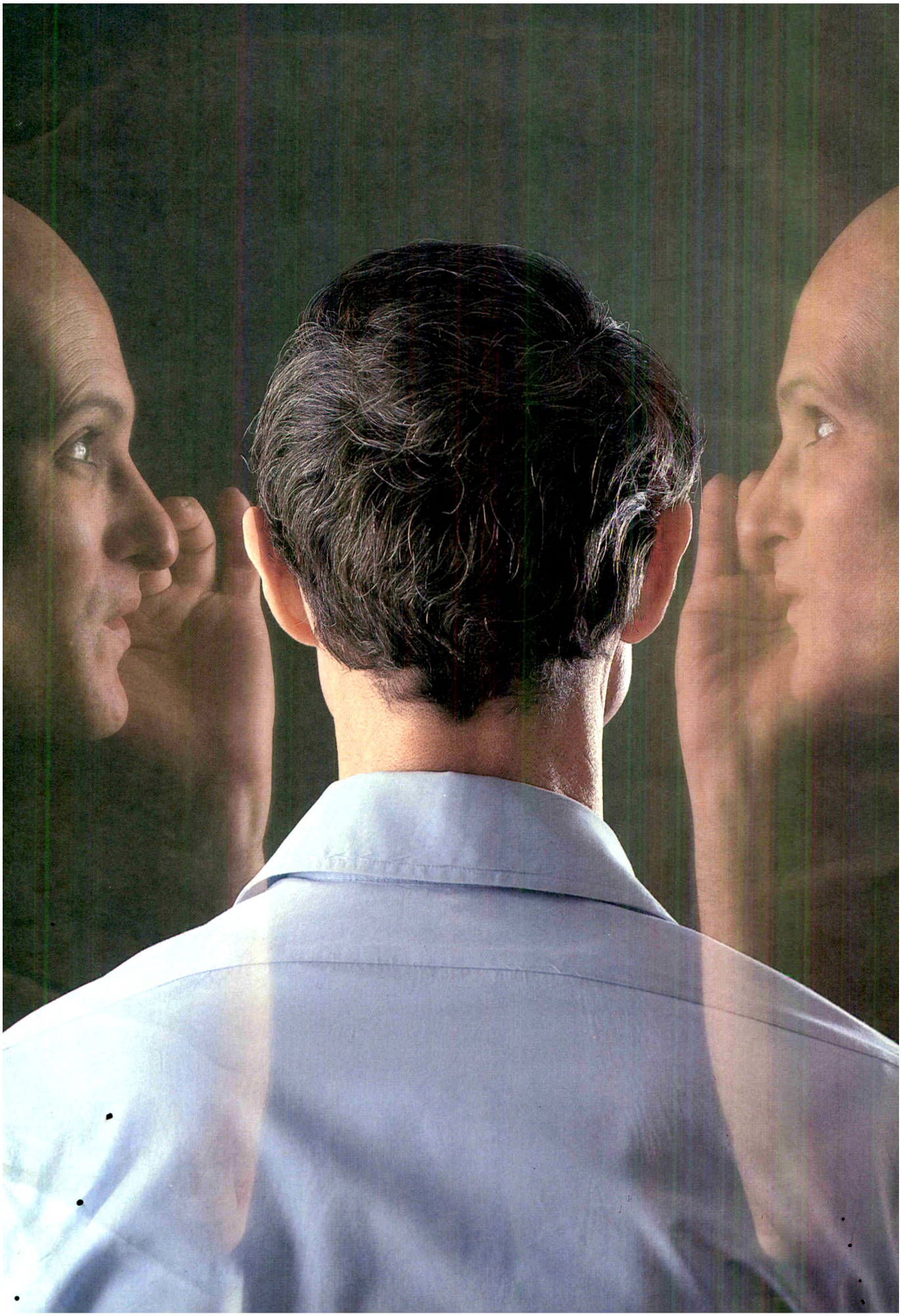
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(thiothixene hydrochloride) Concentrate: 5 mg/ml,
Intramuscular: 2 mg/ml

Contraindications. Navane is contraindicated in patients with circulatory collapse, comatose states, central nervous system depression due to any cause, and blood dyscrasias. Navane is contraindicated in individuals who have shown hypersensitivity to the drug. It is not known whether there is a cross-sensitivity between the thioxanthenes and the phenothiazine derivatives, but this possibility should be considered.

Warnings. *Usage in Pregnancy*—Safe use of Navane during pregnancy has not been established. Therefore, this drug should be given to pregnant patients only when, in the judgment of the physician, the expected benefits from the treatment exceed the possible risks to mother and fetus. Animal reproduction studies and clinical experience to date have not demonstrated any teratogenic effects.

In the animal reproduction studies with Navane, there was some decrease in conception rate and litter size, and an increase in resorption rate in rats and rabbits, changes which have been similarly reported with other psychotropic agents. After repeated oral administration to rats (5 to 15 mg/kg/day), rabbits (3 to 50 mg/kg/day), and monkeys (1 to 3 mg/kg/day) before and during gestation, no teratogenic effects were seen. (See Precautions.)

Usage in Children—The use of Navane in children under 12 years of age is not recommended because safety and efficacy in the pediatric age group have not been established.

As is true with many CNS drugs, Navane may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery, especially during the first few days of therapy. Therefore, the patient should be cautioned accordingly.

As in the case of other CNS-acting drugs, patients receiving Navane should be cautioned about the possible additive effects (which may include hypotension) with CNS depressants and with alcohol.

Precautions. An antiemetic effect was observed in animal studies with Navane; since this effect may also occur in man, it is possible that Navane may mask signs of overdosage of toxic drugs and may obscure conditions such as intestinal obstruction and brain tumor.

In consideration of the known capability of Navane and certain other psychotropic drugs to precipitate convulsions, extreme caution should be used in patients with a history of convulsive disorders or those in a state of alcohol withdrawal since it may lower the convulsive threshold. Although Navane potentiates the actions of the barbiturates, the dosage of the anticonvulsant therapy should not be reduced when Navane is administered concurrently.

Caution as well as careful adjustment of the dosage is indicated when Navane is used in conjunction with other CNS depressants other than anticonvulsant drugs.

Though exhibiting rather weak anticholinergic properties, Navane should be used with caution in patients who are known or suspected to have glaucoma, or who might be exposed to extreme heat, or who are receiving atropine or related drugs.

Use with caution in patients with cardiovascular disease.

Also, careful observation should be made for pigmentary retinopathy, and lenticular pigmentation (fine lenticular pigmentation has been noted in a small number of patients treated with Navane for prolonged periods). Blood dyscrasias (agranulocytosis, pancytopenia, thrombocytopenic purpura), and liver damage (jaundice, biliary stasis) have been reported with related drugs.

Undue exposure to sunlight should be avoided. Photosensitive reactions have been reported in patients on Navane.

Intramuscular Administration—As with all intramuscular preparations, Navane Intramuscular should be injected well within the body of a relatively large muscle. The preferred sites are the upper outer quadrant of the buttock (i.e., gluteus maximus) and the mid-lateral thigh.

The deltoid area should be used only if well developed, such as in certain adults and older children, and then only with caution to avoid radial nerve injury. Intramuscular injections should not be made into the lower and mid-thirds of the upper arm. As with all intramuscular injections, aspiration is necessary to help avoid inadvertent injection into a blood vessel.

Adverse Reactions. *Note:* Not all of the following adverse reactions have been reported with Navane (thiothixene). However, since Navane has certain chemical and pharmacologic similarities to the phenothiazines, all of the known side effects and toxicity associated with phenothiazine therapy should be borne in mind when Navane is used.

Cardiovascular effects: Tachycardia, hypotension, lightheadedness, and syncope. In the event hypotension occurs, epinephrine should not be used as a pressor agent since a paradoxical further lowering of blood pressure may result. Nonspecific EKG changes have been observed in some patients receiving Navane. These changes are usually reversible and frequently disappear on continued Navane therapy. The incidence of these changes is lower than that observed with some phenothiazines. The clinical significance of these changes is not known.

CNS effects: Drowsiness, usually mild, may occur although it usually subsides with continuation of Navane therapy. The incidence of sedation appears similar to that of the piperazine group of phenothiazines, but less than that of certain aliphatic phenothiazines. Restlessness, agitation and insomnia have been noted with Navane (thiothixene). Seizures and paradoxical exacerbation of psychotic symptoms have occurred with Navane infrequently.

Hyperreflexia has been reported in infants delivered from mothers having received structurally related drugs.

In addition, phenothiazine derivatives have been associated with cerebral edema and cerebrospinal fluid abnormalities.

Extrapyramidal symptoms, such as pseudo-parkinsonism, akathisia, and dystonia have been reported. Management of these extrapyramidal symptoms depends upon the type and severity. Rapid relief of acute symptoms may require the use of an injectable antiparkinson agent. More slowly emerging symptoms may be managed by reducing the dosage of Navane and/or administering an oral antiparkinson agent.

Persistent Tardive Dyskinesia: As with all antipsychotic agents tardive dyskinesia may appear in some patients on long term therapy or may occur after drug therapy has been discontinued. The risk seems to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities.

There is no known effective treatment for tardive dyskinesia: antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear.

Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked.

It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time, the syndrome may not develop.

Hepatic effects: Elevations of serum transaminase and alkaline phosphatase, usually transient, have been infrequently observed in some patients. No clinically confirmed cases of jaundice attributable to Navane have been reported.

Hematologic effects: As is true with certain other psychotropic drugs, leukopenia and leukocytosis, which are usually transient, can occur occasionally with Navane. Other antipsychotic drugs have been associated with agranulocytosis, eosinophilia, hemolytic anemia, thrombocytopenia and pancytopenia.

Allergic reactions: Rash, pruritus, urticaria, photosensitivity and rare cases of anaphylaxis have been reported with Navane. Undue exposure to sunlight should be avoided. Although not experienced with Navane, exfoliative dermatitis and contact dermatitis (in nursing personnel) have been reported with certain phenothiazines.

Endocrine disorders: Lactation, moderate breast enlargement and amenorrhea have occurred in a small percentage of females receiving Navane. If persistent, this may necessitate a reduction in dosage or the discontinuation of therapy. Phenothiazines have been associated with false positive pregnancy tests, gynecomastia, hypoglycemia, hyperglycemia, and glycosuria.

Autonomic effects: Dry mouth, blurred vision, nasal congestion, constipation, increased sweating, increased salivation, and impotence have occurred infrequently with Navane therapy. Phenothiazines have been associated with miosis, mydriasis, and adynamic ileus.

Other adverse reactions: Hyperpyrexia, anorexia, nausea, vomiting, diarrhea, increase in appetite and weight, weakness or fatigue, polydipsia and peripheral edema.

Although not reported with Navane, evidence indicates there is a relationship between phenothiazine therapy and the occurrence of a systemic lupus erythematosus-like syndrome.

NOTE: Sudden deaths have occasionally been reported in patients who have received certain phenothiazine derivatives. In some cases the cause of death was apparently cardiac arrest or asphyxia due to failure of the cough reflex. In others, the cause could not be determined nor could it be established that death was due to phenothiazine administration.

Dosage and Administration. Dosage of Navane should be individually adjusted depending on the chronicity and severity of the condition. In general, small doses should be used initially and gradually increased to the optimal effective level, based on patient response.

Some patients have been successfully maintained on once-a-day Navane therapy.

Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

Navane Intramuscular Solution—For Intramuscular Use Only. Where more rapid control and treatment of acute behavior is desirable, the intramuscular form of Navane may be indicated. It is also of benefit where the very nature of the patient's symptomatology, whether acute or chronic, renders oral administration impractical or even impossible.

For treatment of acute symptomatology or in patients unable or unwilling to take oral medication, the usual dose is 4 mg of Navane Intramuscular administered 2 to 4 times daily. Dosage may be increased or decreased depending on response. Most patients are controlled on a total daily dosage of 16 to 20 mg. The maximum recommended dosage is 30 mg/day. An oral form should supplant the injectable form as soon as possible. It may be necessary to adjust the dosage when changing from the intramuscular to oral dosage forms. Dosage recommendations for Navane Capsules and Concentrate appear in the following paragraphs.

Navane Capsules; Navane Concentrate—In milder conditions, an initial dose of 2 mg three times daily. If indicated, a subsequent increase to 15 mg/day total daily dose is often effective.

In more severe conditions, an initial dose of 5 mg twice daily.

The usual optimal dose is 20 to 30 mg daily. If indicated, an increase to 60 mg/day total daily dose is often effective. Exceeding a total daily dose of 60 mg rarely increases the beneficial response.

Overdosage. Manifestations include muscular twitching, drowsiness, and dizziness. Symptoms of gross overdosage may include CNS depression, rigidity, weakness, torticollis, tremor, salivation, dysphagia, hypotension, disturbances of gait, or coma.

Treatment: Essentially symptomatic and supportive. For Navane oral, early gastric lavage is helpful. For Navane oral and Intramuscular, keep patient under careful observation and maintain an open airway, since involvement of the extrapyramidal system may produce dysphagia and respiratory difficulty in severe overdosage. If hypotension occurs, the standard measures for managing circulatory shock should be used (I.V. fluids and/or vasoconstrictors).

If a vasoconstrictor is needed, levarterenol and phenylephrine are the most suitable drugs. Other pressor agents, including epinephrine, are not recommended, since phenothiazine derivatives may reverse the usual pressor action of these agents and cause further lowering of blood pressure.

If CNS depression is present, recommended stimulants include amphetamine, dextroamphetamine, or caffeine and sodium benzoate. Picrotoxin or pentylenetetrazol should be avoided. Extrapyramidal symptoms may be treated with antiparkinson drugs.

There are no data on the use of peritoneal or hemodialysis, but they are known to be of little value in phenothiazine intoxication.

How Supplied. Navane (thiothixene) is available as capsules containing 1 mg, 2 mg, 5 mg, and 10 mg of thiothixene in bottles of 100 and 1,000. Navane is also available as capsules containing 20 mg of thiothixene in bottles of 100 and 500.

Navane (thiothixene hydrochloride) Concentrate is available in 120 ml (4 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, 5 mg, 6 mg, 8 mg, and 10 mg, and in 30 ml (1 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, and 5 mg. Each ml contains thiothixene hydrochloride equivalent to 5 mg of thiothixene. Contains alcohol, U.S.P. 7.0% v/v (small loss unavoidable).

Navane (thiothixene hydrochloride) Intramuscular solution is available in a 2 ml amber glass vial in packages of 10. Each ml contains thiothixene hydrochloride equivalent to 2 mg of thiothixene, dextrose 5% w/v, benzyl alcohol 0.9% w/v, and propyl gallate 0.02% w/v.

References. 1. Stotsky BA: Relative efficacy of parenteral haloperidol and thiothixene for the emergency treatment of acutely excited and agitated patients. *Dis Nerv Syst* 38:967-973, 1977. 2. Bressler B, Friedel RO: A comparison between chlorpromazine and thiothixene in a Veterans Administration hospital population. *Psychosomatics* 12:275-277, 1971. 3. Denber HCB, Turns D: Double blind comparison of thiothixene and trifluoperazine in acute schizophrenia. *Psychosomatics* 13:100-104, 1972. 4. Sterlin C, Ban TA, Lehman HE, Saxena BM: The place of thiothixene in the treatment of schizophrenic patients. *Can Psychiatr Assoc J* 15:3-4, 1970. 5. Brauzer B, Goldstein BJ: Comparative effects of intramuscular thiothixene and trifluoperazine in psychotic patients. *J Clin Pharmacol* 8:400-403, 1968. 6. Engelhardt DM, Rudorfer L: The chronic schizophrenic outpatient in an urban community: Social and vocational adjustment. Presented as a Scientific Exhibit at the 130th Annual Meeting, American Psychiatric Association, Toronto, Canada, May 2-6, 1977. 7. Itil TM, Unverdi C, Wohlrabe J, Larsen V, Levitt J: Drug therapy of psychosis associated with organic brain syndrome, presented as a scientific exhibit at the American Public Health Association Centennial, Atlantic City, New Jersey, November 12-16, 1972. 8. Dillenkofter RL, et al: Electrocardiographic evaluations of schizophrenic patients. Presented as a Scientific Exhibit at the 125th Annual Meeting of the American Psychiatric Association, Dallas, Texas, May 1-4, 1972. 9. Goldstein B, Weiner D, Banas F: Clinical evaluation of thiothixene in chronic ambulatory schizophrenic patients, in Lehmann HE, Ban TA (eds): *Modern Problems in Pharmacopsychiatry*. Basel, S Karger, 1969, vol 2, pp 45-52.

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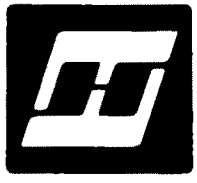
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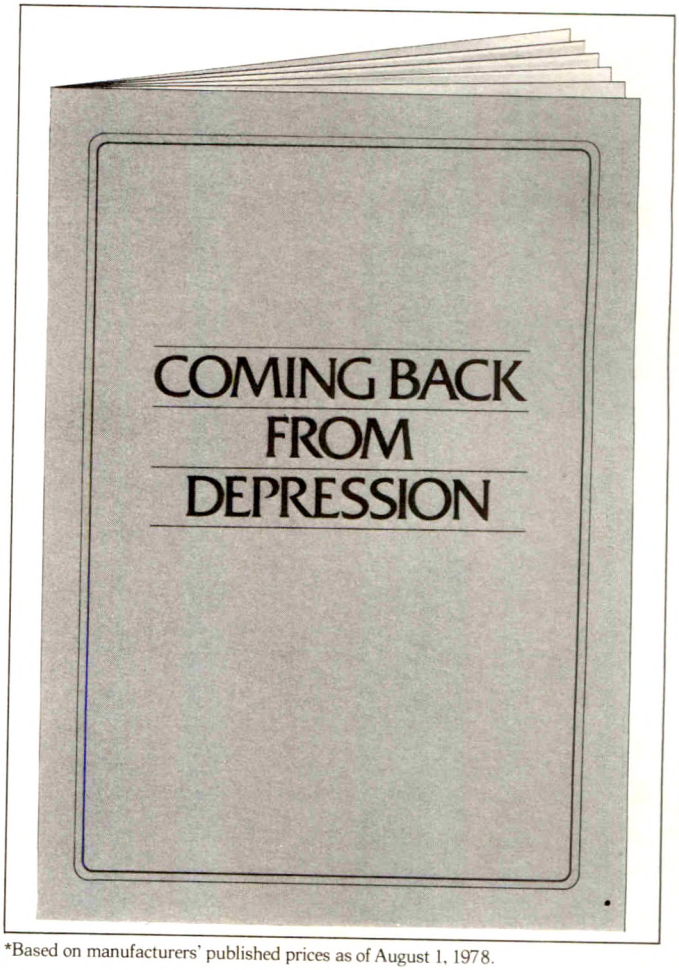
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INDICATIONS: For relief of symptoms of depression alone or accompanied by anxiety. Endogenous depression is more likely to be alleviated than are other depressive states.

CONTRAINDICATIONS: Contraindicated in patients with a history of hypersensitivity to amitriptyline. Do not administer concomitantly with a monamine oxidase (MAO) inhibitor since hyperpyretic crises, severe convulsions, and deaths occurred when tricyclic antidepressants and MAO inhibitors were administered simultaneously. When an MAO inhibitor is to be replaced by amitriptyline, allow 14 days to elapse after discontinuation of the former and then initiate amitriptyline cautiously and gradually increase dosage until optimum response is achieved. Amitriptyline is not recommended for use during the acute recovery phase following myocardial infarction.

WARNINGS: Amitriptyline may block antihypertensive action of guanethidine or similarly acting drugs. Use with caution in patients with history of seizures and (because of atropine-like action of amitriptyline) in patients with history of narrow-angle glaucoma (even average doses may precipitate an attack), increased intraocular pressure, or urinary retention. Closely watch patients with cardiovascular disorders since, in addition to having caused myocardial infarction and stroke, tricyclic antidepressants (including amitriptyline) particularly with high dosage have been reported to produce arrhythmias, sinus tachycardia, and prolongation of conduction time. Close supervision is required when amitriptyline is given to hyperthyroid patients or those on thyroid medication. Amitriptyline may impair mental and/or physical abilities required for performance of hazardous tasks such as operating machinery or driving a motor vehicle. Bear in mind that in patients who may use alcohol excessively the potentiation may increase the danger inherent in any suicide attempt or overdosage.

Usage in Pregnancy: Safe use during pregnancy and lactation has not been established; therefore, in administering the drug to pregnant patients, nursing mothers, or women who may become pregnant, weigh the possible benefits against the possible hazards to the mother and child. Animal reproduction studies have been inconclusive, and clinical experience has been limited.

Usage in Children: At the present time, not recommended for patients under 12 years of age because of lack of experience with use in children.

PRECAUTIONS: Schizophrenic patients may develop increased symptoms of psychosis; patients with paranoid symptomatology may have an exaggeration of such symptoms; manic depressive patients may experience a shift to the manic phase. In these circumstances the dose of amitriptyline may be reduced or a major tranquilizer may be administered concurrently.

Closely supervise and carefully adjust dosage in concomitant use with anticholinergic or sympathomimetic drugs, including combination of epinephrine and local anesthetics. Paralytic ileus may occur with concomitant use of tricyclic antidepressants and anticholinergic-type drugs. Caution is advised if used concurrently with large doses of ethchlorvynol since transient delirium has been reported when one gram of that drug and 75 to 150 mg of amitriptyline HCl were administered. Amitriptyline may enhance response to alcohol and the effects of barbiturates and other CNS depressants. The possibility of suicide in depressed patients remains during treatment and until significant remission occurs. Potentially suicidal patients should not have easy access to large quantities of the drug. Prescriptions should be written for the smallest amount feasible. Limit concurrent administration of this drug and electroshock therapy to patients for whom it is essential since the hazards associated with such therapy may be increased. Discontinue this drug, when possible, several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported. Use amitriptyline with caution in presence of impaired liver function.

ADVERSE REACTIONS: NOTE: A few of the adverse reactions listed below have not been reported with this specific drug, but each of the reactions should be considered when administering amitriptyline because of pharmacological similarities among tricyclic antidepressants.

Cardiovascular: Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, and stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness; tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; and syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, increased intraocular pressure, constipation, paralytic ileus, urinary retention, and dilatation of the urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, and edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, leukopenia, eosinophilia, purpura, and thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, and black tongue. Rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, and elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, and alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache, and malaise. These are not indicative of addiction.

For full prescribing information, consult package insert.

HOW SUPPLIED: Available for oral administration in tablets providing 10, 25, 50, 75, and 100 mg amitriptyline hydrochloride. The 10, 25, and 50 mg tablets are available in bottles of 100 and 1000. The 75 and 100 mg tablets are available in bottles of 100.

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Neither should it be used in the child who exhibits symptoms secondary to environmental factors and/or primary psychiatric disorders, including psychosis.

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(pemoline)

Prescribing Information

Indications: *MINIMAL BRAIN DYSFUNCTION IN CHILDREN*—as adjunctive therapy to other remedial measures (psychological, educational, social).

Special Diagnostic Considerations: The cause of minimal brain dysfunction (MBD) is unknown. Diagnosis of MBD involves the use of medical, psychological, educational, and social tools, since no single diagnostic test is adequate.

MBD is characterized by chronic moderate to severe hyperactivity, short attention span, distractibility, emotional lability, and impulsivity. Nonlocalizing (soft) neurological signs, learning disability, and abnormal EEG may or may not be present. The diagnosis of MBD must be based upon a complete history and evaluation of the child and not solely on the presence of one or more of these characteristics.

Drug treatment is not indicated for all children with MBD. In the primary therapy of MBD, appropriate educational placement is essential and psychosocial intervention is generally necessary. When these measures alon are insufficient, the decision to prescribe stimulant medication will depend upon the physician's assessment of the chronicity and severity of the child's symptoms. Stimulants are not intended for use in the child who exhibits symptoms secondary to environmental factors and/or primary psychiatric disorders, including psychosis.

Contraindications: Cylert (pemoline) is contraindicated in patients with known hypersensitivity or idiosyncrasy to the drug. (See ADVERSE REACTIONS.)

Warnings: Cylert is not recommended for children less than 6 years of age since its safety and efficacy in this age group have not been established.

Sufficient data on the safety and the efficacy of the long-term use of Cylert in children with minimal brain dysfunction are not yet available.

A temporary suppression of the predicted growth rate (weight and/or height gain) has been reported for children receiving long-term stimulant therapy. A definite causal relationship between stimulant drugs and this finding has not been established.

Precautions: Liver function tests should be performed periodically during therapy with Cylert. The drug should be discontinued if abnormalities are revealed and confirmed by follow-up tests. (See ADVERSE REACTIONS regarding reports of abnormal liver function tests and jaundice.)

Cylert should be administered with caution to patients with significantly impaired hepatic or renal function.

The interaction of Cylert with other drugs has not been studied in humans Patients who are receiving Cylert concurrently with other drugs, especially drugs with CNS activity, should be monitored carefully.

Cylert failed to demonstrate a potential for self-administration in primates However, the pharmacologic similarity of pemoline to other psychostimulants with known dependence liability suggests that psychological and/or physical dependence might also occur with Cylert. There have been isolated reports of transient psychotic symptoms occurring in adults following the long-term misuse of excessive oral doses of pemoline Cylert should be given with caution to emotionally unstable patients who may increase the dosage on their own initiative.

Usage during Pregnancy and Lactation: The safety of Cylert (pemoline) fo use during pregnancy and lactation has not been established.

Fertility, reproduction, and teratology studies were conducted in laborator animals. Pemoline, in doses of 18.75 or 37.5 mg./kg./day, had no effect on th fertility of male or female rats. The drug, when given to pregnant rats (from gestation day 15 through weaning) and to rabbits (from gestation days 6-18) at these same dosage levels, produced no teratogenic or embryotoxic effects, and had no effect on the viability of the young at birth. However, increased incidences of stillbirths and cannibalization were observed when pemoline was given to rats at these dosage levels, beginning 14 days prior to conceptio

Adverse Reactions: Insomnia is the most frequently reported side effect c Cylert; it usually occurs early in therapy, prior to an optimum therapeutic response. In the majority of cases it is transient in nature or responds to a reduction in dosage.

Anorexia with weight loss may occur during the first weeks of therapy. In the majority of cases it is transient in nature; weight gain usually resumes within three to six months.

Stomach ache, skin rashes, increased irritability, mild depression, nausea, dizziness, headache, drowsiness, and hallucinations have been reported.

Elevations of SGOT, SGPT, and serum LDH have occurred in patients taking Cylert, usually after several months of therapy. These effects appear t be reversible upon withdrawal of the drug, and are thought to be manifestations of a delayed hypersensitivity reaction. There have also been a few reports of jaundice occurring in patients taking Cylert; a causal relationship between the drug and this clinical finding has not been established.

There have been reports of dyskinetic movements of the lips, face, and extremities occurring with the use of Cylert. Convulsive seizures have also been reported. A definite causal relationship between Cylert and these reactions has not been established.

Mild adverse reactions appearing early during the course of treatment with Cylert often remit with continuing therapy. If adverse reactions are of a significant or protracted nature, dosage should be reduced or the drug discontinued.

How Supplied: Cylert (pemoline) is supplied as monogrammed, grooved tablets in three dosage strengths:

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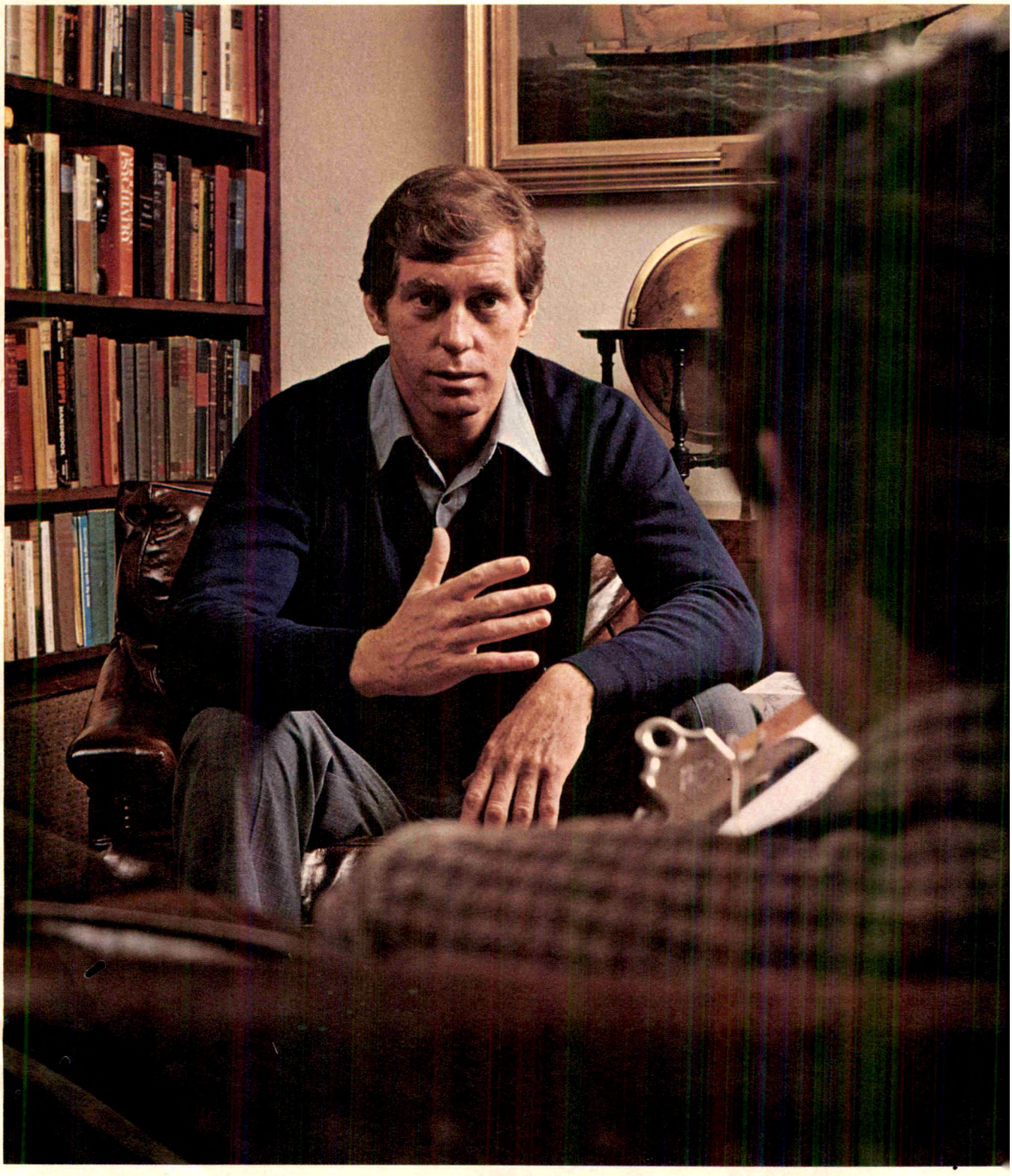
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(AMITRIPTYLINE HCl | MSD)

helping relieve the symptoms of clinically significant depression

MSD
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Contraindications: Known hypersensitivity. Should not be given concomitantly with a monoamine oxidase inhibitor since hyperpyretic crises, severe convulsions, and deaths have occurred. When used to replace a monoamine oxidase inhibitor, allow a minimum of 14 days to elapse before initiating therapy with amitriptyline HCl. Initiate dosage of amitriptyline HCl cautiously with gradual increase in dosage until optimum response is achieved. Not recommended during the acute recovery phase following myocardial infarction.

Warnings: May block the antihypertensive action of guanethidine or similarly acting compounds. Should be used with caution in patients with a history of seizures or a history of urinary retention, or with angle-closure glaucoma or increased intraocular pressure; in patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely; arrhythmias, sinus tachycardia and prolongation of the conduction time have been reported, particularly with high doses; myocardial infarction and stroke have been reported with drugs of this class. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. May impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle. In patients who use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage. Safe use during pregnancy and lactation has not been established; in pregnant patients, nursing mothers, or women who may become pregnant, weigh possible benefits against possible hazards to mother and child. Not recommended for patients under 12 years of age.

Precautions: Schizophrenic patients may develop increased symptoms of psychosis; patients with paranoid symptomatology may have an exaggeration of such symptoms; manic depressive patients may experience a shift to the manic phase. In these circumstances, the dose of amitriptyline HCl may be reduced or a major tranquilizer, such as perphenazine, may be administered concurrently.

When given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required; paralytic ileus may occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs. Use cautiously in patients receiving large doses of ethchlorvynol, since transient delirium has been reported on concurrent administration. May enhance the response to alcohol and the effects of barbiturates and other CNS depressants. The possibility of suicide in depressed patients remains until significant remission occurs. Potentially suicidal patients should not have access to large quantities of this drug. Prescriptions should be written for the smallest amount feasible. Concurrent electroshock therapy may increase the hazards associated with such therapy; such treatment should be limited to patients for whom it is essential. When possible, discontinue the drug several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported. Use with caution in patients with impaired liver function.

Adverse Reactions: *Note:* Included in this listing are a few adverse reactions not reported with this specific drug. However, pharmacological similarities among the tricyclic antidepressant drugs require that each reaction be considered when amitriptyline is administered. *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, increased intraocular pressure, constipation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, leukopenia, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, black tongue, rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache, and malaise; these are not indicative of addiction.

Overdosage: Hospitalize as soon as possible all patients suspected of having taken an overdose. Treatment is symptomatic and supportive. In addition, the intravenous administration of 1 to 3 mg physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage should be repeated as required, particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine.

How Supplied: Tablets containing 10 mg and 25 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100, 1000, and 5000; tablets containing 50 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100 and 1000; tablets containing 75 mg and 100 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100; tablets containing 150 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 30 and 100; for intramuscular use, in 10-ml vials containing per ml: 10 mg amitriptyline HCl, 44 mg dextrose, 1.5 mg methylparaben and 0.2 mg propylparaben as preservatives, and water for injection q.s. 1 ml.

For more detailed information, consult your MSD representative or see full prescribing information. Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa. 19486

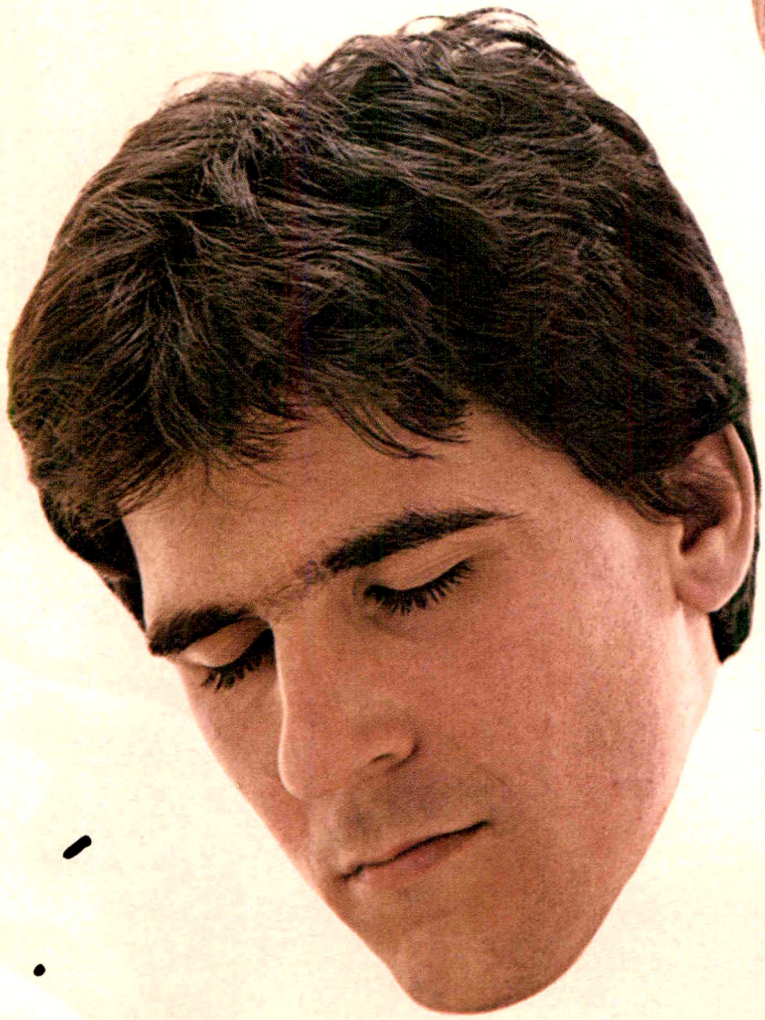
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For the aged person suffering from withdrawn or apathetic senile behavior, Ritalin can often help relieve bothersome symptoms. In one double-blind study[1] of withdrawn, apathetic geriatric patients, Ritalin elicited notable improvement in competence, interest and retardation. While no side effects were reported in this study, blood pressure and pulse variations were noted in both groups but were not considered of exceptional magnitude.





Help for narcoleptic patients

The narcoleptic patient can fall asleep anytime...anyplace... and the consequences can be grave, or at the least, embarrassing. Although there is no known cure for narcolepsy, Ritalin acts promptly to counteract sleepiness and is the medication most frequently used by cited investigators.[2,3] Occasional problems of tolerance may be managed by complete withdrawal of Ritalin during vacation or holidays, then reinstating the drug at lower dosage.[3]

*Ritalin has been evaluated as possibly effective in these indications.

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As Ritalin acts promptly to relieve symptoms of mild depression, mood and outlook usually improve, and the patient can cope again. Generally, one prescription is sufficient. Ritalin is contraindicated in severe depression.

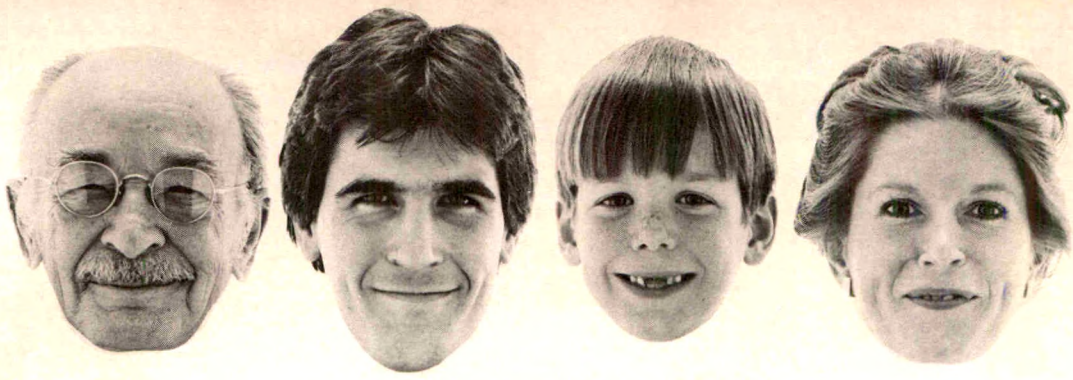


Help for the MBD child

For the child with Minimal Brain Dysfunction (MBD), adjunctive Ritalin is often highly effective when medication is needed to help reduce symptoms such as distractibility,[4] disorganized behavior,[4] and hyperactivity.[4] MBD children responding to Ritalin usually exhibit better classroom performance and improved interpersonal relationships. Moreover, as MBD symptoms are curbed, the affected child often responds more positively to nonpharmacologic modalities.[5] Ritalin should be periodically discontinued to assess the child's condition. Improvement may be sustained when the drug is temporarily or permanently discontinued.

Please turn page for prescribing information.

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RITALIN® C
(methylphenidate)
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Ritalin® hydrochloride C
(methylphenidate hydrochloride USP)

TABLETS

INDICATIONS
Based on a review of this drug by the National Academy of Sciences-National Research Council and/or other information, FDA has classified the indications as follows:
Effective: Minimal Brain Dysfunction in Children—as adjunctive therapy to other remedial measures (psychological, educational, social)
Special Diagnostic Considerations
Specific etiology of Minimal Brain Dysfunction (MBD) is unknown, and there is no single diagnostic test. Adequate diagnosis requires the use not only of medical but of special psychological, educational, and social resources.
Characteristics commonly reported include: chronic history of short attention span, distractibility, emotional lability, impulsivity, and moderate to severe hyperactivity; minor neurological signs and abnormal EEG. Learning may or may not be impaired. The diagnosis of MBD must be based upon a complete history and evaluation of the child and not solely on the presence of one or more of these characteristics.
Drug treatment is not indicated for all children with MBD. Stimulants are not intended for use in the child who exhibits symptoms secondary to environmental factors and/or primary psychiatric disorders, including psychosis.
Appropriate educational placement is essential and psychosocial intervention is generally necessary. When remedial measures alone are insufficient, the decision to prescribe stimulant medication will depend upon the physician's assessment of the chronicity and severity of the child's symptoms.
Effective: Narcolepsy
"Possibly" effective: Mild Depression; Apathetic or Withdrawn Senile Behavior
Final classification of the less-than-effective indications requires further investigation.

CONTRAINDICATIONS
Marked anxiety, tension, and agitation are contraindications to Ritalin, since the drug may aggravate these symptoms. Ritalin is contraindicated also in patients known to be hypersensitive to the drug and in patients with glaucoma.

WARNINGS
Ritalin should not be used in children under six years, since safety and efficacy in this age group have not been established.
Sufficient data on safety and efficacy of long-term use of Ritalin in children with minimal brain dysfunction are not yet available. Although a causal relationship has not been established, suppression of growth (ie, weight gain, and/or height) has been reported with the long-term use of stimulants in children. Therefore, patients requiring long-term therapy should be carefully monitored.
Ritalin should not be used for severe depression of either exogenous or endogenous origin.
Ritalin should not be used for the prevention or treatment of normal fatigue states.
There is some clinical evidence that Ritalin may lower the convulsive threshold in patients with prior history of seizures, with prior EEG abnormalities in absence of seizures, and, very rarely, in absence of history of seizures and no prior EEG evidence of seizures. Safe concomitant use of anticonvulsants and Ritalin has not been established. In the presence of seizures, the drug should be discontinued.
Use cautiously in patients with hypertension. Blood pressure should be monitored at appropriate intervals in all patients taking Ritalin, especially those with hypertension.
Symptoms of visual disturbances have been encountered in rare cases. Difficulties with accommodation and blurring of vision have been reported.

Drug Interactions
Ritalin may decrease the hypotensive effect of guanethidine. Use cautiously with pressor agents and MAO inhibitors.
Human pharmacologic studies have shown that Ritalin may inhibit the metabolism of coumarin anticoagulants, anticonvulsants (phenobarbital, diphenylhydantoin, primidone), phenylbutazone, and tricyclic antidepressants (imipramine, desipramine). Downward dosage adjustments of these drugs may be required when given concomitantly with Ritalin.

Usage in Pregnancy
Adequate animal reproduction studies to establish safe use of Ritalin during pregnancy have not been conducted. Therefore, until more information is available, Ritalin should not be prescribed for women of childbearing age unless, in the opinion of the physician, the potential benefits outweigh the possible risks.

Drug Dependence
Ritalin should be given cautiously to emotionally unstable patients, such as those with a history of drug dependence or alcoholism, because such patients may increase dosage on their own initiative.
Chronically abusive use can lead to marked tolerance and psychic dependence with varying degrees of abnormal behavior. Frank psychotic episodes can occur, especially with parenteral abuse. Careful supervision is required during drug withdrawal, since severe depression as well as the effects of chronic overactivity can be unmasked. Long-term follow-up may be required because of the patient's basic personality disturbances.

PRECAUTIONS
Patients with an element of agitation may react adversely; discontinue therapy if necessary.
Periodic CBC, differential, and platelet counts are advised during prolonged therapy.

ADVERSE REACTIONS
Nervousness and insomnia are the most common adverse reactions but are usually controlled by reducing dosage and omitting the drug in the afternoon or evening. Other reactions include hypersensitivity (including skin rash, urticaria, fever, arthralgia, exfoliative dermatitis, erythema multiforme with histopathological findings of necrotizing vasculitis, and thrombocytopenic purpura); anorexia; nausea; dizziness; palpitations; headache; dyskinesia; drowsiness; blood pressure and pulse changes, both up and down; tachycardia; angina; cardiac arrhythmia; abdominal pain; weight loss during prolonged therapy. Toxic psychosis has been report
Although a definite causal relationship has not be established, the following have been reported in tients taking this drug: leukopenia and/or anemia few instances of scalp hair loss.
In children, loss of appetite, abdominal pain, wei loss during prolonged therapy, insomnia, and tachycardia may occur more frequently; however any of the other adverse reactions listed above n also occur.

DOSAGE AND ADMINISTRATION
Adults
Administer orally in divided doses 2 or 3 times da preferably 30 to 45 minutes before meals. Dosag will depend upon indication and individual response.
Average dosage is 20 to 30 mg daily. Some patie may require 40 to 60 mg daily. In others, 10 to 15 daily will be adequate. Patients who are unable t sleep if medication is taken late in the day should take the last dose before 6 p.m.

Children with Minimal Brain Dysfunction
(6 years and over)
Start with small doses (*eg*, 5 mg before breakfas and lunch) with gradual increments of 5 to 10 mg weekly. Daily dosage above 60 mg is not recommended. If improvement is not observed after appropriate dosage adjustment over a one-mont period, the drug should be discontinued.
If paradoxical aggravation of symptoms or other verse effects occur, reduce dosage, or, if necess discontinue the drug.
Ritalin should be periodically discontinued to as the child s condition. Improvement may be sustained when the drug is either temporarily or per nently discontinued.
Drug treatment should not and need not be indef and usually may be discontinued after puberty.

HOW SUPPLIED
Tablets, 20 mg (peach, scored): bottles of 100 ar 1000.
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Tablets, 5 mg (pale yellow): bottles of 100, 500 ar 1000.

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C I B

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D. R. Lipsitt, Chief of Psychiatry, Mount Auburn Hospital, Cambridge, MA

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General Hospital Psychiatry explores the linkages and interfaces between **Psychiatry, Medicine and Primary Care.** The Journal emphasizes a biopsychosocial approach to illness and health, and provides a forum for communication among professionals with clinical, academic and research interests in psychiatry's essential function in "the mainstream of medicine." Building upon those liaison-consultation and psychiatric services which have burgeoned in the general hospital setting, the Journal expands beyond this base to encourage new contributions to the understanding and treatment of illness-in in-patient, ambulatory, and community settings. Studies of multi-system relationships of life stresses, physical experience, psychosocial factors, interpersonal and intrapsychic reactions, family structure, ecological change, and institutional forces are especially relevant to the Journal's objectives.

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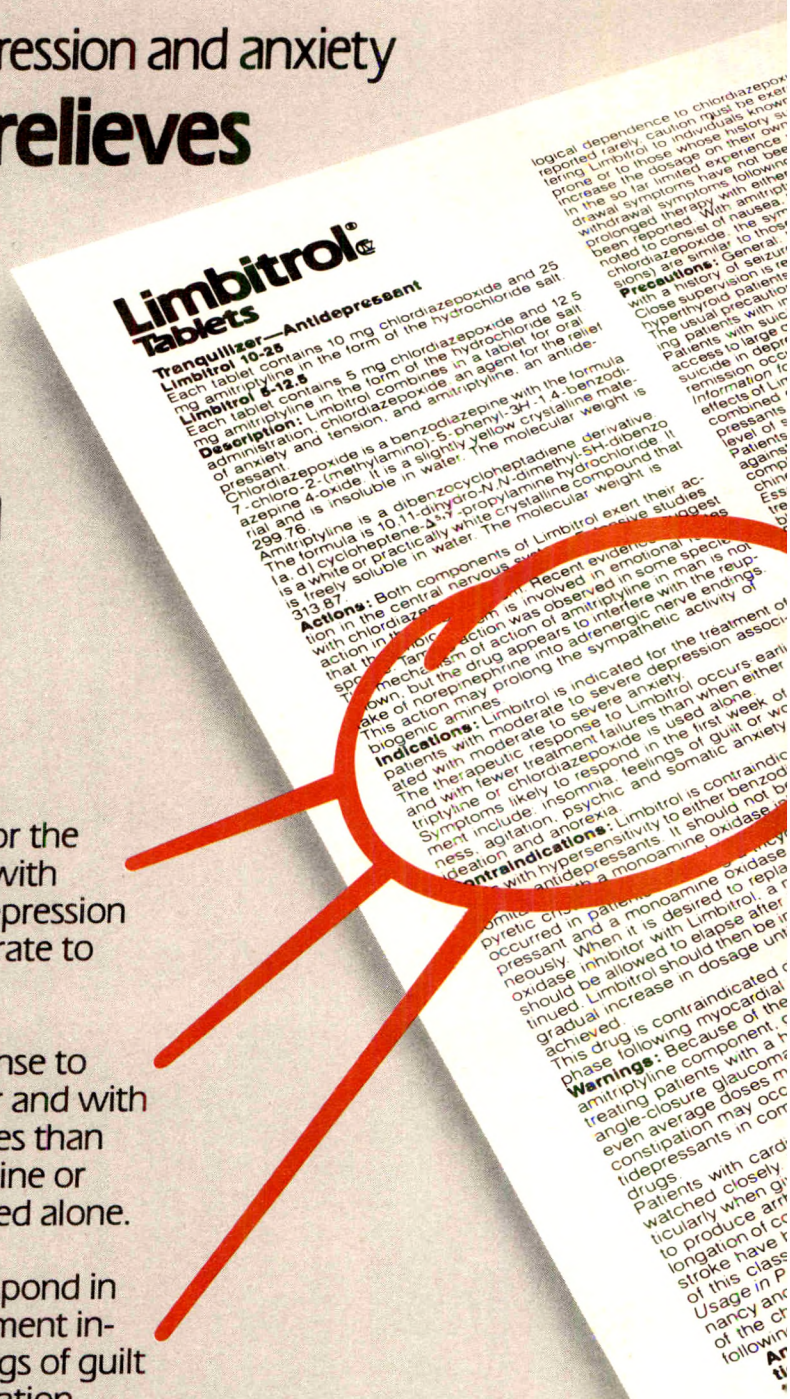
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nifestations: High doses may cause drowsiness
mporary confusion, disturbed concentration or tran-
ient visual hallucinations. Overdosage may cause
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evidence of impaired conduction (such as bundle
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convulsions, severe hypotension, stupor and coma.
Other symptoms may be agitation, hyperactive reflexes,
muscle rigidity, vomiting, hyperpyrexia or any of those
listed under Adverse Reactions.
Treatment: Empty the stomach as quickly as possible by
emesis or lavage. In the comatose patient a cuff
endotracheal tube should be placed in position prior to
either of these measures. The instillation of activated
charcoal into the stomach also should be considered. If
the patient is stuporous but responds to stimuli, only
essential maintenance may be required. It
close observation and nursing care may be required. If
an adequate airway and fluid in-
take. Body temperature should be watched closely and
appropriate measures taken should deviations
occur.
The intramuscular or slow intravenous administration of
1 to 3 mg in adults (or 0.5 mg in children) of physo-
stigmine salicylate (Antilirium®) has been reported to
reverse the manifestations of amitriptyline overdosage.
Because of its relatively short half-life, additional doses
may be needed at intervals of 30 minutes to 2 hours.
Convulsions may be treated by the use of an inhalation
anesthetic rather than the use of barbiturates. Cardiac
monitoring is advisable, and the cautious use of digitalis
or other antiarrhythmic agents should be considered if
serious cardiovascular abnormalities occur. Serum
potassium levels should be monitored and kept within
normal limits by the use of appropriate I.V. fluids. Plasma ex-
panders and corticosteroids may be used to control
circulatory shock.
Dialysis is unlikely to be of value, as it has not proven
useful in overdosages of either amitriptyline or chlor-
diazepoxide. Since many suicidal attempts involve mul-
tiple drugs including barbiturates, the possibility of
dialysis being beneficial for removal of other drugs
should not be overlooked.
Treatment should be continued for at least 48 hours,
along with cardiac monitoring in patients who do not
respond to therapy promptly. Since relapses are fre-
quent, patients should be hospitalized until their condi-
tions remain stable without physostigmine for at least 24
hours.
Since overdosage is often deliberate, patients may
attempt suicide by other means during the recovery
phase.

References:
1. Granacher RP, Baldessarini RJ: Physostigmine: Its use
in acute anticholinergic syndrome with antidepressant
and antiparkinson drugs. *Arch Gen Psychiatry* 32:375-
380, Mar 1975.
2. Burks JS, Walker JE, Rumack BH, Ott JE: Tricyclic
antidepressant poisoning: Reversal of coma,
choreoathetosis, and myoclonus by physostigmine.
JAMA 230:1405-1407, Dec 9, 1974.
3. Snyder BD, Blonde L, McWhirter WR: Reversal of
amitriptyline intoxication by physostigmine. *JAMA*
230:1433-1434, Dec 9, 1974.

Dosage and Administration: Optimum dosage varies
with the severity of the symptoms and the response of
the individual patient. When a satisfactory response is
obtained, dosage should be reduced to the smallest
amount needed to maintain the remission. The larger
portion of the total daily dose may be taken at bedtime.
In some patients, a single dose at bedtime may be
sufficient. In general, lower dosages are recommended
for elderly patients.
Limbitrol 10-25 is recommended in an initial dosage of
three or four tablets daily in divided doses; this may be
increased to six tablets daily as required. Some patients
respond to smaller doses and can be maintained on two
tablets daily.
Limbitrol 5-12.5 in an initial dosage of three or four tablets
daily in divided doses may be satisfactory in patients
who do not tolerate higher doses.
How Supplied: White, film-coated tablets, each con-
taining 10 mg chlordiazepoxide and 25 mg amitriptyline
(as the hydrochloride salt) and blue, film-coated tablets,
each containing 5 mg chlordiazepoxide and 12.5 mg
amitriptyline (as the hydrochloride salt)—bottles of 100
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Paks of 50.

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Limbitrol®

Tablets 10-25 each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt)

Tablets 5-12.5 each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)

CIV

Please see summary of product information on last page of this advertisement.

LIMBITROL® TABLETS Tranquilizer—Antidepressant

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Relief of moderate to severe depression associated with moderate to severe anxiety.

Contraindications: Known hypersensitivity to benzodiazepines or tricyclic antidepressants: Do not use with monoamine oxidase (MAO) inhibitors or within 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use; then initiate cautiously, gradually increasing dosage until optimal response is achieved. Contraindicated during acute recovery phase following myocardial infarction.

Warnings: Use with great care in patients with history of urinary retention or angle-closure glaucoma. Severe constipation may occur in patients taking tricyclic antidepressants and anticholinergic-type drugs. Closely supervise cardiovascular patients. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, especially high doses. Myocardial infarction and stroke reported with use of this class of drugs.)

Usage in Pregnancy: **Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Since physical and psychological dependence to chlordiazepoxide have been reported rarely, use caution in administering Limbitrol to addiction-prone individuals or those who might increase dosage; withdrawal symptoms following discontinuation of either component alone have been reported (nausea, headache and malaise for amitriptyline; symptoms [including convulsions] similar to those of barbiturate withdrawal for chlordiazepoxide).

Precautions: Use with caution in patients with a history of seizures, in hyperthyroid patients or those on thyroid medication, and in patients with impaired renal or hepatic function. Because of the possibility of suicide in depressed patients, do not permit easy access to large quantities in these patients. Caution patients about possible combined effects with alcohol and other CNS depressants and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Periodic liver function tests and blood counts are recommended during prolonged treatment. Amitriptyline component may block action of guanethidine or similar antihypertensives. Concomitant use with other psychotropic drugs has not been evaluated: sedative effects may be additive. Discontinue several days before surgery. Limit concomitant administration of ECT to essential treatment. See Warnings for precautions about pregnancy. Limbitrol should not be taken during the nursing period. Not recommended in children under 12.

In the elderly and debilitated, limit to smallest effective dosage to preclude ataxia, oversedation, confusion or anticholinergic effects.

Adverse Reactions: Most frequently reported are those associated with either component alone: drowsiness, dry mouth, constipation, blurred vision, dizziness and bloating. Less frequently occurring reactions include vivid dreams, impotence, tremor, confusion and nasal congestion. Many depressive symptoms including anorexia, fatigue, weakness, restlessness and lethargy have been reported as side effects of both Limbitrol and amitriptyline. Granulocytopenia, jaundice and hepatic dysfunction have been observed rarely.

This list includes adverse reactions not reported with Limbitrol but requiring consideration because they have been reported with one or both components or closely related drugs:

Cardiovascular: Hypotension, hypertension, tachycardia, palpitations, myocardial infarction, arrhythmias, heart block, stroke.

Psychiatric: Euphoria, apprehension, poor concentration, delusions, hallucinations, hypomania and increased or decreased libido.

Neurologic: Incoordination, ataxia, numbness, tingling and paresthesias of the extremities, extrapyramidal symptoms, syncope, changes in EEG patterns.

Anticholinergic: Disturbance of accommodation, paralytic ileus, urinary retention, dilatation of urinary tract.

Allergic: Skin rash, urticaria, photosensitization, edema of face and tongue, pruritus.

Hematologic: Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia.

Gastrointestinal: Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, black tongue.

Endocrine: Testicular swelling and gynecomastia in the male, breast enlargement, galactorrhea and minor menstrual irregularities in the female and elevation and lowering of blood sugar levels.

Other: Headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, jaundice, alopecia, parotid swelling.

Overdosage: Immediately hospitalize patient suspected of having taken an overdose. Treatment is symptomatic and supportive. I.V. administration of 1 to 3 mg physostigmine salicylate has been reported to reverse the symptoms of amitriptyline poisoning. See complete product information for manifestation and treatment.

Dosage: Individualize according to symptom severity and patient response. Reduce to smallest effective dosage when satisfactory response is obtained. Larger portion of daily dose may be taken at bedtime. Single *h.s.* dose may suffice for some patients. Lower dosages are recommended for the elderly.

Limbitrol 10-25, initial dosage of three to four tablets daily in divided doses, increased up to six tablets or decreased to two tablets daily as required. Limbitrol 5-12.5, initial dosage of three to four tablets daily in divided doses, for patients who do not tolerate higher doses.

Supplied: Limbitrol 10-25 tablets each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt) and Limbitrol 5-12.5 tablets each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)—bottles of 100 and 500; Tel-E-Dose* packages of 100; Prescription Paks of 50.

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How to initiate and maintain therapy

Select dosage strength appropriate for each patient

☐ Limbitrol 10-25 is recommended for most patients

☐ Limbitrol 5-12.5 may be indicated for those who do not tolerate higher doses and for elderly patients

Specify daily dosage based on symptom severity

☐ An initial dosage of three tablets is recommended.

☐ Dosage may be increased to six tablets or decreased to two tablets daily as necessary.

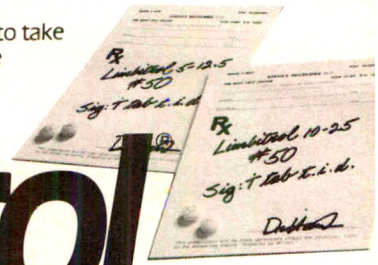
☐ Once a satisfactory response is obtained, evaluate response and, if necessary, reduce dosage to smallest amount needed.

Utilize dosage options to best accommodate individual patient needs

☐ T.I.D. or Q.I.D., familiar regimens most suited for patients who tolerate medication without undue drowsiness

☐ Two tablets one hour before bedtime and one tablet midday may minimize daytime drowsiness and help relieve a common target symptom—insomnia

☐ Entire dosage h.s. to take maximum advantage of the sedative effect



Limbitrol

Tablets 10-25 each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt)

Tablets 5-12.5 each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)

CIV

How to make each patient an informed patient

1. Discuss with patients the probability that they will experience drowsiness, especially during the first week.

2. Reassure your patients that drowsiness is one indication that the medication is working and that it may help alleviate their insomnia.

3. Encourage patients to report if drowsiness becomes troublesome so that, if necessary, dosage schedule can be adjusted or the symptom treated.

4. Caution patients about the combined effects with alcohol or other CNS depressants. Let them know that the additive effects may produce a harmful level of sedation and CNS depression.

5. Caution patients about activities requiring complete mental alertness, such as operating machinery or driving a car.

6. Warn pregnant patients and patients of childbearing age that the safety of Limbitrol in pregnancy has not yet been established.

Please see complete product disclosure for other pertinent information.

Limbitrol should not be used under the following circumstances:

1. Hypersensitivity to benzodiazepines or tricyclic antidepressants.
2. Should not be given with an MAO inhibitor. To replace an MAO inhibitor with Limbitrol, discontinue MAO inhibitor for a minimum of 14 days before cautiously initiating Limbitrol therapy.
3. During the acute recovery phase following myocardial infarction.

a consideration beyond efficacy alone...

Infrequent akathisia and other disabling extrapyramidal effects...

The rapidly rising readmission rate among discharged psychotic patients is mainly due to noncompliance with antipsychotic drug therapy. And this, in turn, may be largely attributed to disabling extrapyramidal side effects, notably akathisia.[1]

Although extrapyramidal effects are characteristic of antipsychotic agents in general, with Mellaril (thioridazine) such effects are infrequent. Adding an antiparkinsonian agent—which can cause its own side effects—can usually be avoided. Mellaril (thioridazine) is contraindicated in patients with severe hypotensive or hypertensive heart disease.

Extrapyramidal Effects of Selected Antipsychotic Agents*[2]

DRUG	EXTRAPYRAMIDAL EFFECTS
Chlorpromazine	Moderate
Perphenazine	High
Prochlorperazine	High
Fluphenazine	High
Acetophenazine	Moderate
Trifluoperazine	High
Chlorprothixene	Moderate
Thiothixene	Moderate
Haloperidol	High
MELLARIL® (thioridazine)	Low

*Based on antipsychotic dosage ranges

References

1. Van Putten T: The rising rehospitalization rate of psychiatric patients. Scientific Exhibit, American Psychiatric Association, 130th Annual Meeting, Toronto, Canada, May 2-6, 1977.

2. Byck R: Drugs and the treatment of psychiatric disorders, in Goodman LS, Gilman A (eds): *The Pharmacological Basis of Therapeutics*, ed. 5. New York, Macmillan Publishing Co, Inc, 1975, pp 170-171.

Mellaril® (thioridazine)

ABLETS: 50 mg, 100 mg, 150 mg, and 200 mg thioridazine HCl, USP

ELLARIL-S™ (thioridazine) SUSPENSION, per 5 ml: thioridazine se equivalent to 100 mg thioridazine HCl, USP

Before prescribing or administering, see Sandoz literature for full product information. The following is a brief summary.

Contraindications: Severe central nervous system depression, comatose states from any cause, hypertensive or hypotensive heart disease of extreme degree.

Warnings: Administer cautiously to patients who have previously exhibited a hypersensitivity reaction (e.g., blood dyscrasias, jaundice) to phenothiazines. Phenothiazines are capable of potentiating central nervous system depressants (e.g., anesthetics, opiates, alcohol, etc.) as well as atropine and phosphorus insecticides; carefully consider benefit versus risk in less severe disorders. During pregnancy, administer only when the potential benefits exceed the possible risks to mother and fetus.

Precautions: There have been infrequent reports of leukopenia and/or agranulocytosis and convulsive seizures. In epileptic patients, anticonvulsant medication should also be maintained. Pigmentary retinopathy, observed primarily in patients receiving larger than recommended doses, is characterized by diminution of visual acuity, brownish coloring of vision, and impairment of night vision; the possibility of its occurrence may be reduced by remaining within recommended dosage limits. Administer cautiously to patients participating in activities requiring complete mental alertness (e.g., driving), and increase dosage gradually. Orthostatic hypotension is more common in females than in males. Do not use epinephrine in treating drug-induced hypotension since phenothiazines may induce a reversed epinephrine effect on occasion. Daily doses in excess of 300 mg should be used only in severe neuropsychiatric conditions.

Adverse Reactions: *Central Nervous System*—Drowsiness, especially with large doses, early in treatment; infrequently, pseudoparkinsonism and other extrapyramidal symptoms; rarely, nocturnal confusion, hyperactivity, lethargy, psychotic reactions, restlessness, and headache. *Autonomic Nervous System*—Dryness of mouth, blurred vision, constipation, nausea, vomiting, diarrhea, nasal stuffiness, and pallor. *Endocrine System*—Galactorrhea, breast engorgement, amenorrhea, inhibition of ejaculation, and peripheral edema. *Skin*—Dermatitis and skin eruptions of the urticarial type, photosensitivity. *Cardiovascular System*—ECG changes (see *Cardiovascular Effects* below). *Other*—Rare cases described as parotid swelling.

It should be noted that efficacy, indications and untoward effects have varied with the different phenothiazines. It has been reported that old age lowers the tolerance for phenothiazines; the most common neurologic side effects are parkinsonism and akathisia, and the risk of agranulocytosis and leukopenia increases. The following reactions have occurred with phenothiazines and should be considered whenever one of these drugs is used. *Autonomic Reactions*—Miosis, obstipation, anorexia, paralytic ileus. *Cutaneous Reactions*—Erythema, exfoliative dermatitis, contact dermatitis. *Blood Dyscrasias*—Agranulocytosis, leukopenia, eosinophilia, thrombocytopenia, anemia, aplastic anemia, pancytopenia. *Allergic Reactions*—Fever, laryngeal edema, angioneurotic edema, asthma. *Hepatotoxicity*—Jaundice, biliary stasis. *Cardiovascular Effects*—Changes in terminal portion of electrocardiogram, including prolongation of Q-T interval, lowering and inversion of T-wave, and appearance of a wave tentatively identified as a bifid T or a U wave have been observed with phenothiazines, including Mellaril (thioridazine); these appear to be reversible and due to altered repolarization, not myocardial damage. While there is no evidence of a causal relationship between these changes and significant disturbance of cardiac rhythm, several sudden and unexpected deaths apparently due to cardiac arrest have occurred in patients showing characteristic electrocardiographic changes while taking the drug. While proposed, periodic electrocardiograms are not regarded as predictive. Hypotension, rarely resulting in cardiac arrest. *Extrapyramidal Symptoms*—Akathisia, agitation, motor restlessness, dystonic reactions, trismus, torticollis, opisthotonus, oculogyric crises, tremor, muscular rigidity, and akinesia. *Persistent Tardive Dyskinesia*—Persistent and sometimes irreversible tardive dyskinesia, characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and sometimes of extremities may occur on long-term therapy or after discontinuation of therapy, the risk being greater in elderly patients on high-dose therapy, especially females; if symptoms appear, discontinue all antipsychotic agents. Syndrome may be masked if treatment is reinstituted, dosage is increased, or antipsychotic agent is switched. Fine vermicular movements of tongue may be an early sign, and syndrome may not develop if medication is stopped at that time. *Endocrine Disturbances*—Menstrual irregularities, altered libido, gynecomastia, lactation, weight gain, edema, false positive pregnancy tests. *Urinary Disturbances*—Retention, incontinence. *Others*—Hyperpyrexia; behavioral effects suggestive of a paradoxical reaction, including excitement, bizarre dreams, aggravation of psychoses, and toxic confusional states; following long-term treatment, a peculiar skin-eye syndrome marked by progressive pigmentation of skin or conjunctiva and/or accompanied by discoloration of exposed sclera and cornea; stellate or irregular opacities of anterior lens and cornea; systemic lupus erythematosus-like syndrome.

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Infrequent akathisia and other disabling extrapyramidal effects...



a consideration beyond efficacy alone for the discharged patient who needs an antipsychotic agent

Mellaril® [thioridazine]

TABLETS: 50 mg, 100 mg, 150 mg, and 200 mg thioridazine HCl, USP

MELLARIL-S™ (thioridazine) SUSPENSION, per 5 ml:
thioridazine base equivalent to 100 mg thioridazine HCl, USP

For brief summary, please see preceding page.

Am J Psychiatry 136:4A, April 1979

THE AMERICAN JOURNAL OF PSYCHIATRY

Platelet Monoamine Oxidase Activity in Schizophrenia: A Review of the Data

BY RICHARD JED WYATT, M.D., STEVEN G. POTKIN, M.D., AND DENNIS L. MURPHY, M.D.

The authors review studies of platelet and white cell monoamine oxidase (MAO) activity in schizophrenic patients. The data on acute schizophrenic patients remain inconclusive. Review of 26 reports of chronic schizophrenic patients leaves little doubt, however, that there is a subgroup in which the enzyme activity is decreased. Despite the strong association of decreased MAO activity and chronic schizophrenia, the etiological relationship of low platelet MAO activity to schizophrenia has not been demonstrated. More complete diagnostic descriptions will shed light on precisely which patients have lowered MAO activity. Further metabolic investigations with such patients are needed to determine the physiological significance of this phenomenon.

AT LEAST 26 STUDIES have been published concerning the enzyme monoamine oxidase (MAO) in blood platelets and white cells of chronic schizophrenic patients (1-37) (see table 1). Although at times the deviation from control subjects has been very small, in all but 4 instances (22, 23, 33, 35) a decrease in MAO activity has been found. In 19 studies the reported decrease was statistically significant. Using the sign test, the chance of 22 of 26 studies demonstrating a decrease is less than 1 in 4,000, and the possibility of 19 of 26 being statistically significant at the .05 level by chance alone is less than 1 in 10^{19} (binomial test). In spite of this evidence, the finding continues to be thought of as controversial. We believe that what is controversial about the finding is not whether or not platelet MAO activity is decreased in some schizophrenic patients, but to what degree, in which patients, and what, if any, biological significance this decrease might have.

Received June 5, 1978; accepted Oct. 17, 1978; revised Jan. 9, 1979.

Dr. Wyatt is Chief and Dr. Potkin is Staff Psychiatrist, Laboratory of Clinical Psychopharmacology, Division of Special Mental Health Research, Intramural Research Program, William A. White Bldg., Rm. 536, St. Elizabeths Hospital, Washington, D.C. 20032. Dr. Murphy is Chief, Clinical Neuropharmacology Branch, National Institute of Mental Health, Bethesda, Md.

DEGREE OF DECREASE

The magnitude of the decrease in MAO activity in schizophrenia is important because profound alterations are usually demonstrated in diseases in which enzyme deficiencies have been found to be causative. Although the extent of the difference in platelet MAO between schizophrenic patients and normal subjects has varied from study to study, no study has reported a mean reduction greater than 68% from the MAO activity of normal control subjects (6). Group means, however, obscure individual differences, and in several of the studies some of the schizophrenic patients were found to have extremely low MAO activity. If these platelet alterations were shown to be systemic, they would be consistent with enzyme deficits found in many other genetic diseases. It should be noted that some genetically based disorders like porphyria may be associated with only partial enzyme reductions (on the order of 50% or less)—and that in other genetic disorders a wide variation of measured enzyme activity changes may be associated with various forms of the disorders (38).

Nevertheless, marked deficits in MAO activity are not characteristic of most schizophrenic patients; they occur in normal subjects as well as in individuals with other psychiatric diagnoses. In fact, great variability exists among the studies of schizophrenic patients.

SOURCES OF VARIABILITY

There are many potential sources of variability in studies comparing patients and control subjects. The

TABLE 1
Peripheral MAO Activity in Chronic Schizophrenic Patients Compared with Control Groups

Study Number	Reference[a]	Patient Description	Percent of MAO Activity of Control Group[b]
1	Murphy and Wyatt, 1972 (1)	Chronic	41
2	Wyatt and associates, 1973 (2)	Chronic	61
3	Shaskan and Becker, 1974 (3)	Chronic and acute depressed	Low
	Shaskan and Becker, 1975 (4)		
	Becker and Shaskan, 1977 (5)		
4	Meltzer and Stahl, 1974 (6)	Chronic	32–52[c]
5	Zeller and associates, 1975 (7)	Chronic and acute men	39–43[c]
		Chronic and acute women	32–38[c]
6	Bailey and associates, 1975 (8)	Chronic	91[c, d]
	Owen and associates, 1976 (9)		
	Brockington and associates, 1976 (10)		
	Joseph and associates, 1977 (11)		
7	Takahashi and associates, 1975 (12)	Chronic	Low
8	Murphy and associates, 1974 (13)	Chronic men	71[c]
	Murphy and Wyatt, 1975 (14)	Chronic women	53[c]
	Wyatt and Murphy, 1975 (15)		
	Wyatt and associates, 1975 (16)		
	Wyatt, 1975 (17)		
	Wyatt and Murphy, 1976 (18)		
9	Schildkraut and associates, 1976 (19)	Chronic and acute nonparanoid	100[d]
		Chronic and acute paranoid	66
10	Domino and Khanna, 1976 (20)	Chronic	46
11	White and associates, 1976 (21)	Chronic	90[d]
12	Belmaker and associates, 1976 (22)	Chronic	107–112[c, d]
13	Belmaker and associates, 1977 (23)	Chronic	113[d]
14	Idrisov, 1976 (24)	Chronic nuclear (leukocyte)	78
15	Berrettini and associates, 1977 (25)	Chronic	59
16	Berrettini and associates, 1978 (26)	Chronic	51–67[c]
17	Sullivan and associates, 1977 (27)	Chronic nonparanoid	74[d]
	Sullivan and associates, 1978 (28)	Chronic paranoid	55
18	Sullivan and associates, 1978 (29)	Chronic (lymphocytes)	65
		Chronic (platelets)	67
19	Demisch and associates, 1977 (30)	Schizophrenic defect	87[c, d]
		Paranoid	60[c]
20	Berger and associates, 1978 (31)	Chronic, undifferentiated	64
		Paranoid	66
21	Wyatt and associates, 1978 (36)	Chronic undifferentiated	70
		Paranoid	39
22	Potkin and associates, 1978 (32)	Chronic undifferentiated	80
		Chronic undifferentiated with paranoid features	51
		Paranoid	49
23	Groshong and Baldessarini, 1978 (33)	Chronic (platelet)	103[d]
	Baldessarini and Lipinski, 1978 (37)		
24	van Valkenburg and Crowe, 1978 (34)	Chronic hebephrenic	46
		Paranoid	51
25	Friedhoff and associates, 1978 (35)	Chronic medicated	45–72[c]
26	Friedhoff and associates, 1978 (35)	Chronic drug free	104–116[c, d]

[a]When possible, we have made an attempt to distinguish discrete investigations. Each numbered study refers to a separate and unique group of patients investigated, but each may include several publications.
[b]The MAO activities of the schizophrenic patients are expressed as percentages of the MAO activities of the control subjects.
[c]Used multiple substrates.
[d]Differences were not statistically significant.

history of schizophrenia research indicates that schizophrenic patients in most studies have larger inter- and intra-subject variability than control subjects and other patient populations (39). We expect that the use of rigorous and standardized descriptive criteria across studies would decrease this variability.

Acute Versus Chronic Schizophrenia

Despite the lack of consensus among schizophrenia researchers as to which patients are diagnosed as acute and which as chronic, this separation does decrease variability (40). Since much more variability in platelet MAO activity has been found in studies of acute than of chronic patients, separating patients by clinical course is essential (see tables 1 and 2). It has also been suggested that separating acute (1–7, 24, 35 40–44) and chronic schizophrenic patients s essential in psychophysiological studies (45).

TABLE 2
Peripheral MAO Activity in Acute and Remitting Schizophrenic Patients Compared with Control Groups

Study Number	Reference[a]	Patient Description	Percent of MAO Activity of Control Group[b]
1	Murphy and Wyatt, 1972 (1)	Acute	Decreased
2	Wyatt and associates, 1973 (2)	Acute	No difference
3	Shaskan and Becker, 1974 (3) Shaskan and Becker, 1975 (4) Becker and Shaskan, 1977 (5)	Acute and chronic depressed	Low
4	Friedman and associates, 1974 (41)	Remitting	83[c]
5	Nies and associates, 1974 (42)	Less than 1 year of hospitalization	72–86[d]
6	Meltzer and Stahl, 1974 (6)	Acute	45–69[d]
7	Carpenter and associates, 1975 (40)	Acute	97
8	Zeller and associates, 1975 (7)	Acute and chronic men Acute and chronic women	39–43[d] 32–38[d]
9	Cookson and associates, 1975 (43)	Acute	Low
10	Takahashi and associates, 1975 (12)	Newly admitted	No difference
11	Idrisov, 1976 (24)	Circular (leukocytes)	100
12	van Kammen and associates, 1978 (44)	Improving and nonimproving schizophrenic and schizo-affective	Generally decreased[c]
13	Sullivan and associates, 1978 (29)	Platelet Lymphocyte	93[c] 90[c]
14	Friedhoff and associates, 1978 (35)	Acute	103–131[c, d]

[a]When possible, we have made an attempt to distinguish discrete investigations. Each numbered study refers to a separate and unique group of patients investigated, but each may include several publications.
[b]The MAO activities of the schizophrenic patients are expressed as percentages of the MAO activities of the control subjects.
[c]Differences were not statistically significant.
[d]Used multiple substrates.

Control Groups

Selection of the control group is important. Berger and associates (31) noted that certain medical and professional hospital research personnel had unusually low MAO activity compared with nonprofessional hospital employees and college students (7.93 versus 11.17 nmol product per 10^8 platelets per hour). Professional hospital personnel are likely candidates for inclusion in control groups. In addition, Murphy and associates (46) and Buchsbaum and associates (47) found that college student volunteers with low platelet MAO activity had evidence of more psychopathology and had histories of more psychiatric and psychological counseling than students with higher platelet MAO activity. There was an increased risk of suicide in the families of male college students with lower platelet MAO activity. Subjects with low MAO activity have been described as being stimulus-seekers—people highly attuned to seeking out sensations from their environment (32). It is possible that students with low platelet MAO activity might be more likely to volunteer to be research subjects.

In addition to careful selection and description of both patient and control groups, factors such as sex of the subjects, age, administration of drugs, severity of illness, hormonal status, and dietary intake have been thought to be of potential significance. The sex of the subjects probably plays a role in platelet MAO activity: female subjects have been found to have a somewhat higher platelet MAO activity than male subjects (48, 49). Sex of the subjects has not always been taken into consideration in studies comparing control subjects with schizophrenic patients.

Age

The effect of age on MAO activity is disputed (48, 50), although there appears to be no correlation between the two in the age range in which schizophrenic patients are usually studied (20 to 50 years of age) (49). Nevertheless, in a study that did not find a statistically significant difference between chronic schizophrenic patients and control subjects (19), age of the patients could have been a factor. The age of the schizophrenic patients was 56.6 years, versus 47.9 years for the control subjects. If MAO activity increases after 50 years of age, studying older schizophrenic patients, especially if they are older than the control subjects, may alter the study's conclusions.

Drug Status

Several authors have suggested that drug status might contribute to the low levels of MAO activity seen in patients. Tricyclic antidepressants have been reported to inhibit MAO activity (51, 52). Becker and Shaskan (5) studied the clinical effects of tricyclic antidepressants in depressed schizophrenic patients and noted a decrease in MAO over time in 8 chronic schizophrenic patients who had high MAO activity. In view of the action of the tricyclics on MAO activity, it is possible that the decrease in MAO over time in some of these patients could have been due to the addition of tricyclics. However, the drugs most commonly used to treat schizophrenia, the phenothiazines and butyro-

phenones, have, if anything, a tendency to increase MAO activity (10, 13, 18).

Severity of Illness

Severity of illness may also be an important factor with regard to platelet MAO. Becker and Shaskan (5) found an inverse relationship between platelet MAO and severity of illness and symptoms, something we have also noted (2). However, Becker and Shaskan found no difference between combined schizophrenic inpatients and outpatients and normal control subjects, although borderline significant differences (χ^2=3.11, two-tailed, p=.078) exist between normal control subjects and inpatients at baseline. This latter finding was not reported because inpatients and outpatients were not originally examined separately in their publication.

Hormones and Diet

Hormones and nutrition are known to affect MAO activity (53, 54). The highest MAO activity in normal adult female human (55) and nonhuman primates (56) occurs just before ovulation; the lowest activity occurs in the postovulatory period. Estrogen is known to decrease MAO activity (57, 58). In severe iron-deficiency anemia, platelet MAO is markedly reduced, and this can be reversed by iron treatment (54).

To examine hormonal and nutritional issues in relation to the observed decreases in MAO activity in chronic male schizophrenic patients, a group of 21 male chronic schizophrenic patients were studied (59). The mean serum triiodothyronine of these patients was in the middle of the normal range, but 2 of the patients had slightly elevated concentrations. The mean serum testosterone was in the lower portion of the normal range; 3 of the patients had concentrations less than 300 ng/ml. The mean serum iron concentrations, iron-binding capacity, and calculated mean percent saturation were near the middle of the normal range. None of these parameters had a correlation coefficient that even approached statistical significance. Thus, the hormonal and nutritional factors studied so far do not seem to play an important role in the decreased MAO activity found in these male schizophrenic patients.

Assay Procedure

Other sources of important variance can come from the assay itself. One problem with many biochemical assays is that their reliability in the lower range is poor—at times, the only thing that is measured is noise. Thus, when an assay is insensitive to low values, it is unable to make distinctions between those values. This would decrease any statistical significance that might occur between groups. Few of the papers on platelet MAO have addressed this problem. In the assay we are currently using (49), the values remain linear, even for subjects with low activity.

Preparation of platelets themselves can produce problems. Depending on how the platelets are separated, there may be a difference in platelet size and, perhaps, in MAO activity (35, 60). Differences in MAO activity in normal control subjects have been found when methods of platelet preparation are varied. No differences between control and schizophrenic groups were found by Friedhoff and associates (35) when high-speed centrifugation was used. Higher speed centrifugation tends to exclude the heavier platelets, which have the most MAO activity (60). However, Friedhoff and associates also found significant differences between control subjects and schizophrenic patients when platelets were harvested by lower-speed centrifugation, which yields more complete platelet recovery. The MAO increase for control subjects was 41% for benzylamine when the higher yield procedure was used (p<.01, two-tailed t test); no change was seen in the schizophrenic groups. Unfortunately, the second schizophrenic group was receiving neuroleptics, and the effect of drugs on platelet distribution and size has not yet been studied.

TABLE 3
Control Subjects' Mean Platelet MAO Activity with Tryptamine as Substrate in 12 Studies

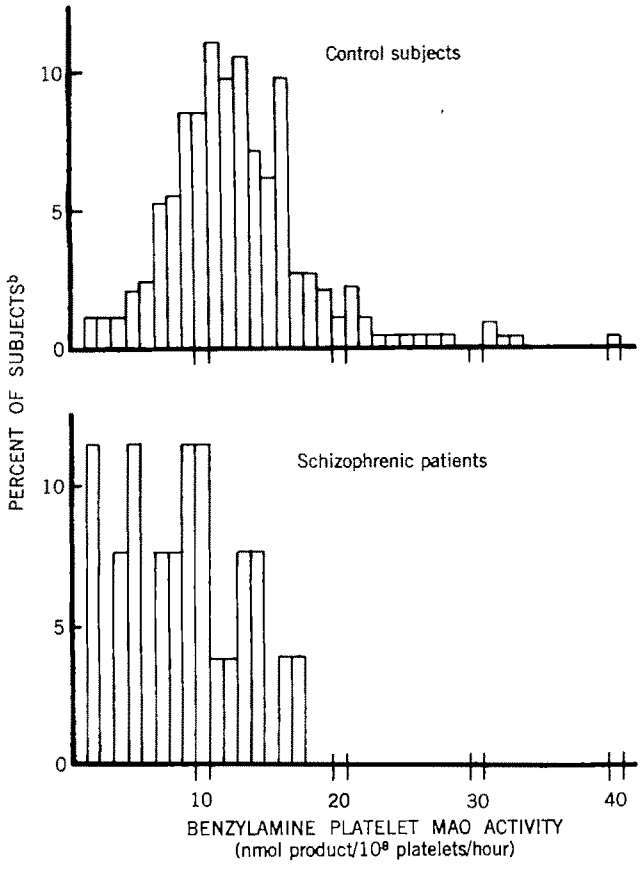
Reference	Number of Subjects	MAO Activity (nmol/hr/mg protein)
Murphy and Wyatt, 1972 (1)	22	6.35
Wyatt and associates, 1973 (2)	23	6.4
Nies and associates, 1974 (42)	12	3.6
Friedman and associates, 1974 (41)	23	1.91
Meltzer and Stahl, 1974 (6)	15	5.24
White and associates, 1976 (21)	12	2.0
Domino and Khanna, 1976 (20)	13	7.14
Owen and associates, 1976 (9)	70	3.6
Berrettini and associates, 1978 (26)	10	3.7
Demisch and associates, 1977 (30)	42	2.0
Schildkraut and associates, 1978 (61)	28	5.72
Friedhoff and associates, 1978 (35)	37	8.46

Although the reasons for differences in the assays are not always apparent, we can readily see that they exist. Table 3 shows that, using the same substrate and somewhat similar assay procedures, platelet MAO activity in normal subjects varied from a low of 1.91 to a high of 8.46 in different studies. Although some of this variation could well be due to subject differences, the majority of it is probably due to differences in the preparation of platelets or assay procedures.

Statistical Issues

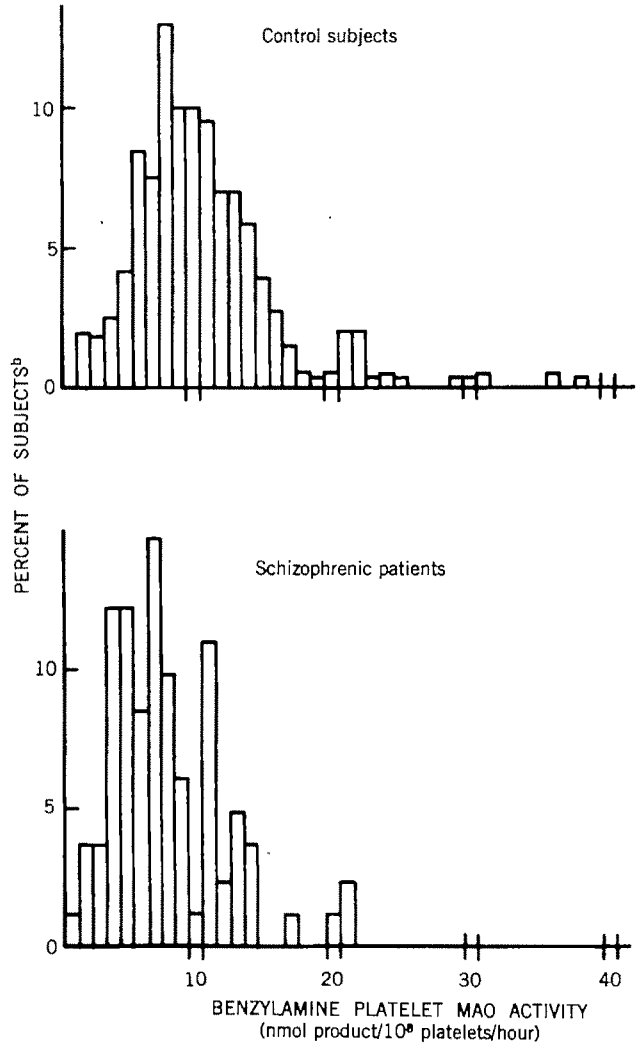
One type of statistical issue has already been addressed—that of not taking into consideration all the known sources of variance. Another statistical issue arises when there appears to be an obvious but not understood source of variance. Such a case may have occurred in the study reported by Brockington and associates (10). They presented data for control subjects and schizophrenic patients and found no significant differences. The mean MAO activity with tyramine as a substrate for their 70 control subjects was 28.4; for their 56 chronic schizophrenic patients it was 27.5. Al-

FIGURE 1
Frequency Distribution for Platelet MAO Activity in 332 Female Control Subjects and 26 Female Chronic Schizophrenic Patients[a]



[a]The mean level of MAO activity of the control subjects was 13.29±.29; the mean level of the schizophrenic patients was 8.44±.90. The difference between the group means was significant by two-tailed t test ($p<.001$).
[b]Bounded by one unit of MAO activity.

FIGURE 2
Frequency Distribution for Platelet MAO Activity in 348 Male Control Subjects and 122 Male Chronic Schizophrenic Patients[a]



[a]The mean level of MAO activity of the control subjects was 11.04±.29; the mean level of the schizophrenic patients was 7.04±.46. The difference between the group means was significant by two-tailed t test ($p<.001$).
[b]Bounded by one unit of MAO activity.

though these means are essentially identical, the histogram in their report shows that 8 of the schizophrenic patients had lower MAO activities than any of the control subjects. This suggests that there may be a subgroup of schizophrenic patients with low MAO activity who were not revealed in a comparison of group means. The observation that 8 schizophrenic patients had lower MAO activity than any of the control subjects is statistically significant at the $p=.01$ level (Fisher exact probability test).

Another source of variance lies in the distribution of subgroups of schizophrenic patients from population to population. Patient populations from different hospitals may not have the same distributions of subgroups of schizophrenic patients. If low platelet MAO occurred primarily in some of these subgroups, then, solely on the basis of selection, all studies would not be expected to demonstrate differences in platelet MAO. In all probability, schizophrenia, like pneumonia and mental retardation, is a number of diseases. If different investigators examine very different groups (e.g., process versus reactive, paranoid versus nonparanoid), they might be expected to find differences in etiological or associative factors. These potentially important differences become confounding factors when they are not explicitly separated in the experimental design.

MAO in Schizophrenia (NIMH Studies)

Figures 1 and 2 are frequency histograms of our sizable group of control subjects and chronic schizophrenic patients. For both male and female chronic schizophrenic patients (those whose onset was largely insidious and who have been hospitalized for more than 2 years), there was a marked mean decrease in platelet MAO activity in comparison with the control groups.

Delineation of Subgroups

In our initial studies of both acute and chronic schizophrenic patients (1, 2) we did not find any subgroup that had low MAO activity compared with other subgroups. Subsequently, we noted that the acute schizophrenic patients as a group were not different from control subjects (40). Among the acute patients, however, those with impaired reality testing, less restlessness, better prognostic scores, and paranoid and grandiose delusions tended to have lower MAO activity than other patients (15). Although most other studies of acute schizophrenic patients have not found this subgroup to have lower MAO activity, Schildkraut and associates (19) found in a mixed group of schizophrenic patients that those identified by the presence of auditory hallucinations had lower platelet MAO activity than others. The schizophrenic group with low platelet MAO activity was referred to as paranoid on the basis of the persecutory and accusatory nature of their delusions and hallucinations.

We have undertaken additional studies to look at the relationship of platelet MAO and subgroups of schizophrenia. The first (36) was a retrospective chart study in which many psychological and physiological parameters were correlated with platelet MAO activity. Low platelet MAO activity correlated with the presence of seizures and abnormal EEGs. Low MAO was also significantly more apparent in paranoid than in nonparanoid schizophrenic patients. A prospective study that eliminated patients with a history of seizures or abnormal EEGs and subgrouped patients on the basis of a prospective interview as well as a historical review (32) confirmed the association of low MAO activity with the paranoid subgroup.

A third study done in collaboration with investigators at the Palo Alto Veterans Administration Hospital in California (31) was unable to demonstrate a difference between paranoid and nonparanoid schizophrenic patients when subtyping was by chart diagnosis. Why one group of paranoid schizophrenic patients should be different from another is unclear. However, there is one likely difference between patients in a Veterans Administration hospital and those in St. Elizabeths Hospital. The patients at the Veterans Administration hospital were sufficiently intact at one time to have been inducted into military service and were functional for long enough to have gained veterans' benefits. The patients at St. Elizabeths Hospital may have had an earlier onset of their illness and may have been more process than reactive. Studies measuring these parameters will have to be carried out with both groups of patients. However, a recent report by Sullivan and associates (28), who also studied patients at a Veterans Administration hospital, found a difference between paranoid and nonparanoid schizophrenic patients. This finding suggests that the answer will be harder to determine.

To date, the clinically descriptive variables that have best distinguished paranoid from nonparanoid schizophrenic patients by a linear discriminant function analysis are, in order of importance, degree of suspiciousness, preoccupation with and systematization of delusions, delusions of control, stereotyped behavior, and intact memory (32).

Other investigators have attempted to subdivide patients in other ways. For example, Brockington and associates (10) found no difference in MAO activity between schizophrenic patients with positive symptoms (thought disorder, hallucinations, or delusions) and those with negative symptoms (flattening of affect, mutism). Brockington and associates also studied a group of schizo-affective patients and found that they had higher platelet MAO activity than the schizophrenic or control subjects. Elevated platelet MAO activity has also been reported in a group of patients described as having schizophrenia-related depressive disorders (61).

BIOLOGICAL SIGNIFICANCE

Is the Enzyme Different in Schizophrenia?

Initial observation (15, 62) suggested that the physical properties of MAO in schizophrenic patients and control subjects was not different. A number of lines of evidence are beginning to suggest that this may not be the case. Zeller and associates (7) reported a differential inability of platelets of schizophrenic patients to metabolize paraiodobenzylamine compared with tyramine and metaiodobenzylamine. Furthermore, subgroups of schizophrenic patients studied by Demisch and associates (30) appeared to have a differential lowering in their platelet MAO activity depending on the substrate examined. The paranoid subgroups had the lowest activities. In addition, lower Kms (Michaelis affinity constants) in schizophrenic patients have been found by Berrettini and associates (26). However, Belmaker and associates (23) found high Kms in schizophrenic patients, but their values, which were calculated from amphetamine inhibition curves for normal subjects, were 13-fold lower than our values at similar pH and substrates (19). Further studies are needed to determine if the physical and chemical differences in platelet MAO activity of schizophrenic patients are real.

Genetic Control

Several studies have indicated that platelet MAO activity in man is at least in part under genetic control. Normal (63) and affectively disordered (64) monozygotic twins have a much higher correlation of enzyme activity than do dizygotic twins or siblings. Monozygotic twins discordant for schizophrenia (2, 16) also have a high correlation of platelet MAO activity, as do first-degree relatives of schizophrenic patients who have high and low platelet MAO activity (16).

Low Platelet MAO Activity in Nonschizophrenic Groups

Low platelet MAO activity has been described in a number of groups of people. As previously stated, male college students with the lowest MAO activity (46, 47) had a high frequency of psychiatric counseling, more convictions for legal offenses, and increased suicides and suicide attempts in relatives. Alcoholics (65, 66), heavy users of marijuana (67), and bipolar depressed patients (68) also have been reported to have some decrease in platelet MAO activity.

It is tempting to speculate what these groups might have in common psychologically. Before any psychological commonality across disorders can be pursued very far, however, we need to know more than we do about the biochemical commonality. We know with varying degrees of certainty that these groups have reduced platelet MAO activity, but we do not know if MAO activity is reduced for the same reasons.

Cause or Association?

The decreased peripheral MAO activity seen in some schizophrenic patients could be related to the cause of the illness or could simply be associated with the disorder. A causal relationship would be particularly exciting because of the many biogenic amine hypotheses of schizophrenia. The dopamine (69), transmethylation (70), phenylethylamine (71), and norepinephrine hypotheses would be consistent with a decrease in MAO activity. However, only an association has been established to date—i.e., low platelet MAO activity accompanies certain forms of schizophrenia.

MAO activity has been studied in the brains of schizophrenic patients who have come to autopsy. In all but one of the studies (15), brain MAO activity of schizophrenic patients has been no different from that of control subjects. As yet, no attempt has been made to subgroup the schizophrenic patients (e.g., paranoid versus nonparanoid) whose brains have been studied. Studies of tissues other than blood components and brain have not been completed. There is one negative study comparing fibroblasts in schizophrenic patients and control subjects. However, fibroblasts predominantly contain a different form of MAO than the platelet and lymphocyte (33).

Presuming that peripheral tissues other than the platelet and white cells are involved, but not the brain, can the decrease in MAO activity in those tissues be causative? We believe that it could be, but there is no evidence thus far that it is. An example of how this might take place is readily at hand. Phenylketonuria, a disorder whose principal symptoms and signs are derived from altered brain function, is not primarily a brain disorder, but one of the liver. The enzyme phenylalanine hydroxylase, which converts phenylalanine to tyrosine, is deficient in the liver (little or none is normally present in the brain), and abnormally high metabolites cross from the blood into the brain, disrupting normal function.

A similar phenomenon could occur in schizophrenia. For example, of the known endogenous substrates for platelet MAO, phenylethylamine (PEA) has the greatest affinity for the enzyme. PEA is structurally similar to amphetamine (α-methyl-PEA) and produces many analogous behavioral phenomena in animals (72, 73). PEA readily crosses the blood-brain barrier, and if it were elevated in schizophrenia it might be expected to produce a psychosis similar to that produced by amphetamine. Of the 3 published studies of urinary PEA in schizophrenic patients (74–76), however, only 1 (74) suggested that PEA might be elevated.

If decreased MAO activity is causative in schizophrenia, why does inhibition of MAO by drugs not produce a psychosis? In fact, although MAO inhibitors are used as antidepressants, their use can be accompanied by significant behavioral toxicity, including some prominent stimulant effects in some individuals (77). However, most patients treated with MAO-inhibiting drugs do not develop any behavioral toxicity and certainly do not develop schizophrenia. Several possible explanations for this paradox deserve exploration. Since schizophrenia is primarily a disorder beginning shortly after puberty, data regarding short-term therapeutic inhibition of MAO in the adult may not be relevant to the still-developing adolescent brain.

Alternatively, perhaps simply having decreased MAO activity is not enough to produce disease at any time; perhaps other genetic disorders or environmental factors are needed. There is a body of literature, for example, suggesting that a higher proportion of schizophrenic patients are heterozygotes for phenylketonuria than would be expected in the general population (71). Could two genetic abnormalities (having low MAO and being a heterozygote for phenylketonuria) that are relatively innocuous when found by themselves produce a major disorder when they are found together in the same person? Alternatively, ameliorative factors may be present in most people but missing in others (78).

CONCLUSIONS

We believe there is an association between low platelet MAO activity and some forms of schizophrenia. The physiological significance of this finding is very much open to question insofar as this abnormality has been found in other populations and no alteration in amine metabolism has been found in association with low MAO. Certainly we need better diagnostic descriptions of the patients with the abnormality so that more meaningful comparisons can be made across study populations.

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Delirium After Cataract Surgery: Review and Two Cases

BY WILLIAM K. SUMMERS, M.D., AND THEODORE C. REICH, M.D.

The authors define the delirium seen after cataract surgery on the basis of Dupuytren's original description and review studies using the criteria they set up on this basis. They then present prospective data on 2 patients with postcataractectomy delirium and 25 patients who did not experience this delirium. The data suggest that delirium after cataract surgery represents anticholinergic toxicity.

CLEAR DEFINITION of criteria is necessary for accuracy of diagnosis, especially with respect to psychiatric diagnosis (1). The many studies on psychiatric phenomena after cataract surgery are often conflicting and vague because of a lack of diagnostic criteria.

In this study, we will use a definition of the delirium seen after cataract surgery that is consistent with Dupuytren's original description (2). Specifically, postcataractectomy delirium refers to an acute change in a patient's mental status after cataract surgery and requires the presence of disorientation *and* disturbance of recent or remote memory as well as the presence of two or more of the following six symptoms: delusions, hallucinations, illusions, depersonalization, change in affect (anxiety, fear, depression, or euphoria), and abnormal psychomotor activity.

LITERATURE REVIEW

Using these criteria, we found that several studies of postcataractectomy delirium had inadequate data for interpretation of case reports (3–20). Table 1 gives the reported incidence, age, sex, time of onset of delirium, and duration of the delirium in 20 published studies (2, 21–39) as well as in the one reported here. Excluding the highest and lowest, the range of incidence represented in these studies is .3% to 15.9%. The zero incidence reported by Burns (27) is questionable because his patients had surgery at home, were not followed up until a week after surgery, and apparently were not specifically asked about delirium. Questioning is important because patients tend not to report these episodes spontaneously (38, 40, 41) However, Burns noted "several" episodes of delirium in his hospitalized cases. The 62% incidence reported by Linn and associates (35) was in a selected population with a 72% incidence of chronic organic brain syndrome.

The mean age range of patients with postcataractectomy delirium shown in table 1 was 50 to 85. The mean number of days after surgery that delirium occurred in the 85 patients in whom onset was observed was 3.1 days. Analysis of usable data shows that 82.5% of the cases of delirium occurred before the third postoperative day. As in most acute organic brain syndromes, the course of the delirium was noted to wax and wane (1, 26, 28, 29, 32, 33).

The duration of delirium after cataract surgery is either acute or chronic (34). The acute course has been reported to have a duration of minutes to more than 16 days. A chronic course has been reported in a small number of individuals (8, 28, 34). Table 1 shows the range of duration of delirium in 68 patients. The average number of days an acute course lasted was 2.6 days. Return to preoperative mental status by the fourth day after onset of delirium occurred in 87% of these cases.

Predisposing or causal factors in postcataractectomy delirium cited in 19 published studies (2, 21–26, 28–39) and in the one reported here are listed in table 2. Previous psychiatric illness, especially chronic organic brain syndrome and alcoholism, have often been mentioned (10, 17, 21, 28, 31, 33). There is evidence for an association of preoperative chronic organic brain syndrome and postcataractectomy delirium (25, 42). Association between the delirium and alcoholism or other psychiatric disease is unclear for lack of data in a comparison population (28, 34). Data on the association of the delirium and uremia (14, 18) also lack information on control subjects. The theory that sensory deprivation from bilateral patching is causative is interesting but unproven (3, 7, 11, 12, 37, 41, 43–45) (see table 3). Several authors (3, 10, 17, 22, 23) have reported the delirium in the absence of bilateral eye patching. In addition, the removal of the patch from the unoperated eye often does not result in rapid reversal of the delirium (22, 26, 31, 32, 33, 35). Analysis of the literature for response to removal of a patch from the unoperated eye indicates an immediate improvement in only 9 of 47 cases (19.1%). There are no reports evaluating orientation or memory testing after removal of patch-

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TABLE 1
Epidemiology of Postcataractectomy Delirium (PCD)

Reference	Number of Patients Studied	Patients with PCD (N=147)		Age[a]		Sex[a]		Onset[a, b]	Duration[a, c]
		N	%	Range	Mean	Men (N=50)	Women (N=30)		
Dupuytren, 1833 (2)	21	2	9.5	61 and over	—	0	1	Up to 15	Up to 3
Schnabel, 1880 (21)	183	12	6.6	—	—	—	—	—	—
Posey, 1900 (22)	—	19	—	50–80	—	—	—	1–4	—
Kipp, 1903 (23)	—	6	—	50–78	63.8	4	2	6–35	1–4
Finlay, 1904 (24)	294	1	0.3	—	66.0	0	1	1 and more	—
David, 1910 (25)	362	12	3.3	—	—	—	—	—	—
Parker, 1913 (26)	376	11	2.9	53–82	72.0	7	4	1–6	1–17
Burns, 1916 (27)	232	0	—	—	—	0	0	—	—
Brownell, 1917 (28)	962	30	3.1	51–82	72.3	24	6	3–12	1–5
Fisher, 1920 (29)	200	4	2.0	64 and over	—	—	—	Up to 11	1 and more
Lowe, 1922 (30)	—	1	—	82	82.0	0	1	Up to 3	Up to 2
Pfingst, 1923 (31)	—	5	—	60	60.0	4	1	Up to 2	—
Thomas, 1926 (32)	—	2	—	60–68	64.0	0	2	3–4	2–4
Preu and Guida, 1937 (33)	—	4	—	56–83	70.0	2	2	1–2	1–16 and more
Boyd and Norris, 1941 (34)	—	1	—	61	61.0	0	1	Up to 5	Up to 6
Linn and associates, 1954 (35)	21	13	61.9	45–85	75.0	7	6	1–2	1–7
Weisman and Hackett, 1958 (36)	—	2	—	72–78	75.0	1	1	1 and more	1 and more
Ziskind and associates, 1960 (37)	88	14	15.9	—	—	—	—	—	—
Jackson, 1969 (38)	60	5	8.3	—	—	—	—	—	—
Steahly, 1976 (39)	—	1	—	75 and over	75.0	1	0	1 and more	Up to 2
Summers and Reich, 1979	27	2	7.4	76–82	79.0	0	2	Up to 2	1–9

[a]Refers to patients with PCD only.
[b]Onset refers to the range of number of days after surgery delirium occurred. The day of the operation was counted as day 1.
[c]Duration refers to the range of number of days delirium lasted.

TABLE 2
Predisposing Factors In Reports of Postcataractectomy Delirium

Reference	Number of Patients (N=142)	Suspicion of Psychiatric Diagnosis			Postsurgical Complication (N=13)	Medical Diagnosis	
		Alcohol Abuse (N=15)	Chronic Organic Brain Syndrome (N=21)	Other (N=3)		Renal (N=8)	Other (N=4)
Dupuytren, 1833 (2)	2	—	1	—	1	—	—
Schnabel, 1880 (21)	12	—	—	—	—	—	—
Posey, 1900 (22)	19	—	2	0	1	—	—
Kipp, 1903 (23)	6	0	0	0	0	0	0
Finlay, 1904 (24)	1	0	—	—	0	1	0
David, 1910 (25)	12	—	—	—	—	—	—
Parker, 1913 (26)	11	—	—	—	1	1	—
Brownell, 1917 (28)	30	11	—	—	1	0	0
Fisher, 1920 (29)	4	0	—	—	2	—	—
Lowe, 1922 (30)	1	0	0	0	0	1	—
Pfingst, 1923 (31)	5	—	—	—	—	0	—
Thomas, 1926 (32)	2	0	0	1	1	2	0
Preu and Guida, 1937 (33)	4	0	1	1	0	1	1
Boyd and Norris, 1941 (34)	1	0	0	1	0	0	1
Linn and associates, 1954 (35)	13	—	13	—	—	—	—
Weisman and Hackett, 1958 (36)	2	—	1	—	—	—	—
Ziskind and associates, 1960 (37)	14	4	2	—	5	—	—
Jackson, 1969 (38)	5	—	—	—	—	—	—
Steahly, 1976 (39)	1	0	0	0	0	0	1
Summers and Reich, 1979	2	0	1	0	1	2	1

es. The term "improvement" seems to mean that a patient was less agitated.

The following investigation is a pilot prospective study of psychiatric symptoms after cataract surgery, using psychiatric research criteria (1).

METHOD

Thirty-three subjects undergoing intracapsular cataract extraction at Barnes Hospital in St. Louis, Missouri, were asked to participate in this investigation.

TABLE 3
Effects of Bilateral Patching in Reports of Postcataractectomy Delirium

Reference	No Bilateral Patch (N=10)	Bilateral Patch (N=47)	Effect of Patching: Immediate Improvement[a] (N=9)	Effect of Patching: Delayed Improvement[b] (N=38)
Dupuytren, 1833 (2)	—	—	—	—
Schnabel, 1880 (21)	—	—	—	—
Posey, 1900 (22)	—	9	0	9
Kipp, 1903 (23)	5	1	0	1
Finlay, 1904 (24)	0	1	0	1
David, 1910 (25)	—	—	—	—
Parker, 1913 (26)	—	6	1	5
Brownell, 1917 (28)	1	3	1	2
Fisher, 1920 (29)	—	1	0	1
Lowe, 1922 (30)	1	0	—	—
Pfingst, 1923 (31)	0	5	2	3
Thomas, 1926 (32)	0	2	0	2
Preu and Guida, 1937 (33)	0	4	1	3
Boyd and Norris, 1941 (34)	0	1	0	1
Linn and associates, 1954 (35)	0	13	3	10
Weisman and Hackett, 1958 (36)	—	1	1	0
Ziskind and associates, 1960 (37)	—	—	—	—
Jackson, 1969 (38)	—	—	—	—
Steahly, 1976 (39)	1	0	—	—
Summers and Reich, 1979	2	0	—	—

[a]Improvement within 1–2 hours after removal of bilateral patches.
[b]Improvement more than 2 hours after removal of bilateral patches.

After the study procedure had been explained to these patients, as required by the hospital's Human Usage Committee, 27 patients agreed to participate. Each subject was interviewed a minimum of four times. The presurgery interview included a medical-surgical history and a psychiatric history (1). Memory was tested using the "names-learning test" of Irving and associates (46). A mental status examination and the memory testing was done within 12 hours of surgery and again 48–72 hours after surgery. A fourth interview was conducted either by telephone or in person between 7 and 10 days after surgery. Laboratory values, nursing observations, and medications were obtained from the patients' charts.

The patients who had had no diagnosable episode of postcataractectomy delirium 7 days after surgery were considered our control group.

All of the subjects were given the names-learning test (46). A score of less than 44 of a possible 60 was considered indicative of a disturbance of recent memory. Patients who had had conditions requiring daily medications and/or medical surveillance at least every 3 months before their cataractectomy were considered as having a previous medical diagnosis.

A numerical system devised to estimate the patients' exposure to delirium-inducing agents during a given 24-hour period was calculated by multiplying the class number of the drug (described below) by the daily effective dosage level number (also described below). The result was termed the "drug risk number." Criteria for the class number of drugs were as follows: class 1=known synergistic effect with anticholinergic agents but not known to be a direct cause of delirium; class 2=known to cause delirium but currently not known to have CNS anticholinergic properties; and class 3=known to cause delirium reversible by physostigmine (i.e., known to cause an acute anticholinergic syndrome). The criteria for daily effective dosage level numbers were as follows: dosage level 1=dose range that would not have therapeutic effect for a 24-hour period; dosage level 2=dose range that would have therapeutic effect for a 24-hour period; and dosage level 3=dose range that exceeds the usual therapeutic range. All of the dose ranges were verified using standard pharmacological sources (47, 48).

As an example of the numerical system, a patient receiving 50 mg of secobarbital h.s. as his or her only charted medication would have a drug risk number of 3. Secobarbital is a class 3 drug but would typically require 200 mg per day to achieve therapeutic effect (i.e., sleep) over a 24-hour period. The total drug risk number for a 24-hour period was calculated as the sum of the drug risk numbers for each agent used in the same 24-hour period.

RESULTS

Two of the 27 patients (7.4%) developed postcataractectomy delirium.

Case Reports

Case 1. Ms. A, a 76-year-old married woman, had a 6-month history of decreasing visual activity and had been admitted for bilateral intracapsular cataract extraction. Her medical history revealed mild glaucoma that did not require medication, occasional "forgetfulness," and a cholecystectomy with an uneventful postoperative course. There was no family history of neuropsychiatric illness. Initial examination revealed a visual activity of 20/400 in both eyes with no evidence of glaucoma. She was well oriented and showed no evidence of memory impairment.

On the second hospital day the patient underwent a right intracapsular cataract extraction with local anesthesia of 2% lidocaine (Xylocaine) with epinephrine and hyaluronidase (Wydase). Light cautery was required for a "slight amount of bleeding." The postoperative course was uneventful mentally and physically. On the fifth hospital day the patient underwent a left intracapsular cataract extraction with the same anesthesia and no complication. At 8:00 p.m. on the seventh hospital day (36 hours after surgery) the patient became confused. She thought she was at home cleaning her house and was disoriented to time. Only the more recently operated eye was patched, and her room was well lighted. The next morning she continued to be disoriented and scored only 14 of a possible 60 on the names-learning test. Anticholinergic agents were withheld. Her mental status continued to wax and wane with mood swings, delusions ("someone is going to bury me alive"), and auditory and visual hallucinations ("of knights and princesses").

Physical examination showed mydriasis of the right pupil, a regular pulse of 96, temperature of 37°C, and slightly dry skin. There were no focal neurological signs. Her BUN was 24 mg/100 ml, creatinine was 1.4 mg/100 ml, fasting blood sugar was 101 mg/100 ml, serum calcium was 9.8 mg/100 ml, serum phosphorus was 2.9 mg/100 ml, alkaline phosphatase was 79 mU/ml, and serum glutamic-oxaloacetic transaminase was 38 mU/ml. Urinalysis, complete blood count, serum electrolytes, skull X ray, EEG, ECG, and brain scan showed no significant abnormality.

Twenty-four hours after the onset of delirium, 1.5 mg of physostigmine was given slowly I.V. with ECG monitor to rule out acute anticholinergic syndrome (49, 50). Before infusion the patient was disoriented to time, place, and person. She admitted that she had visual hallucinations. Her affect was consistent with fear, and she scored only 1 on the names-learning test. Testing done 15–30 minutes after physostigmine administration revealed recognition of family members, minimal disorientation to time (Ms. A stated the date of her operation), and a score of 46 on the names-learning test. However, the physostigmine trial was marred by gastrointestinal side effects. Two hours after physostigmine infusion the patient was again grossly disoriented. She gradually improved over the next 10 days. A computerized tomography scan was read as showing slight enlargement of the lateral ventricles and cerebral atrophy. On the 16th hospital day the patient was consistently oriented and scored 50 on the names-learning test. Follow-up interview revealed no further difficulties with memory or orientation and no hallucinations.

Case 2. Ms. B, an 82-year-old widow with bilateral cataracts, had noted decreasing visual activity for 8 months. Her history revealed mild hypertension for 15 years requiring various medications for 6 years and a left cerebral vascular accident with nearly total recovery 2 years before this admission. She and her family denied any psychiatric or surgical history. A granddaughter noted that the patient occasionally forgot where she put items but had not observed any gross disorientation. On admission the patient was taking 40 mg of furosemide (Lasix) o.d. and .15 mg of digoxin o.d. Physical examination showed mild obesity with grade ii/vi systolic ejection murmurs and S_4 gallop. Ocular examination revealed a visual activity of 20/200 on the right and 20/400 on the left, with no increase in intraocular pressure. The patient was oriented and her initial names-learning test score was 54.

On the second hospital day Ms. B underwent a right intracapsular cataract extraction under local anesthesia, as described in case 1, without complication. Twenty-six hours after surgery, at 9:00 a.m., the patient was observed sitting on a stool picking at imagined bugs and giggling. The most recent medications she had received were 200 mg of secobarbital the night before the operation; 50 mg of I.M. diphenhydramine (Benadryl), 10 mg of I.M. prochlorperazine (Compazine), and three drops of 1% Cyclogyl in the right eye at the time of the operation; and 1% atrophine twice since the operation. The operated eye had been examined about 1 hour before the episode. Physical examination 45 minutes after the beginning of the episode revealed a temperature of 37.2°C, a pulse of 100, and slightly moist skin. The patient's right eye was patched, and her left pupil was mydriatic. Mental status examination revealed a change of affect from euphoric to tearful within minutes. The patient was hyperactive, picking at her arms while giggling and saying, "This is wonderful." She admitted that she saw unusual things but refused further comment. There was gross disorientation to time, but the patient knew which hospital she was in and recalled her surgery. She recalled one of three objects after five minutes but could not recall her home address. Her names-learning test score was 10.

On repeat mental status examination 3 hours after the onset of the episode the patient was normal. She recalled that "it was like a dream. I saw different things coming by, people, the girl next door, and other things."

Anticholinergic agents and sedatives were withdrawn, and the patient had no further episodes. On the eighth hospital day she underwent a left intracapsular cataract extraction without subsequent delirium.

Comparison Group

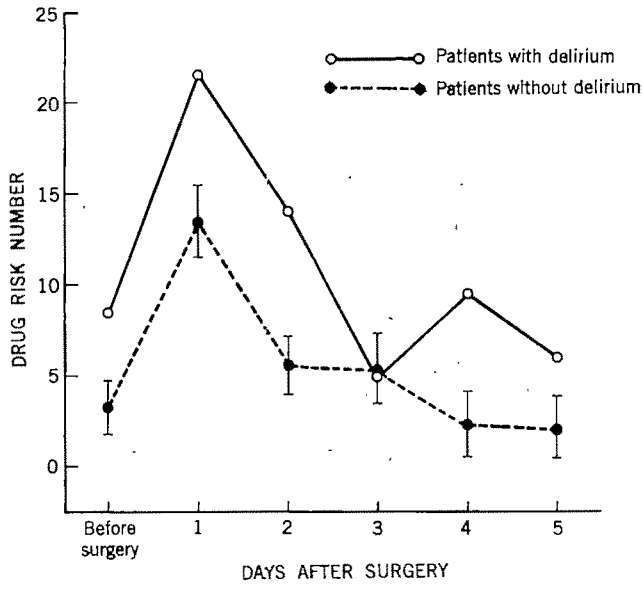
Twenty-five of the 27 subjects in this study did not have psychiatric sequelae to cataract removal. The mean age of these control subjects was 71.9±11.1 years (range, 54–96). The mean age of the control subjects is similar to that reported for patients with postcataractectomy delirium (see table 1). Twenty-two of the control subjects were white, and 3 were black; 17 were women, and 8 were men; 9 had had previous psychiatric illnesses; 1 had had a previous postcataractectomy delirium; and 8 had a positive family history of psychiatric disorders.

Table 2 allows comparison of the data for our control subjects and those for patients with delirium reported in the literature. Two of the control subjects (8%) were suspected of alcohol abuse, compared with a mean of 22.7% of the 66 patients with delirium for whom such data were available in the literature. None of the control subjects was suspected of having chronic organic brain syndrome, compared with 33.3% of 63 delirium patients in the literature. Fourteen (56%) of the control patients, however, were suspected of having some other form of psychiatric illness, compared with 13% of 23 delirium patients. Five (20%) control subjects experienced some postsurgical complication, compared with 16% of 81 delirium patients. Three (12%) control patients had a preoperative medical diagnosis of renal disease, compared with 13.6% of 59 delirium patients. Finally, 19 (76%) control patients had some other preoperative medical diagnosis, compared with 8.5% of 47 delirium patients.

Comparison of preoperative laboratory results for patients who did or did not experience postcataractectomy delirium in the literature was not possible. Comparison of our laboratory data by t test (one-tailed) showed that the 2 patients who experienced delirium had significantly elevated BUN and creatinine levels ($p<.001$). Similar comparisons were not significant for hematocrit, white blood cell count, serum sodium, serum potassium, serum bicarbonate, serum glutamic-oxaloacetic transaminase, lactic dehydrogenase, bilirubin, alkaline phosphatase, serum calcium, and creatine phosphokinase.

Exposure to psychoactive drugs was measured by drug risk number values; these are displayed in figure 1. The mean drug risk number of patients who experienced delirium was greater than the mean plus the

FIGURE 1
Mean Drug Risk Numbers of 2 Patients With Postcataractectomy Delirium and 27 Patients Without Postcataractectomy Delirium[a]



[a]SEM = 1.96.

SEM (1.96) drug risk number of the control subjects for the preoperative period and for postoperative days 1, 2, 4, and 5.

DISCUSSION

The occurrence of 2 cases of postcataractectomy delirium in 27 subjects in this series does not allow reliable statistical comparison. Nevertheless, there are several points of interest. The 7.4% incidence of delirium in this series is consistent with the incidence in the literature. The mean age (71.9±11.1 years) and age range (54–96) of our control subjects was not remarkably different from those of delirium patients noted in the literature (see table 1).

The theory that delirium is associated with older age (8, 11, 21) appears unlikely. When comparison was possible, no significant differences were found between our control subjects and our patients with delirium or those in the literature for race, sex, previous psychiatric illness, previous medical disease, family history of psychiatric disorder, or postoperative eye complications. Thus, the suggested association of delirium with poor general health (4, 21, 22, 26) or with any previous psychiatric illness (3, 11, 17, 33) is not supported by our findings. (The only patient with chronic organic brain syndrome in this series developed postcataractectomy delirium. This is not mathematically consistent with the 72.2% frequency of delirium in patients with chronic organic mental syndrome found by Linn and associates [35].)

The apparently significant decrease in renal function of the 2 patients with delirium in our study was unexpected. This finding was reinforced by a slight elevation in BUN and creatinine in another patient with postcataractectomy delirium who was not part of this study. It would appear that the hypothesis of renal autotoxicity advanced by Fromaget in 1900 [6] merits further investigation.

The bilateral patching or sensory deprivation hypothesis appears improbable on the basis of the literature (see table 3). Both of the patients in our series who developed delirium had experienced no apparent sensory deprivation.

Our results suggest that postcataractectomy delirium may be an acute anticholinergic syndrome. The clinical presentation and course of the two syndromes is similar (51). This similarity was noted as early as 1910 (52). It is known that mydriatic agents can induce delirium at therapeutic doses (53, 54), and Dameshek and Feinsilver (55) demonstrated that mydriatic delirium was caused by anticholinergic toxicity. Further, the 2 patients with delirium in our series received greater exposure to all psychoactive agents (see figure 1). Janowsky and associates (50) postulated that such agents may act synergistically to produce acute anticholinergic syndromes.

Most impressive was the rapid clearance of delirium in 1 of our patients for 2 hours after administration of physostigmine. It has been suggested that rapid improvement after physostigmine constitutes proof that a patient had an acute anticholinergic syndrome (49, 50). There are several explanations for the cholinergic gastrointestinal side effects precipitated by physostigmine in this patient. Most probably, the dose of physostigmine was excessive for the degree of anticholinergic intoxication. It seems likely that this patient had anticholinergic intoxication. It seems unlikely that the delirium would have spontaneously cleared for a period consistent with the known duration of action of physostigmine. In short, it seems unlikely that the physostigmine was totally unopposed by exogenous anticholinergic substances.

Finally, the patient may have had isolated CNS toxicity. Here it may be postulated that the mydriatic agents would have access to the vitreous because of the disruption in the usual ocular physiology in which the drug would be taken up by the neurons and transported by anterograde axonal transport into the CNS (56). This would result in isolated CNS anticholinergic toxicity. Given these circumstances physostigmine would cause clearing of CNS anticholinergic toxicity while precipitating peripheral cholinergic toxicity.

The etiology of delirium after cataract surgery remains unclear. This study suggests that the syndrome represents anticholinergic intoxication. Further work needs to be done to test this hypothesis; to clarify the association of postcataractectomy delirium to chronic organic brain syndromes; and to establish any relationship of postcataractectomy delirium to renal pathology.

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Adult Mental Health Services in a Health Maintenance Organization

BY SIMON H. BUDMAN, PH.D., JUDITH FELDMAN, M.D., AND MICHAEL J. BENNETT, M.D.

The authors describe an adult mental health service within a health maintenance organization and present information on the department's setting, staffing patterns, programs, and philosophy. They believe that the emphasis on brief focal psychotherapy, the staff of mental health care providers who are committed to this type of setting, and the thorough integration of the program into the larger HMO system all contribute to the provision of high-quality services at a reasonable cost.

HEALTH MAINTENANCE ORGANIZATIONS (HMOs) are prepaid group practices characterized by integration of facilities, voluntary enrollment, and an emphasis on preventive services (1). Recent surveys indicate that 7 million Americans now belong to such plans and that this figure is increasing (2). At first, prepaid health plans provided mental health services only on a limited basis, usually through a special rider at extra cost. The federal HMO Act of 1973 (P.L. 93-222) (which requires that qualified HMOs must provide short-term outpatient mental health evaluation and treatment to all members) is contributing to an increase in the number of HMOs providing direct mental health services as a standard benefit.

In this article, we will describe an HMO mental health department that is heavily engaged in direct patient care. We will examine the nature of the setting, the professional staff, referral patterns, and treatment modalities and discuss some of the unique aspects of such a system.

THE SETTING

The Harvard Community Health Plan (HCHP) in Boston, Mass., was started in 1969. Until Jan. 1, 1977, HCHP had a contractual relationship with Massachusetts Blue Cross and 10 private insurance companies. This contractual relationship has been phased out, and the plan is now responsible for marketing, member services, billing, and premium collection. All plan physicians have teaching appointments at the Harvard Medical School, and medical patients are hospitalized mainly at Harvard teaching hospitals.

At present, the plan has two centers: the original Kenmore Center with 38,000 members, and the newer Cambridge Center with 31,000 members (only the former will be described in this paper). The Kenmore Center currently employs the full-time equivalent of 40 physicians and 32 nurses, who are organized by specialty into separate areas of one building.

The HCHP monthly premium is approximately 20% below that of a Blue Cross-Blue Shield Master Medical Policy. It covers all inpatient and outpatient services with a small copayment for each visit.

The majority of plan members are between the ages of 20 and 40 and subscribe to the plan through their place of employment. Because the plan is offered to an entire company or organization, members vary widely in income and occupation. Four percent of the population is indigent and their care is supported through a contract with the Massachusetts Welfare Department. Less than 1% of the population is enrolled under a special health cost supplementation program of the U.S. Public Health Service.

THE MENTAL HEALTH DEPARTMENT

The adult mental health department includes the full-time equivalent of 3 psychiatrists, 3 psychologists (Ph.D. level), 1 social worker (M.S.W. level) 2 psychiatric nurses (M.S. level), and one half-time alcoholism counselor. (There is also a fully staffed child psychiatry service, which will not be described here.) The average age of the staff is 38 and the average clinical experience 13 years.

Staff members are selected on the basis of sound psychodynamic training, interest and experience in working in a high-pressure medical setting, interest in a group practice, and commitment to problem-oriented short-term psychotherapy. Applicants are interviewed by several members of the mental health department and also by members of other departments within the plan. The psychiatrists and psychologists are members of the HCHP physicians group and are part of the same salary contract as the other plan physicians. Psychiatric nurses belong to planwide nursing groups and

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committees. The department does not have a hierarchical structure: all staff members share equally in evaluations and treatment, regardless of discipline. Supervision within the department is mandatory for new clinicians for 6 months and optional thereafter. A full-time mental health staff member (40 hours a week) spends about 70% of his or her time doing clinical service. For part-time staff, this fraction is even higher. Staff time not spent in direct clinical service is used for consultation, administration, and staff meetings.

The mental health unit is located entirely within the Kenmore Center. Like other departments in the plan, it participates in the plan's automated record system and also maintains its own appointment system and operates within its own budget. In 1978 the mental health department had 20,000 outpatient visits, representing about 8% of total plan visits. A recent survey indicated that about 15% of the plan members who use health services use the mental health department at some time during their membership.

Before January 1976, when the Massachusetts Mental Health Law took effect, the plan excluded from coverage individuals with chronic or recurrent psychiatric illness and offered only brief therapy. HCHP is now legally obligated to offer the equivalent of $500/year of outpatient mental health service to all members. Rather than contracting out for the additional services, the department chose to expand its programs. Currently, 5% of the monthly premium is used for mental health benefits. The benefit structure provides 20 outpatient sessions per year at the copayment fee, which is usually $1. One hour of individual treatment or 1.5 hours of group treatment equals one visit. Additional visits are billed at the rate of $40 per session for individual treatment and $10–$20 per session for group treatment, depending on income. HCHP also provides 60 days of full hospitalization or 120 days of partial hospitalization (in a psychiatric hospital) and 30 days of inpatient alcohol or drug detoxification per year.

PRIMARY CARE AND MENTAL HEALTH SERVICES

When a family joins HCHP, each adult member chooses a physician/nurse primary care team from the internal medicine department. All subsequent medical visits (routine and emergency) are scheduled with that team. Other departments are generally used by the primary care team as consultant specialists. The triage (emergency) area, staffed by nurses with physician backup, provides care to new members who have not yet chosen a primary care team. It also provides off-hour coverage to all plan members.

At HCHP, mental health benefits are broad and available to all members, but mental health care is also seen as an essential function of the primary care team. In order to reconcile the expectations for specialized services with the commitment to a primary care model, the department has attempted to create and sustain a high level of awareness throughout the organization of the importance of social and psychological issues in medical care. Consequently, such issues are addressed throughout the system rather than being relegated to the mental health specialist.

Mental health professionals have conducted in-service presentations and seminars on medical-psychological issues for both primary care trainees and staff doctors and nurses. In addition, mental health staff meet regularly with primary care, obstetrics/gynecology, and surgical staff to review cases and problem issues. There are also several joint clinical programs, such as continuing care and a crisis group. The social worker in the mental health department consults in primary care areas and helps providers deal with social, emotional, and economic issues in their practices. Finally, physical contiguity allows for frequent informal discussions about shared patients and problems.

TREATMENT IN THE MENTAL HEALTH DEPARTMENT

After the primary provider completes a form stating the nature and urgency of the patient's problem, the patient is scheduled for an appointment on the basis of his or her extent of symptomatic distress and functional impairment. After the evaluation is completed, the therapist sends a note back to the referring provider with a diagnostic impression and recommendations for no treatment, referrral back to primary care, or treatment within the mental health department.

The predominant mode of treatment in the department is focused short-term individual or conjoint couples therapy (defined as 20 or fewer sessions, but usually fewer than 10). Another approach is the crisis group, run jointly by a mental health provider and a triage nurse, which meets twice a week for an hour and a half. The group has a rotating membership and leadership. Patients are referred during a life crisis (marital separation, death of relative, etc.) and may attend the group for up to 8 sessions. This group is focal, active, task-oriented, and very supportive. Initial data (3) indicate that patients have an overwhelmingly positive response to the group and often require no further treatment.

Based on the favorable follow-up data from the crisis group, there has been increasing work with short-term groups for selected problems. These groups are used to treat individuals and couples with developmental or life-stage issues and to provide a "corrective" social experience for those with focal interpersonal concerns (4, 5). In 1978 about 300 of the 1,300 new patients were seen in short-term group therapy. Some groups are open-ended and have rotating membership; others are closed groups and run for about 15 sessions. There is often an attempt to set up groups with fairly homogeneous membership (couples, young adults, persons dealing with mid-life issues, etc.).

Some staff members have specialized training and may treat patients with hypnosis, direct sexual therapy, etc. Such techniques may at times be used as an adjunct to other forms of therapy. Patients with chronic mental illness who require support, medication, and structure may be followed in several ways: by their primary care team, by a mental health provider (in widely spaced half-hour sessions), or in the continuing care program. This program (6) is run by the mental health department, with a nurse from internal medicine participating. A large, informal group meets twice a week, and patients may attend as often as they wish. The group functions to provide support and socialization. The leaders hold brief individual sessions with patients as needed to regulate medication. Patients are often referred after hospitalization in the hope that this follow-up may reduce the frequency or severity of recurrent illness.

In addition to the approaches described above, since January 1976, HCHP has provided long-term, open-ended individual or group treatment for those who need it (7). This program is very carefully monitored. A therapist referring a patient for long-term treatment writes an extensive evaluation, which must be approved by a committee of mental health department members. Patients continue with the referring therapist if he/she has time available (each staff member may carry up to 4 long-term individual treatment patients) or are assigned to another staff member. Cases are scheduled to be reviewed yearly. The criteria for referral were developed by an ad hoc committee of mental health department members after discussion within the entire group and emphasize the patient's need for extended treatment rather than simply his or her capacity to benefit from it. Although department staff initially feared they would be inundated by referrals, between Jan. 1, 1976, and Jan. 1, 1979, only 114 patients were referred for long-term therapy. We estimate this to be less than 2% of the patients seen during this period.

In the alcohol abuse program, a psychiatrist and an alcoholism counselor provide information and backup to primary providers. They conduct educational groups, see patients and families in consultation, and occasionally provide direct service—usually short-term treatment.

Staff members rotate emergency coverage on a weekly basis. A mental health provider is available 24 hours a day by portable pager for telephone consultation to the internist or triage nurse on call. The mental health provider may talk with the patient directly or arrange hospitalization when indicated. Time is also allotted during the working day for unscheduled patients who require immediate intervention.

Since HCHP does not have its own hospital, arrangements have been made to hospitalize plan patients at a nearby private psychiatric hospital. To be approved and covered, hospitalization must be arranged by a member of the mental health staff. The department has worked with the hospital to develop programs for partial hospitalization (overnight admissions and day care). A psychiatric nurse maintains daily contact with the hospital treatment team (not employed by HCHP) to help coordinate inpatient treatment and aftercare. In 1978, there were 43.4 hospital days per 1,000 members, representing 116 patients hospitalized, with an average length of stay of 14.1 days.

An individual patient may be involved at various times in several of the above treatment approaches and may return to a therapist for several courses of brief treatment. With most short-term approaches, it is assumed that the working through process continues after formal therapy ends. Brevity of treatment is enhanced by early access and by favorable patient and staff attitudes toward psychotherapy.

UNIQUE ASPECTS OF THE HCHP MENTAL HEALTH SYSTEM

Strupp and Hadley (8), in describing a conceptual model for evaluating psychotherapy outcome, propose that there are three "interested parties" in these evaluations: society, the individual, and the mental health professional. A tripartite model also fits well in understanding the unique demands of providing mental health services within this HMO system. At HCHP, the three interested parties are the institution itself, the patient, and the provider. Institutional demands are experienced by providers in an HMO much more strongly than they are in other mental health delivery systems. The treatment relationship in private practice can be insulated from institutional or societal demands. Even when an insurance company or the state is paying for treatment, the interests of those institutions often can be ignored or kept at a low priority by the clinician and patient. The HCHP system differs in that the plan is the insurance company. Clinicians wear three hats simultaneously: they represent the plan in allocating its finite mental health resources, they represent the patient's interest in "feeling better," and they represent their own perspective as clinicians with an interest in enhancing the patient's personality structure and coping style. The tension generated by these multiple perspectives is considerable.

Overtreating (allocating too much time to a patient) has direct institutional implications. In the short run, other patients may go without or have to wait for therapy. There may be added clinical demands placed on providers in mental health and other departments because of the backlog of patients awaiting treatment. In the long run, excessive waiting time may mean decreased consumer satisfaction and withdrawal from the plan or increased premiums in order to hire more therapists to treat patients for a longer period. Of course, in increasing premiums, the institution again risks losing members.

Undertreating (allocating too little time to a patient) has implications from all three perspectives: the un-

dertreated patient may 1) feel shortchanged and choose to quit HCHP, 2) seek emotional support elsewhere in this closed system, making excessive demands on other health care providers, or 3) return to the mental health department for additional treatment. Undertreatment also has major implications for the satisfaction of mental health practitioners. For clinicians with an understanding of psychodynamics and personality theory and a wish to be helpful to others, morale would plummet if it were felt that treatment was only superficial and temporary and did not influence the patient's coping and adaptational styles.

Thus mental health providers within the system must maintain multiple perspectives quite unlike the perspective(s) maintained by providers within other mental health systems. These multiple perspectives generate a dynamic tension in each provider, since providers exist within and are responsible to a peer group (the mental health group practice). This leads to a careful scrutiny of how mental health services are being allocated and a tendency toward problem-oriented psychotherapy.

It appears that the HCHP mental health department can treat most patients briefly without constantly undertreating because of the unique structure of the organization. A patient who finishes a course of psychotherapy does not leave the system. He or she maintains membership, continues to see his or her primary care providers as needed, and has ready access to the mental health department. The overall institution provides the patient with a supportive matrix of services that can take up where the psychotherapist leaves off.

THE FUTURE

The major challenge facing the mental health department is to provide total, high-quality care to a large population with varied needs and to keep costs at an acceptable level. The department is now moving in the direction of more group psychotherapy, better crisis and emergency treatment, preventive programs, and research and evaluation.

An NIMH-supported project on preventive mental health is studying the economic feasibility of providing plan members who have recently undergone marital separation with a program called "Seminars for the Separated" (9). This series of didactic and experiential meetings is aimed at lessening the trauma associated with marital separation.

There is an active concern within the department about education, scrutiny, and review. In an attempt to generate evaluative research, staff members meet weekly on an informal basis to examine the short-term group psychotherapy program. It is the goal of this research group, composed of interested staff members, to monitor services, evaluate outcome, and to help clarify the quality and cost-effectiveness of this program.

CONCLUSIONS

The experience of the mental health department at HCHP suggests that a complete range of mental health services can be provided in a cost-effective manner. The total cost of all services—adult and child, inpatient and outpatient—is approximately $2 per member per month.

The opposing tensions within this system necessitate an emphasis on brief, problem-oriented treatment. In order to make this approach work, the mental health department must be large enough to support a variety of programs and be fully integrated into the total health care system. Mental health providers must be committed to the concept of primary care and to the health plan as a whole.

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A Behavioral Program for the Treatment of Chronic Patients

BY JOHN F. HOFMEISTER, M.D., ALBERT F. SCHECKENBACH, PH.D., AND SHIRLEY H. CLAYTON

The authors report the development of a short-term behavioral therapy program for chronically hospitalized schizophrenic patients who had responded only minimally in a therapeutic community. The basic program was a token economy, which concentrated on personal care activities, productive activities, and leisure activities, combined with drug treatment. During the first 9 months of the program 105 patients were admitted and 79 were discharged—24 to nursing or group homes and 47 to their own home or family. At 1-year follow-up 62 patients were still in the community without any readmissions. The findings that a token economy can be effective with chronic patients at comparatively little additional expense has broad implications for custodial care facilities with limited resources.

SINCE THE EARLY 1960s Fort Logan Mental Health Center has been a leader in the development of a therapeutic community. Maxwell Jones, a leading advocate of the concept of the therapeutic community, was first a visiting consultant and then resident consultant at Fort Logan. Under his guidance a strong program was developed that benefited a large number of Fort Logan patients (1) and significantly influenced the development of therapeutic programs throughout the world. However, Kraft and associates (1) found a hard-core group of patients at Fort Logan—primarily chronic schizophrenics—who were minimally responsive to milieu treatment.

Over the last 5 years the psychiatric inpatient population of Fort Logan has been shifting to an increasing percentage of chronic patients with severe deficits in interpersonal functioning. These patients were poorly equipped to involve themselves in the vigorous give-and-take of an active therapeutic community. They characteristically drifted to the periphery of the ward treatment program as passive observers. A few assumed a deviant role. Others became scapegoats of the community, e.g., the clown, the stupid one, the crazy one. Despite the staff's vigorous efforts to intervene in this destructive group dynamic, polarization of the patient subgroups hardened. These patients were often frightened by the staff's attempts to involve them. The staff experienced frustration in trying to draw them out. Active intervention with these patients showed a gradual but steady decrease. The patients themselves became frustrated and discouraged by their repeated failures. Their returns to the community became less frequent and of shorter duration. The length of their hospitalizations began to increase, and a number became chronically hospitalized.

The increasing length of hospitalization among these patients and their relatively poor treatment response had serious implications. Dunham and Weinberg (2) reported that length of hospitalization has been shown to be inversely correlated with the probability of release. Gurel (3) and Ullman (4) found that of patients hospitalized for 2 years, only about 6% are discharged. Chronic patients have extremely high rates of rehospitalization (5, 6). Van Putten (7) cited 8 studies to show that milieu therapy fails to produce significant improvement in chronic schizophrenic patients.

Staff concern for the chronic patients at Fort Logan prompted a search for an alternative treatment modality for them. In 1968 Ayllon and Azrin (8) reported on a behavioral program employing a token economy with long-term chronic patients at Anna State Hospital in Illinois. These patients had been hospitalized for an average of 16 years before the behavioral program began; almost all of them showed a significant improvement in work performance. Lloyd and Garlington (9) reported similar success in increasing the self-care behavior of chronic patients. Schaefer and Martin (10) worked to improve the apathetic behavior of chronic patients, with impressive results. Since then there have been a large number of studies employing behavioral programs with chronically hospitalized patients. The current literature has been reviewed by Hagen (11) and Stahl and Leitenberg (12).

The decision to set up a behavioral treatment unit at the Fort Logan center to treat the most severely disabled patients was made only after considerable discussion, evaluation, and a good deal of apprehension.

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The entire program was scheduled for reevaluation at the end of 9 months. A group of the most severely disabled and chronic patients, with the longest inpatient stays, was targeted for special, continuing follow-up.

METHOD

The behavioral treatment unit was established in November 1975; it had a capacity for 20 inpatients. The treatment staff consisted of 1 clinical administrator, 1 psychiatrist, 1 half-time psychologist, 1 social worker, 7 nurses, 7 mental health workers, and 1 half-time activity therapist. An outside consultant was selected who had particular expertise in behavioral treatment modalities with chronic patients. He gave 6 seminars to the staff to teach them the basics of behavioral therapy and helped design the ward treatment program. Staff members were also provided with basic textbooks on behavioral therapy for readings and reference (8, 13–15).

Patient Profile

The patients selected during the first 9 months for the behavioral unit were those who were relatively refractory to the therapeutic community based on prior history of treatment. The sample consisted of male and female patients; the average patient was between the ages of 20 and 55 and either had never married or was no longer married. Each patient had had multiple prior psychiatric hospitalizations and had been diagnosed as schizophrenic during 2 or more hospitalizations. Each was admitted to Fort Logan Mental Health Center only after several failures in a number of outpatient programs. The patient's relationships with parents and siblings were either ruptured or severely strained; previous friends were estranged. The average patient had either never held a job or worked only sporadically; he or she was usually supported by public assistance or social services. This group of patients ranked highest on a Fort Logan scale measuring disruptive behavior; their average score was 74%, compared with 50%–60% for all of the other adult inpatients. Most of these patients had previous admissions to the therapeutic community at Fort Logan; a representative sample of 30 patients had prior goal attainment scores more than one standard deviation below the mean for the entire inpatient population. The targeted group of chronic patients (average prior inpatient stay=1,200 days) were treatment failures on the therapeutic community, manifested by a gradual decline in socially adaptive behavior, an increase in maladaptive behavior (e.g., violent temper tantrums, negativism, extreme isolation), and chronic hospitalization.

The Program

A multilevel system for performance and privileges was developed. Each level was designed to gradually shape behaviors needed for successful adjustment outside the hospital. All patients received the basic necessities of hospital life (i.e., food, bed, medical care) without having to earn them. Positive reinforcement (rewards) for adaptive behaviors was the basis of the program. Maladaptive behaviors were usually treated by extinction (ignoring the behavior). The basic program was a token economy in which patients earned points on a scale of performance for adaptive behaviors. Adaptive behaviors were defined as those useful for life outside the hospital. The points could later be exchanged for luxuries on the ward (i.e., cigarettes, extra coffee, candy, cosmetics). Extremely disabled patients who could not comprehend the token system received tangible items directly for adaptive behavior. Written contracts were established for less disabled and greatly improved patients. These contracts, which were drawn up in cooperation with the patients, enabled them to obtain special needs from the staff in exchange for adaptive behaviors. Patients moved from one level of the program to another as their needs and performance changed.

The token economy program concentrated on personal care activities, productive activities, and leisure activities. Instruction in basic skills was included in each activity program. Personal care activities included personal hygiene, oral care, use of toilet items, clothing, and room care. Used clothing, jewelry, games, cards, and so forth could be purchased with points in the hospital's used-clothing shop or by shopping trips to the community for those who had their own money. Basic toilet articles (e.g., deodorant, soap, shampoo) were provided free on request to each patient. Productive activities included an arts and crafts program, physical exercises and games, cooking and meal planning, sewing, and a sheltered workshop on the grounds. Patients who elected to work in the sheltered workshop received financial compensation that met or exceeded the minimum wage as well as points. Payment for all activities was scaled to performance, appropriate behavior, and on-time attendance. Leisure time activities included individual and group games, leisure planning, dancing, camping, bowling, trips, and swimming.

Whenever practicable, the scope of the program was expanded through the use of community services and facilities, e.g., vocational or job placement services, stores, swimming pools, museums. A halfway house on the hospital grounds was used to assist patients' transition to the community. Referral to a local mental health center was arranged prior to discharge to promote continuity of care.

The medium of payment for adaptive behaviors consisted of points stamped by the staff on each patient's card. This system was chosen to minimize the trading of points from one patient to another by maladaptive behaviors (i.e., threats, sexual favors, buying friends). Points were stamped in multiples of 10 and were cancelled by the staff with a paper punch when they were exchanged for goods or services. Staff attention

proved to be a very potent reinforcer and was directed toward patients engaged in adaptive behaviors. Points were given immediately after adaptive behaviors, coupled with an explanation of how the points were earned. At the same time social approval was presented consistent with the patient's level of achievement and capacity to perform the behavior. Patients' earnings in 19 separate categories were recorded each day by the treatment staff. Areas of strength or weakness and abrupt or gradual changes in performance could be readily assessed by a review of these records.

Treatment was individualized on admission by placing the patient at a level corresponding to his or her capacity to function, i.e., tangible reinforcer level, token economy level, or contract level. After a period of staff observation and review of the patient's recorded performance, special needs or problems were targeted for an individual operant program; these problems included eating with the hands, severe weight loss, neglect of self-care, wrist cutting, mutism, extreme isolation, and assaultiveness. Each patient's treatment program was reviewed weekly by the treatment team, and changes were made after review of the performance records, individual program records, and clinical observation.

Just as adaptive behavior resulted in positive reinforcement, extreme maladaptive behavior met with negative consequences. Most inappropriate behavior was ignored (extinction process). Extreme maladaptive behavior included assaultiveness, extreme agitation, serious destruction of state property, self-mutilation, serious threats, and elopement. These behaviors resulted in the patient's separation from staff and patients; the length of the separation depended on the patient's regaining control of his or her behavior. There was no overall system of negative consequences for the entire ward (e.g., fines), but negative consequences were employed in selected individual treatment plans. Restraints and/or seclusion for a specified time was used as a last resort to assist the patient in regaining control. Patients in restraints or seclusion were checked every 15 minutes by a staff member. The average time spent in seclusion or restraints was approximately 1 hour.

Each patient received a complete medical and neurological examination, including specialized studies when indicated. Antipsychotic and other psychoactive medications were used in accepted dosages (75–2000 mg of chlorpromazine or the equivalent dosage of another antipsychotic medication). In addition, megadoses of haloperidol (100–160 mg) were employed for several severely ill patients whose psychoses did not respond to usual doses of antipsychotic agents.

The program was evaluated after 9 months by the administration of a staff questionnaire and review of several objective measures that might be expected to correlate with effectiveness, i.e., length of hospitalization, recidivism rate, disposition of patients, property damage, sick leave used by the staff, and staff requests for transfer.

RESULTS

A total of 105 patients were admitted to the behavioral treatment unit during the first 9 months of operation, and 79 patients were discharged during this period. The average inpatient stay was 58 days. The chronicity and extreme disruption of interpersonal functioning in these patients led to a general expectation among hospital staff that a short-term treatment program averaging 6 weeks would achieve little more than a protective community placement, i.e., nursing home. In contrast to these expectations, only 24% of the discharged patients (N=19) were placed in nursing homes. Fifty-nine percent (N=47) were discharged to their own home, apartment, or family. Six percent (N=5) were discharged to a boarding home or lodge (work-oriented group home). Five percent (N=4) were discharged against medical advice. Four percent (N=3) transferred to other wards or hospitals. There was one death from a preexisting cardiac condition. Thirty percent of the patients (N=32) worked regularly in the industrial therapy workshop. With one exception, none of the patients was discharged to a behaviorally oriented day-care program (there was only one such program in the area at the time of this study).

The course of the discharged patients was reassessed one year after discharge. Excluding the deceased patient, 80% of the patients (N=62) were in the community without any readmissions. Twelve percent (N=9) were readmitted to Fort Logan with an average stay of 32 days; 2.5% (N=2) had 2 admissions with an average stay on each admission of 43 days; and 5.5% (N=4) had 3 readmissions with an average stay on each of 37 days. The one patient discharged to a behaviorally oriented day-care program was not readmitted.

The target population of long-term chronic schizophrenics (N=6) with an average prior inpatient stay at Fort Logan of 1,200 days was discharged within 9 months to less intensive treatment modalities, where the patients' average length of stay was 6 months. Follow-up 1 year after discharge showed that 1 patient was readmitted for 129 days; the others were in the community with no readmissions to Fort Logan.

During this study there were no suicides or serious self-inflicted injuries among the patients. Property damage on the ward was minimal (one broken window). There were two staff requests for transfer to other units. One staff member cited dislike of the behavioral treatment modality as a reason for requesting a transfer; the other cited difficulty working with chronic patients. Both staff members who transferred to other units subsequently left employment at Fort Logan. Staff turnover on the behavioral unit was the lowest among the adult psychiatric inpatient units at Fort Logan. Staff sick leave showed an abrupt rise during the first 2 months of the program to twice the hospital average (1.4 versus .746 days per employee per month). From the third to the ninth month of the program, the

sick leave of the behavioral unit staff remained at or below the hospital average. At 9 months the staff unanimously rated the behavioral program as effective; 61% rated it "extremely effective" and 39%, "moderately effective." The clinical status of the patients at discharge was evaluated by the attending psychiatrist; the mean rating was moderately improved.

DISCUSSION

This study indicates that a behavioral treatment program in combination with adequate diagnostic workup and drug treatment is effective in treating disabled chronic patients. This finding concurs with the results obtained by a number of other researchers. These studies have been reviewed by Stahl and Leitenberg (12). However, few studies have focused on the effect of short-term hospitalization with chronic patients. The results at Fort Logan Mental Health Center suggest that recently admitted patients with a severe chronic illness can improve sufficiently for discharge within 2 months (the average inpatient stay was 58 days). Patients with a long prior inpatient stay required a much longer period of hospitalization before they were ready for discharge. (The average inpatient stay was 6 months). The present practice at Fort Logan is to admit directly to the behavioral unit any adult patients with a severe psychotic illness associated with very low levels of performance and very poor interpersonal skills. In addition, adult patients on the therapeutic communities who fail to show significant progress within 6 weeks are regularly evaluated for transfer to the behavior modification unit.

The clinical impression at Fort Logan is that antipsychotic medications in adequate dosages work synergistically with a behavioral program for patients admitted in acute decompensation of chronic disease. However, this clinical impression has not been subjected to critical long-term evaluation.

The treatment staff has begun an evaluation of the posthospital adjustment of chronic patients treated on the behavioral ward. Preliminary findings are that a high level of performance in self-care activities, domestic skills, crafts, work, and other activities is not predictive of optimum posthospital adjustment. Of the high-point earners (600 points per day or more), those who are able to learn to form friendships and relate to others in a mutually satisfying manner have the best posthospital course. High-point earners who relate to peers by emotional exploitation (threats, manipulation, demeaning others), extreme submissiveness, or avoidance of peers combined with continued efforts to be a favorite of the staff show a much less adaptive course after hospitalization. The behavioral program at Fort Logan is now being revised to increase the emphasis on teaching and reinforcing interpersonal skills directed to appropriate conversation with others, helping others to do for themselves, cooperatively working with others, and cooperative leisure activities.

The cost of a behavioral program is another important consideration in these times of rapidly accelerating medical care costs. The cost of the behavioral treatment ward at Fort Logan is roughly equal to that of the center's therapeutic communities i.e., $100 per patient per day. The staff-to-patient ratio is 1:1, and the psychiatrist-patient ratio is 1:20. A custodial placement in the Denver area is far less expensive—$30 per day. However, costs are favorable if length of hospitalization is considered. The present cost for a 58-day course of treatment on the behavioral unit is $5,800; a year of custodial care in the Denver area costs approximately $10,900.

Preliminary indications in the literature are that a token economy can be implemented with comparatively little additional expense. This finding has broad implications for custodial care facilities with limited resources (e.g., nursing homes) that are seeking to upgrade the treatment of patients. Foster (16) analyzed the comparative costs of running a custodial ward and a token economy ward for 32 patients for 1 year and found that the cost of the token economy ward was $424 more than that of the custodial ward. Hagen (11) cited several successful behavioral programs that cost no more or little more to implement than traditional custodial care (up to $2.60 per patient per month). In addition, he found that several programs resulted in considerable savings for the institution in terms of decreased destruction of property, use of linens, and use of medications.

These findings have prompted the staff at Fort Logan to initiate an active behavioral consultation program with several nursing homes in the Denver area to assist them in developing token economies for their patients. The long-range goal is to provide long-term active behavioral treatment programs in the community for the 24% of our patients who do not respond to our short-term treatment program.

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The *Journal* will continue this year its policy of early review of annual meeting papers. Final versions of papers that are ready before the Chicago meeting will be accepted for review. Do *not* submit your paper early if you anticipate substantive revisions after discussion at the meeting. Manuscripts should be submitted in triplicate, double-spaced throughout, and prepared in conformance with *Journal* style specifications, including length limitations. Papers written in "speech" format should not be submitted.

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The Impact of Normal Volunteers on a Psychiatric Research Unit

BY PHILIP GOLD, M.D., YOLANDE B. DAVENPORT, M.S.W., THOMAS WEHR, M.D., AND FREDERICK K. GOODWIN, M.D.

Normal subjects were admitted to a psychiatric unit devoted to the study and treatment of affective illness. Initially the authors were concerned about the difficulties volunteers might have living with psychiatric patients. However, the volunteers adjusted with relative ease, while the patients' depressive symptoms were exacerbated. Their confrontation with the volunteers' "normality" triggered an acute awareness of their underlying sense of failure. This response is analogous to depressed patients' reactions before discharge, when they struggle not only with their special vulnerability to separation and loss but with inevitable challenges to their fragile self-esteem during reintegration into the outside world. The presence of normal volunteers highlighted these issues and led to increased therapeutic work and considerable resolution.

IN CLINICAL PSYCHOBIOLOGICAL research it is essential that baseline data be obtained from normal subjects in a controlled setting. For over 20 years, "normal volunteers" have been hospitalized on most inpatient research units at the Clinical Center of the National Institutes of Health to provide control data for clinical projects. The psychiatric units were the only inpatient services that had not established the tradition of admitting normal volunteers. Counter to the strong local tradition, which was embedded in a vague fear of some unspoken risks, we admitted four college-age volunteers for a 3-month stay on a clinical research unit devoted to the study and treatment of affective disorder. Although a fairly extensive literature exists concerning issues of patient care on psychiatric research units (1-9), to our knowledge there have been no reports assessing the effects of resident normal control subjects on the life of a psychiatric research unit organized as a therapeutic milieu.

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PATIENT CHARACTERISTICS AND SETTING

All patients were diagnosed as having major affective disorder by standard criteria (10, 11). The patients lived on a 12-bed research unit organized as a therapeutic community (12, 13) and participated in a variety of behavioral, biological, and pharmacologic studies that involved observer- and self-ratings, a controlled monoamine diet, blood samples, 24-hour urine collections, sleep studies, infusions, measurement of evoked potentials, lumbar punctures, and continuous monitoring of activity. In addition, each patient spent 2–3 hours a week in individual psychotherapy and 6 hours a week in group psychotherapy.

The volunteers participated in a similar regimen, but they did not undergo lumbar punctures, nor were their mood and behavior rated by the nursing staff. Moreover, they did not receive psychotherapy per se but met for 2 hours each week with a psychiatrist (P.G.) and for another hour each week with a social worker (Y.D.) to discuss their experiences on the unit. In addition, one of us (T.W.) had weekly contact with the patients and volunteers that focused on research participation. Like the patients, the volunteers' lives revolved around the unit, where they ate, slept, and spent much of their spare time, although they worked for several hours each weekday at a "career assignment" in a laboratory or other unit of their choice.

The material comprising this report was gathered from individual interviews, nurses' notes, and recordings of group meetings on the unit. The clinical observations were also the subject of regular group discussions among the authors.

EFFECTS OF THE NORMAL VOLUNTEERS ON THE PATIENT COMMUNITY

The response of the patient community to the normal volunteers will be described longitudinally in three separate phases: 1) the initial "activation" response, 2) the phase of depressive reaction, and 3) the partial identification and resolution of the depressive process. The duration of each phase was approximately 4 weeks.

Initial Response

The patients were given 3 weeks' advance notice that normal volunteers would be joining the milieu and

initially showed no signs of being affected by this announcement. However, the content of the next few group therapy sessions suggested that on a certain level the patients recognized that the volunteers would be, in effect, representatives of the "normal" community, raising the issue of the distinction between psychiatric patients and normal, healthy individuals. It was during one of these sessions that a patient who had been anticipating discharge with apparent confidence surprised the group by expressing doubts that he would be accepted as an equal in the normal outside world after having been hospitalized on a psychiatric unit. Other patients recalled similar predischarge fears during prior hospitalizations, fearing the reactions not only of their employers but of their friends and families. None of the patients related his or her current anxiety about returning to the community to the anticipated arrival of the college-age volunteers.

Other indirect evidence that the patients felt anxious about the volunteers surfaced in subsequent meetings. For instance, several patients requested that there be an increase in group and individual therapy hours, which suggested that they feared losing the sustained interest of the staff to the volunteers.

More direct acknowledgment of feelings concerning the volunteers took the form of anger at the administration for discriminating against patients by using the term "normal volunteer." This anger was elaborated in fantasies about scandalizing the young, innocent volunteers with feigned crazy behavior. One patient recalled a previous hospitalization for mania during which she had gotten on her hands and knees, barked, and chased a horrified visitor off the ward. The memory of her identification with an unruly animal provided a glimpse of her impoverished sense of self, derived in part from a past experience of uncontrolled behavior during manic episodes.

The patients seemed to divert many of the feelings accompanying such self-images into a variety of energy-consuming activities, such as making and then breaking alliances with the staff and among themselves or turning from one cause or project to another. In addition to serving a diversionary function, each of these maneuvers also seemed to protect the patients from a specific anxiety or feeling associated with the volunteers' anticipated arrival.

The next several days were characterized by the first of these maneuvers, an effort on the part of the entire patient community to strengthen the alliance with the staff. For example, patients busied themselves with a variety of tasks designed to lighten the burden of the staff, and for the first time in several months they thoroughly cleaned their kitchen and recreation areas. Compliance with research procedures increased, and the patients formed their own committee to monitor compliance with such tasks as the recording of self-ratings and temperatures. No objections were raised to the room changes needed to accommodate the volunteers, despite the fact that room changes had been a controversial issue in the past. Moreover, during group and individual therapy meetings, patients talked earnestly about past losses and disappointments, but with an indifference to the feelings usually associated with these experiences. This apparent alliance with the staff may have served to diminish the patients' anxiety about losing the staff's interest to the volunteers.

During this time (about 1 week before the arrival of the volunteers), the patients also seemed to be considerably more involved with one another. This increased alliance was highlighted by the apparent reconciliation of two patients who had shown intense antipathy for each other for some time. Such solidarity was relatively unusual for the patients and seemed to lack authenticity; it was as if they needed not only to lose themselves in one another's affairs but also to deny the antagonism they usually felt toward the illness in others and themselves.

Shortly after the arrival of the volunteers, the patients began to undermine overtly their carefully nurtured alliance with the staff. There was a marked increase in resistance to group and individual psychotherapy and to the prescribed routines on the unit, including research procedures. Conflict between the staff and the patients increased considerably as the staff became angered and bewildered by the change in the patients' behavior. Although there were no obvious signs that the patients' sense of shame about their illness and their patient status had increased as a result of the presence of volunteers designated "normal," it seems in retrospect that the resistance was an attempt to ward off this sense of shame.

The apparent cohesiveness among the patients also dissolved during this period. In particular, those who were healthier and on the way to recovery insisted that they were going to form their own therapy groups and activities from which the more regressed and less communicative patients would be excluded. The need for these patients to renounce any identification with the more regressed patients constituted a striking change from their earlier solicitous attitudes.

The Phase of Depressive Reaction

Although a number of patients claimed responsibility for their own psychotherapy and activities, none of the plans ever materialized. Just as alliances were first made and then broken, interest in any specific endeavor failed to be sustained. It appeared that these interests were not authentic but were diversions from feelings that had to be avoided.

At this point many patients felt frustrated and a few disparaged themselves for being unable to use their time effectively. Group and individual therapy meetings were generally unproductive, since patients continued to resist examining their feelings. Although the level of overt distress among the more recovered patients did not become particularly intense, most seemed to be in a somewhat suspended state, during which little was ventured or achieved. For the more

depressed patients the overt reaction of withdrawal was more pronounced.

A patient who had been a highly successful attorney before his illness was the first to break out of this quiescent phase by becoming acutely aware of his intense shame over an illness that he felt rendered him less functional and less human than the volunteers. In the prevailing climate of a diminished alliance with staff and an aversion to the exploration of feelings, he persevered in his request for a lengthy pass, despite what he later revealed to be a growing suicidality. Fortunately, he withdrew the request when his therapist noticed that he appeared more agitated. When confronted with these feelings during a group therapy session, he initially dealt with them by directing a great deal of anger at the staff, whom he accused of treating him as impaired. His anger seemed to catalyze other patients in the community, who emerged from their quiescence and began voicing anger at what they saw as a double standard on the unit. The volunteers had their own phone, flexible curfews, and occasional exemption from the diet; they were not subjected to lumbar puncture, their behavior was not rated, and their moods were not regularly subject to interpretation. Moreover, the patients felt that the volunteers generally received more respect from staff and administration. The patients' expression of these feelings provided a context for the elaboration of their deep sense of shame about their illness and other attributes they judged to be imperfect.

Partial Identification and Resolution of the Depressive Response

Once the patients began to become aware of previously repressed feelings, they proceeded to explore the events of the past few months. The major issue that came to light was the patients' acknowledgment that the presence of the volunteers (who by definition and in actuality were more functional than they) placed them in a situation in which they were acutely reminded of their imperfections and weaknesses. One patient stated,

> After they arrived I was faced with people who were called normal and I couldn't get away from that even though I try not to think about the fact that I've had more serious problems than most other people. For a while I began to feel that the volunteers and the staff and even the other patients saw me as a failure—like I was too weak to get through life without getting sick, and I began to feel that way about myself. . . . Somehow I got busy with all the things going on around the unit, and I didn't feel that way for long.

As these feelings emerged it became clear that our patients shared a pervasive sense of imperfection derived in part from their experience of themselves as psychiatric patients who had failed to successfully negotiate the important tasks necessary for establishing a sense of happiness and balance. Their repeated hospitalizations for affective episodes seemed to confirm this thoroughgoing sense of failure. Thus, the complex interaction of biological and psychological factors that rendered them more vulnerable to affective episodes imprinted upon them a sense of defectiveness. Moreover, this sense of defectiveness seemed to initiate a vicious cycle, in which patients viewed many of the inevitable disappointments or losses in living as further evidence of their particular inability to influence adequately the events of their lives. The extreme dysphoria associated with directly experiencing this sense of defectiveness or "negative identity" was intolerable, and elaborate defenses against it became a necessity. It is not surprising that the normal volunteers presented a particularly highly charged stimulus for the emergence of these feelings.

As the patients continued to reconstruct their experiences following the arrival of the volunteers, they recognized the defensive and avoidance function of the various energy-consuming maneuvers. They admitted that they had initially attempted to placate staff and avoid disagreement, despite the fact that this meant remaining silent over issues that hurt or angered them. They also noted their efforts to bypass staff and assume responsibility for running the unit and providing therapy for themselves. When these activities failed to produce the desired result, they turned to other avoidance defenses, particularly withdrawal from others and dampening of feelings. Although most patients were successfully defended by these avoidance maneuvers, they were also shielded from a sense of their own strengths and from the possibilities of gratification from others and therefore experienced an aching emptiness that further contributed to depression. The patient who was unable to defend against his intense shame by such maneuvers spoke movingly of the suicidality he experienced when he felt himself to be without any redeeming features. Other patients discussed similar episodes of experiencing acutely their sense of negative identity, which often (but not always) preceded suicide attempts, numbing depressions, or episodes of mania.

It was during this time that the patients began to develop friendships with the volunteers. The young man who had come so close to suicide stated,

> I began to see the volunteers as human beings who I could reach out to. Especially after we talked about how they had affected us here, I was able to pay more attention to them as people in their own right and I began to see that they had feelings like my own. As I got to like them, they seemed more interested in me as a person and this helped me to feel better. We started sharing things with one another, discussing our anxieties. Take their feelings about the research procedures—that started drawing us closer together, because we had that in common. We got to the point of joking about it. The sharing was the nicest part. A lot of the stuff like the procedures, the staff, sort of came into focus for us, nothing was held back.

As time progressed, the level of communication deepened. The volunteers began exploring the illness

with some of their patient-friends, asking, for example, "How did you get sick? It's so difficult for me to imagine that happening to you." Patients responded positively to such openness. One patient stated,

> I felt able to talk about my past and answer their questions because I could accept the problem in myself for the first time without feeling horrible about myself. Their being here helped. It was like a trial thing, having a "normal" come up to you and ask you what you know others must be wondering about all the time and then be able to answer without having to cover up. And I taught them something. I wanted to show them that even though I was hospitalized with depression I knew something about life and had something to say—and they seemed to accept my illness as a part of me, but just a part, and to see that there is more to me than that. I think they helped me to see this in myself as much as anyone else here.

Interestingly, it was not until the patients acknowledged that their experience with the volunteers had ultimately proven helpful that the staff was fully able to accept the presence of the volunteers on the unit. The staff was initially dismayed at the disruption in the milieu and saw this as a failure of the research personnel to protect the therapeutic community rather than as a manifestation of an important part of the patients' depressive process. Gradually, a number of close relationships developed between the staff and the volunteers, and many staff members came to express their gratitude to the volunteers for their warmth and openness in relating to the patients.

Although many barriers between patients and volunteers fell, some patients, particularly those with the longest history of illness, continued to feel the distinction between normal and abnormal. This sense of distinction was illustrated by an evening group meeting held shortly before one of the volunteers left the unit. The volunteer was spontaneously invited to attend as she passed the meeting room. Her description of the meeting is as follows:

> I wore a pantsuit with my robe over it and was walking by the group; perhaps it looked strange and one of them thought they had better invite me to the group before I did something drastic. When I mentioned the exact time of my leaving they said, "No, you can't go, you must stay the usual four months, because all your symptoms are right for manic-depressive illness." One said that I must also be ill and that they too had denied it, and that anyway my overdressing was characteristic.

This complex communication expressed the patients' grief and anger at the volunteer's departure, as well as residual anger at her designation as normal and apart from them. The underlying fantasy, in which the volunteer becomes a patient, in effect expresses the wish for closeness. For some patients, however, this closeness cannot exist in the realm where all acknowledge their mutual humanity but only where all are seen as patients apart from the normal community. Thus, for these patients, the volunteers were no longer the "other" but were recruited as "patients."

EFFECT ON THE NORMAL VOLUNTEERS

Although a detailed description of the reaction of the volunteers to living on our research unit is beyond the scope of this paper, a few observations can be made. Initially, the volunteers found the unit hectic and confusing. One of the volunteers had just left her boyfriend halfway across the country and felt like "crying all the time for the first few days," but she was afraid to cry because "if the staff saw me crying, they'd think I was crazy and have me in therapy before I knew it." Another volunteer described her initial discomfort around the patients:

> I had a stereotyped idea of a psychiatric ward and of patients. I felt that patients would be violent and unpredictable and would talk incomprehensibly. I actually saw one patient go into seclusion on my second day and heard certain staff members talking about having been kicked or bitten by him some months ago.

The volunteers began to question staff members about the rationale for certain interventions and limits. Initially, some thought that the staff tended to be too strict with the patients. Shortly after one volunteer voiced this opinion, a hypomanic young female patient entered the volunteer's room and insisted that she terminate a phone call to her boyfriend and speak with her. She did exactly as the patient requested. After this experience she talked with a staff member and reached the conclusion that "being direct with people about what you like and don't like is a way of showing them respect. You don't have to baby or humor anyone here, you react as you would to anyone else."

As the volunteers became more interested in the patients' behavior, they began to investigate their own feelings. For example, after a month, one volunteer noted that "the more I think about it, the more I feel that the depression the patients get is like the depression I get, only theirs seems so much more severe."

Gradually, as they grew more comfortable with the patient community, the volunteers became more open and were able to acknowledge some of their problems and occasionally asked for assistance in solving them.

Although in general the volunteers' perceptions of mentally ill people changed profoundly and they saw their common dilemmas and humanity much more deeply than before, they retained a sense that there were some important differences between the patients and themselves. In fact, the volunteers seemed to have learned about some of the crucial clinical aspects of manic-depressive illness, such as the liability to sudden affective shifts, the tendency to avoid painful emotional issues, the often unyielding perfectionism, the tendency toward self-destructiveness, and the often painfully low self-esteem. This was summed up by one of the volunteers just before she left:

I try to compare myself with patients sometimes, what makes me different. I was sad when I came here, definitely, but for some reason I could cope with it and keep my senses clear. It seemed it was easier for me to face what was bothering me than for some of the patients. Many of the patients seem to go one way or the other when they face something very painful. Maybe there is a physical factor that accounts for some of that. I think too that the patients expect too much of themselves and if they fail, it is more than they can bear. It's like their expectations are more important than they are, like they don't have a good enough feeling about themselves to begin with. Also, I have a natural instinct to stay afloat. I feel some of the patients work against themselves. For instance, if I were a lifeguard and I were involved in a rescue mission, I'd really try to save the life of the other person. But, if I recognized that I couldn't save the person, and that if I persisted I'd die, I would recognize the situation for what it was and attempt to save my own life. In other words, don't give up without a fight but don't lose yourself in a losing battle. People here lose themselves. They can't accept things, but they are not supermen, and sometimes they can't recognize they need help. I was that way a lot in my life, but it seems I could change it when I needed to.

CONCLUSIONS

The need for normative data in clinical psychobiological research set the stage for a social and psychological "experiment," in which psychiatric patients and normal volunteers were hospitalized together for several months on a psychiatric research unit. The plans we fashioned to deal with the possible problems that might arise from the experiment centered around the assumption that the volunteers would have considerably more difficulty exchanging their own familiar environment for an active psychiatric unit than the patients would have adapting to a unit that would be enlarged by a few volunteers. This assumption proved to be incorrect; we underestimated both the resiliency of the volunteers and the extent to which patients consider themselves to be inferior to those whose lives have not been disrupted by a chronic psychiatric illness.

This impoverished self-concept of our patients largely determined their complex reaction to the volunteers. The associated shame initially required a number of energy-consuming avoidance defenses. Although these diversionary maneuvers worked temporarily, it became clear that they would not solve the underlying issues, and several patients began to disparage themselves and withdrew into a numbing but insulating depression. Thus, the patients' response to the volunteers described a vicious cycle, in which their sense of failure necessitated the institution of complex defensive maneuvers, which further exacerbated their vulnerability to depression.

The patients' sense of failure, which comes in large part from their experience of themselves as chronic psychiatric patients, is particularly crucial in the predischarge period, when they face the difficult issues of separation from the hospital and reintegration into their families and communities. By admitting volunteers, we in effect brought the community to the hospital, which gave the patients an opportunity to deal with the difficulties involved in reintegration without having to deal with the complex issue of separation at the same time. Since the problems raised by reintegration occurred entirely within a therapeutic setting, they could be dealt with more reliably than in the postdischarge period. Moreover, the openness and the warmth of the volunteers facilitated the therapeutic work of the staff in leading to the considerable resolution of these highly charged issues.

The extent to which repeated psychiatric hospitalizations and recurrent affective episodes influence the self-concept of patients with affective illness underlines the importance of a program that combines psychopharmacologic and psychotherapeutic intervention in an attempt to prevent relapse of the illness. When a patient does relapse, careful attention should be paid in the predischarge period to the possibly damaging effect of his self-disparagement, since such attitudes substantially compromise the chances of a successful reintegration into his environment and enhance the likelihood of further affective episodes. The admission of normal volunteers to the unit can facilitate a more effective response to these problems.

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Whatever Happened to Psychiatry? The Deprofessionalization of Community Mental Health Centers

BY PAUL J. FINK, M.D., AND STEPHEN P. WEINSTEIN, PH.D.

The authors describe the dichotomy in the concept of community mental health centers. One view regards the community mental health center as a service provider for underserved populations, and another holds that it is an agent of social change. Deprofessionalization of community mental health centers has resulted from this lack of clear purpose, curtailment of funds, and conflicts over authority, service delivery, and control of the centers. Deprofessionalization has led to a decline in the number of psychiatrists in community mental health centers and a potentially negative impact on the quality of patient care.

RECENTLY A NUMBER of papers[1] (1-5) have been published in defense of or in opposition to the community mental health center movement. A series of positive papers on mental health manpower, particularly psychiatric manpower, has noted the failure of community mental health to attract and, more importantly, to retain psychiatrists. It appears that as a result (or perhaps cause) many centers have chosen to be nonprofessional or at least to operate with few professionals on their staff. This phenomenon is extolled by some and is of great concern to others. Another disturbing phenomenon is the lack of understanding of the concept of community mental health and the mission of community mental health centers. There is no uniformity in the understanding of the concept, and there is sufficient misunderstanding to result in a great deal of confusion, not only among those working in community mental health centers but also among the observers, funding agencies, legislators, academicians, and, of course, the consumers.

This article addresses the issues surrounding the ambiguity of the community mental health concept and the roles of professionals and nonprofessionals within a community mental health framework.

TWO VIEWS OF CMHCS

There are several dichotomies that express the problems and areas of conflict in the community mental health movement. Is it merely a new type of delivery system or an entirely new concept? Is mental health a euphemism for mental illness or is it an attempt to satisfy community needs, resulting in happiness for all citizens? Are community mental health centers social welfare agencies or health (medical model) agencies? Were community mental health centers created to care for the seriously mentally ill or to solve the community's problems? Can all persons designated as therapists deliver the same quality of care regardless of education, training, background, or experience or are such credentials necessary in service delivery roles?

In response to the Community Mental Health Centers Act of 1963 and its amendments, two distinct schools of thought developed. One group believed the act was designed to 1) provide access to psychiatric care for the millions of citizens who could not afford private care; 2) provide care for the seriously mentally ill closer to home in their own communities thus obviating the need to send them off to a remote state hospital; and 3) develop organized care settings throughout the nation, thereby developing a team approach in the delivery of care to mentally ill and emotionally disturbed persons.

Another group apparently saw the community mental health center as an entirely different type of agency, one that would address the problems of mental illness by ridding the community of what were presumed to be the causes of mental illness: poverty racism, unemployment, poor housing, crime, riots, etc.

Langsley, an early leader in the community mental health movement, recently expressed his views regarding this dichotomy:

> The attempt to meet all needs of all people—the attempt to make community mental health programs responsible for the quality of life rather than for the treatment and pre-

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[1] "Psychiatric and Mental Health Manpower," by Donald G. Langsley, M.D., prepared for the President's Commission on Mental Health; and "Toward a National Policy for Psychiatric Manpower Problems, Assets, and Recommendations," prepared by the executive committees of the American Association of Chairmen of Departments of Psychiatry and the American Association of Directors of Psychiatric Residency Training for the President's Commission on Mental Health, 1977.

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> vention of illness, is a distortion of the original program, its legislation and its intent, at least in my opinion. (personal communication, January 1978)

The concern for the community's social needs versus the needs of mental patients, of prevention versus treatment, of social services versus psychiatric services, has become the universal area of conflict surrounding the community mental health movement. Public Law 94-63 and other public documents support the concept of community control of community mental health centers, thereby moving community boards away from advisory roles toward governing roles. This is destined to reshape the definition of community mental health as a narrow focus of treatment into a broader concept defined by the community. This situation is most effectively explicated by Zwerling (6) in a variety of definitions that are the result of a special effort to redefine community mental health and to distinguish it from traditional mental health practice. According to Zwerling the fundamental components of community mental health are that

> first, the target of effort is the entire population of a defined community, rather than persons identified as "patients"; second, it seeks to achieve the maintenance and promotion of health rather than to devote exclusive concern to the treatment of illness; third, it seeks an understanding of the family, community, social class and cultural sources of human disturbance in addition to its biological and psychological sources and in turn the promotion of mental health by intervention in family, community, social class and cultural structures rather than only in biological and psychic structures. (6, p. 25)

The legislation establishing community mental health centers mandated five basic services: inpatient, outpatient, emergency, partial hospitalization, and consultation-education. The first four were designed to provide direct treatment to sick people. Consultation-education was viewed by many as a highly experimental "service"; it was frequently interpreted as the preventive area of the center and it became a focus of community groups and social changers. In understanding community mental health as a force for social change many communities maintained a plea for increases in this "preventive service." However, as Langsley pointed out there is no hard evidence for the ability of the field to prevent mental illness and the scientific base for such prevention is at best rudimentary. The demands for preventive services are often more a matter of rhetoric than substance, but, more importantly, these demands reflect a basic misunderstanding or misreading of the purpose of the community mental health effort. In fact, many consultation and education services do address themselves to aspects of preventive psychiatry in the context of a more narrowly defined mission than that described by those who see community mental health centers as social change agents rather than service delivery agencies.

If the fulfillment of the social change definitions were possible, it would take far more funds than have been allocated to the entire community mental health center movement. The available funds can barely pay for direct services, and if the current center funding is not specifically for direct patient care services then there is no existing federal structure to fund such services and what began as an academic difference of opinion around definitions escalates into a crisis in the treatment of mental illness. The direction taken by NIMH in its Forward Plan (7) was to move toward having all psychiatric services in the nation performed in community mental health centers. However, the confusion between the role of the mental health center and the delivery of mental health services places psychiatry in a tenuous if not treacherous position, particularly if the nontreatment or social change definition of mental health persists.

REDUCED PROFESSIONAL INVOLVEMENT

In addition to this disconcerting situation, a concurrent trend has been evolving within the community mental health center system—deprofessionalization. This involves a gradual movement away from the use of professionals in both the administration of centers and direct treatment of patients. This emerging climate appears to be a repetition of the situation that developed in the state hospital system earlier in this century, when the quality of patient care deteriorated, professional involvement was reduced or eliminated, and the hospitals became repositories for the poor. Issues of turf, funds, and the objectives of the community mental health center movement provide the causal framework for the current efforts at deprofessionalization, particularly in respect to psychiatry.

Between 1970 and 1975 the number of full-time-equivalent psychiatrists in community mental health centers dropped from 6.8 to 4.3 per center (8, p. 25, table 2; 9) even though the total staff of these centers almost tripled during the same period. The number of psychologists nearly doubled (from 4.9 to 8.5) and social workers increased from 9.7 to 12.2 full-time equivalents per center during that time. Staffs have been supplemented by paraprofessionals and indigenous workers.

Threats to Psychiatrists' Involvement

From the beginning of the community mental health center movement there was an effort to use community residents (indigenous workers) to deliver some services because of their familiarity with their own ethnic group in the community. This role became distorted to the point where such workers felt and were supported in the belief that their therapeutic interventions were at least equivalent to those delivered by trained professionals. Appropriate life experience and a six-week inservice training program somehow was

interpreted as the equivalent of a medical school education and three years of residency. Once one denies that a psychiatrist, psychologist, social worker, or psychiatric nurse has any unique training, skill, or theoretical framework, the leap to an antiprofessional point of view is simple. In fact, if the goal of community mental health is to make the community a better place to live, then professionals may have little to offer in their capacities as therapists.

Even within the more traditional concept of the community mental health center, continuing conflicts among the various professionals for authority and control have weakened the position of all professionals. This has resulted in undue pressure on the system to eliminate, where possible, psychiatrists from all controlling positions despite their skills at organizing and developing clinical services and despite their training (or, for that matter, lack of training) in administration.

Being the highest paid professional within a community mental health center has been a triple-edged sword. First, psychiatrists' services are expensive, thereby restricting the number hired. In their place large numbers of personnel with less or different training whose concept of community mental health and approaches to patients were very different were hired. Second, psychiatrists assumed that centers were medical agencies and that doctors therefore were the highest authorities. Finally, psychiatrists frequently were needed only to prescribe medications where large numbers of chronically mentally ill patients were treated, thereby limiting their program involvement. In a setting in which community mental health was seen as an agent of social change, psychiatrists maintained a psychodynamic orientation and a general belief that patients in some way contributed to their own distress, so that they were driven out of the center, embroiled in a continual battle with other members of the staff and community, or co-opted, agreeing that they had very little different to offer. In general, community mental health centers became relatively unsatisfactory places for psychiatrists to work.

The situation is further exacerbated by available funding mechanisms, which make it less advantageous for centers to adhere to the medical model and retain a significant number of psychiatrists on the staff. As a center's federal funds are reduced, the administration must turn to state and local resources to support the level of effort they had developed. However, these sources lack the broad federal tax base (i.e., funds) and are forced to tell centers to reduce staff or services. When this is combined with an antiprofessional community bias, the most straightforward conclusion is to save money by eliminating the higher priced personnel and substituting more cheaply acquired mental health workers. This view is frequently tacitly supported, if not fully endorsed, by government officials who for the most part are not themselves psychiatrists and frequently hold the view that professionals are not necessary for the delivery of mental health services.

The Fallacy of the Social Change Definition

The guarantee of good health for all is a marvelous concept but should be understood to be a practical impossibility. The best we can hope to guarantee is the availability of health *care*. This distortion is by no means minor and in fact tends to support the trend toward deprofessionalization. It is easy to see why some say that the medical model of service delivery should be supplanted or replaced by a social action model or for some to call for a redefining of mental illness because of an increasing belief among both professionals and the public that many individuals considered mentally ill by mental health professionals are in fact persons who are appropriately responding to stresses in their environment. If health is guaranteed and manipulation of the environment can provide health, then psychiatrists and psychologists, who are trained to understand and help people change their behavior, have no legitimate place in such a system.

Despite being a part of the rhetoric, the issue of quality of care is rarely studied or addressed directly. It becomes clear from this vicious circle that the quality of care will deteriorate, the individual patient will lose, and the nature of centers will change. What was hailed as an opportunity for all citizens to have equal access to quality care is degenerating into another two-class system in which the poor will once again receive inferior care from less qualified workers who have been seen through rationalization to provide equal quality services.

Deprofessionalization can be seen as resulting from a confluence of mistaken ideas, not the least of which is the strange confusion between illness and health and the even stranger confusion between health and health care. Zwerling cites "the recognition of health, including mental health, as a right of all, and not as a privilege available for purchase by those who can afford the services" (6, p. 81) as one of the essential components of community mental health.

As prestigious a figure in American medicine as John Knowles in a recent article directed our attention to the same seemingly innocent error. It is not clear if he himself believes it or if he is pointing out the unreality of it:

> The evolution of the various professional, governmental and consumer groups interested in, and responsible for, health—now viewed by most American citizens as a fundamental "right"—represents a typical American response. (10, p. 5)

CONCLUSIONS

For a number of years psychiatrists have attempted to define their role (11–13). Specifically, these definitional efforts have tended to place great emphasis on the care of the seriously mentally ill and the biological aspects of mental illness. Where the role of psychiatrists and other professionals has become equivocal

has been in those areas where others have professed equal facility in the treatment of specific conditions and in a system that emphasizes social change over patient care.

Whatever happened to psychiatry? is not an idle question. If all psychiatric funding is relabeled mental health funding and if psychiatry is then found to be irrelevant to community mental health, which is viewed as an agent of social change, then psychiatrists can look forward to a severely diminished role in the entire mental health/mental illness system. At the risk of professing professionalism and elitism, we suggest that the delivery of services to people with problems, whether marital and family problems or schizophrenia, should be monitored if not delivered by professionals. To achieve this objective it is imperative that clarity be brought to the definition, goals, and purposes of community mental health centers and that in lieu of an idealistic, clearly unachievable goal of bringing "mental health" and the "good life" to every community in America we narrow our focus to an achievable goal of providing access to and care for millions of mentally ill and emotionally disturbed citizens. In such a system, professionals would be responsible for the services delivered and would receive the regard consistent with that role. Similarly, community workers, paraprofessionals, and citizen volunteers would have appropriate positions, roles, and tasks within a system organized according to the medical model. It is possible to develop a system of services that is both needed and desired by the community and that affords patients dignity in addition to excellent care. There is no disagreement with Zwerling's statement that

> the thing that we're about is dignity. It's important that a person sees himself as having some worth, and I contend that a service should be developed in a way that a person can work better and serve in dignity, as well as have service in dignity. (6, p. 123)

If it is no longer possible to make the community mental health center system an outstanding therapeutic service delivery system we must immediately redefine it so that it will be clearly distinguishable from psychiatric treatment and rehabilitation and each system can proceed to greater definition, autonomy, growth, and achievement.

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Plasma Growth Hormone: Effect of Anxiety During Flooding in Vivo

BY GEORGE C. CURTIS, M.D., RANDOLPH NESSE, M.D., MARTIN BUXTON, M.D., AND DAVID LIPPMAN, M.D.

The treatment of phobias by rapid in vivo exposure to the feared situation was accompanied by an increase in plasma growth hormone levels on at least one of two occasions in 8 of 11 subjects. The average growth hormone response was greater on the second occasion, even though subjective anxiety was less. Plasma growth hormone was not elevated during the subjects' adaptation to the laboratory. Strong subjective and behavioral anxiety responses failed to elevate plasma growth hormone levels in some subjects. The probability of a growth hormone response was the same whether or not baseline levels were elevated prior to exposure.

PLASMA GROWTH hormone levels are elevated in man and animals by a variety of physiological and psychological stimuli (1). Little is known about the specific psychological events triggering growth hormone release in man. There have been several reports of growth hormone elevations in situations that are presumably emotionally arousing, such as venipuncture (2, 3), cardiac catheterization (4), gastroscopy (5), arousing movies (6), final examinations (7), and stress interviews (8). However, apparently only a minority of subjects studied in these situations prove to be growth hormone responders. With the possible exception of Greene and associates' study of patients undergoing cardiac catheterization (4), most of the work involving humans has been conducted in situations that are only mildly arousing. Affective arousal independently assessed by psychological methods has been reported by some researchers (8) to distinguish growth hormone responders from nonresponders; other researchers (3, 6) have reported that it does not. One report (5) suggested that anticipation of an arousing experience may have a greater effect on growth hormone than does the event itself, a finding reminiscent of much work in the corticosteroid field (9).

The treatment of severe specific phobias by flooding in vivo produces anxiety responses that are usually quite powerful as judged by behavioral and subjective criteria (10), and to some extent by autonomic (11) and corticosteroid responsiveness (12, 13). We now report the results of 2 experiments using this approach for a further assessment of the relationship between anxiety and growth hormone secretion.

METHOD

The subjects consisted of 11 medically healthy, medication-free persons (9 women aged 20–52 and 2 men aged 25 and 42) with severe phobias for physical objects (e.g., snakes, spiders, birds) manifested by panic on encountering the phobic stimulus and by a fixed pattern of elaborate, irrational, life-restricting maneuvers to avoid encountering it, often including avoidance of photographs or motion pictures of the object. One 52-year-old woman had stopped menstruating 2 years previously but denied hot flashes or other menopausal symptoms. The remaining women, all in their 20s and early 30s, were menstruating normally.

The design of the study was disclosed to each subject in advance, and informed consent was obtained. Each subject spent 5 three-hour sessions in the laboratory on separate days. At 20-minute intervals throughout each session, blood samples were taken through an indwelling needle, and the Subjective Units of Distress were self-rated on an analogue scale of 0 (no distress) to 100 (maximal distress).

With exceptions to be noted, the subjects sat quietly throughout every session, engaging in self-selected sedentary activities, such as reading or working crossword puzzles. The first 2 sessions were for adaptation. During the second hour of the third and fourth sessions, the phobia was treated by flooding in vivo. This consisted of live confrontation with the phobic stimulus at the maximum rate that the subject would allow, keeping anxiety at the maximum tolerable level until it

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subsided and the patient became comfortable in the presence of the stimulus. The fifth session was a control. During the middle hour of the adaptation and control sessions, the subjects were engaged in casual conversation in order to control for the presence of another person during the hours corresponding to the flooding therapy. If additional treatment was needed and desired, it was given at a later date without further blood sampling.

In order to determine the beginning time of the laboratory sessions, each subject recorded his or her time of rising and retiring for 14 days. From these records each subject's average time of midsleep was computed and used as the zero reference for his or her circadian phase. In one experiment with 6 of the subjects, sessions in the laboratory began 3 hours after the individual's average time of midsleep, usually around 7:00 a.m. In the other experiment with 5 subjects, sessions began 15 hours after the individual's average time of midsleep, usually around 7:00 p.m.

Growth hormone was measured by radioimmunoassay (14). Plasma cortisol findings in these subjects have been reported elsewhere (12, 13).

RESULTS

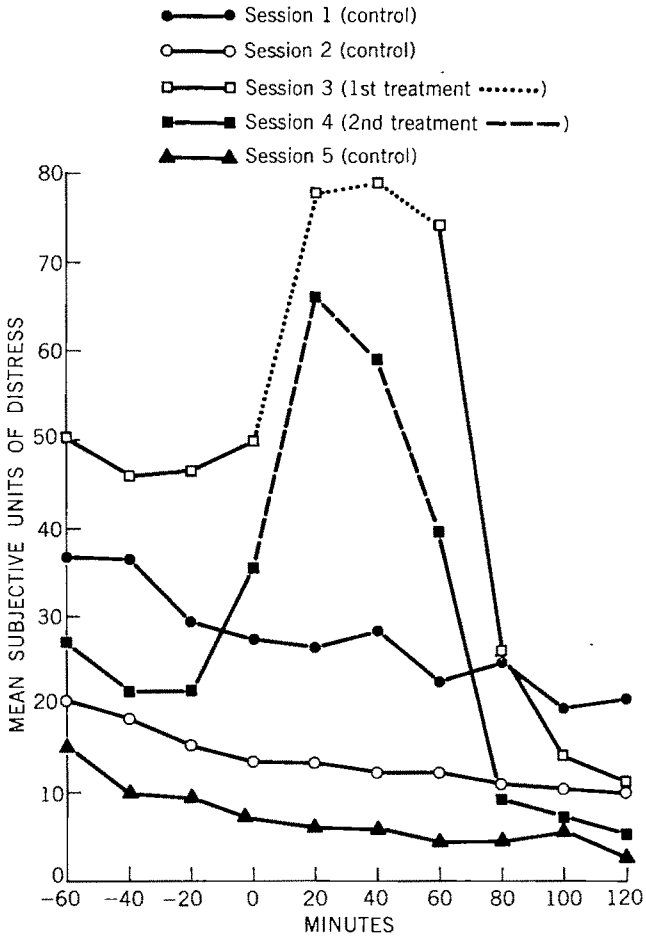
Although not entirely uniform across subjects or across time, the affective response to flooding in vivo was regularly intense, often including weeping, screaming, attempts to get away, gross tremors, chattering teeth, gooseflesh, and rapid, labile heartbeat. As these manifestations subsided at one level of exposure, the therapist pushed rapidly ahead to more intense exposure. Overall, it was a grueling experience, usually described as the most frightening of the patient's life.

Data analyses of growth hormone levels and self-ratings of anxiety from the two experiments yielded similar results and conclusions. Therefore the two data sets were pooled and will be reported as a single experiment.

Figure 1 presents ratings of Subjective Units of Distress averaged across all 11 subjects at each time point within each session. The levels were usually low, except for moderate elevations during the initial adaptation session and prior to the treatment hours and for marked elevations during the two treatment hours in sessions 3 and 4. Subjects usually attributed their uneasiness during session 1 to the experiment as a whole or to blood drawing. A few mentioned fear of the possibility of being confronted with the phobic stimulus immediately, although they had been assured that this would not happen until session 3. During the anticipatory uneasiness in session 1 the subjects did not appear intensely disturbed, as they did before and during the confrontation itself. Uneasiness prior to treatment hours was regularly attributed to anticipation of treatment.

Three-way analyses of variance (Sessions×Subjects×Time-Within-Sessions) of the data on Subjective Units of Distress yielded Sessions×Time-Within-Sessions interactions significant at the .001 level, reflecting mainly the large rise during flooding. Subject×Session interactions were also significant ($p<.001$), reflecting mainly individual differences in self-reported distress at times other than flooding. Main effects of Subjects, Sessions, and Time-Within-Sessions were also significant at levels ranging from .05 to .001.

FIGURE 1
Mean Subjective Distress Ratings for All 11 Subjects[a]

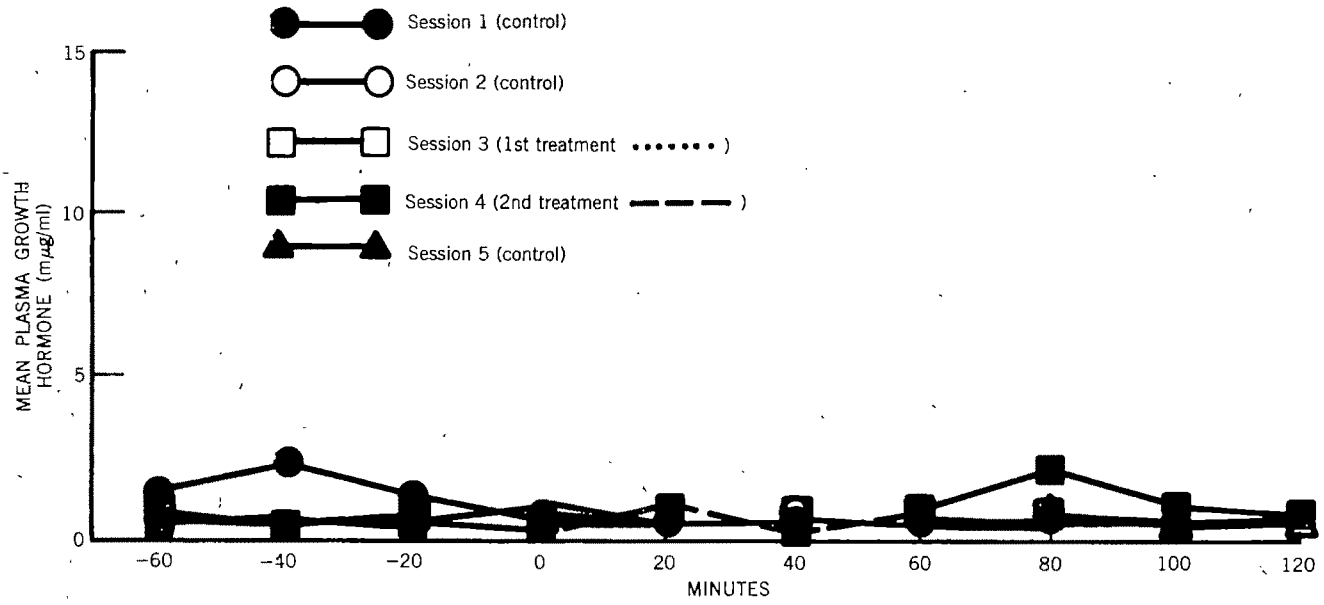


[a]Flooding from 0–60 minutes in sessions 3 and 4.

Growth hormone levels were quite labile in some subjects and stable in others. Some of the lability coincided with flooding, but a great deal did not. Some subjects appeared to make strong growth hormone responses to flooding while others clearly made no response at all.

When growth hormone data for each time point were averaged across subjects, suggestive rises during flooding occurred in both groups of subjects, but they were not confirmed by significant main effects of Sessions or by Sessions×Time-Within-Sessions interactions in either group. The main effects of Subjects and the Subjects×Sessions interactions were significant ($p<.005$ or better).

FIGURE 2
Mean Plasma Growth Hormone Values in a 25-Year-Old Male Nonresponder[a]



[a]Flooding from 0–60 minutes in sessions 3 and 4.

A separate analysis of the data from each subject in order to examine the interactions revealed significant main effects of Sessions in 5 of the 11 subjects. In every case the highest mean growth hormone level occurred during one of the sessions containing a treatment hour. Contrary to the cortisol results (12, 13), there was not even a suggestive tendency toward growth hormone elevation in session 1. Averaging the values across sessions diluted any effect of flooding, since each treatment hour was preceded and followed by a nontreatment hour.

Three of the 11 subjects showed significant main effects of Time-Within-Sessions. In one, this was due to elevations during the first hour in most sessions, in one to elevations during the third hour of most sessions, and in one to elevations during the first and third hours.

Thus, 4 main types of individual response patterns were noted: 1) near-zero values at all times, as in figure 2; 2) many elevations, mainly in the first and/or third hours of most sessions, and apparently unrelated to flooding; 3) a relatively specific response to flooding, greater during the first treatment hour (session 3); and 4) a relatively specific response to flooding, greater during the second treatment hour (session 4). Pattern 4 was observed most frequently. The foregoing results suggest a specific effect of flooding, perhaps obscured by individual differences and by unexplained baseline lability, primarily during the hours preceding and following those which corresponded with flooding. Consequently, a three-way analysis of variance was performed using data from all subjects and all sessions, but using only the 2 time points immediately before and the 3 time points during the flooding hours, together with matching time points from adaptation and control sessions (−20 minutes through +60 minutes in figures 1 and 2).

The results of this analysis are summarized in table 1. They are similar to the results from analysis of all data points from the two experiments separately, except that the main effect of Sessions is now significant at the .01 level. Figure 3 depicts the values, averaged across subjects for each time point within each session. This illustration shows mean elevations attributable to flooding and suggests a possible enhancement of response at the second treatment, with perhaps an anticipatory rise before the second, but not the first, treatment. However, the interactions still point to great individual differences in terms of responders and nonresponders.

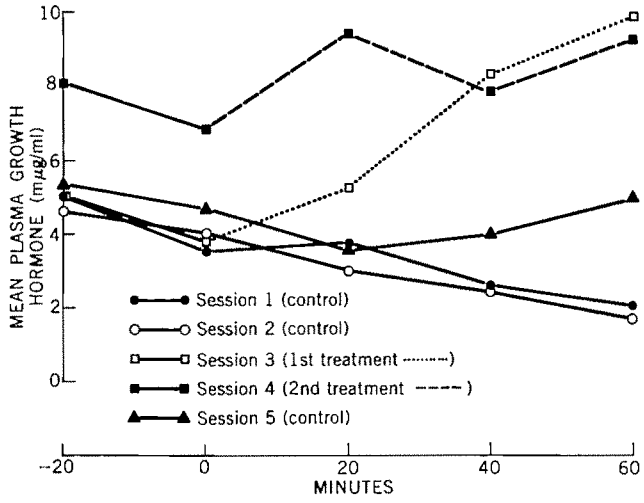
Considerable evidence (15–17) suggests that growth hormone responses to stimuli may be inhibited if baseline values are elevated just prior to the stimulus. Since values were labile in some of the present subjects, could elevated baseline values account for some of the nonresponses to flooding? This does not seem to be the case.

If baseline elevation is defined as 5 mμg/ml or more, and response as a rise of 5 mμg/ml or more above baseline, 8 of the 11 subjects responded in one of the treatment hours and of these, 7 subjects responded in both. If the baseline was not elevated, responses occurred in 64% of the treatment hours and in 12% of the matching control hours. If the baseline was elevated, the corresponding percentages were 63% and 0%. The mixed between-and-within-subject design precludes performing most nonparametric statistical analyses of this finding.

TABLE 1
Analysis of Variance for Plasma Growth Hormone Levels

Item	Mean Square	df	F	p
Subjects	259.40	10	4.0	<.001
Sessions	263.10	4	4.05	<.01
Time-Within-Sessions	10.31	4	<1	n.s.
Subjects × Sessions	65.00	40	2.96	<.025
Subjects × Time-Within-Sessions	33.59	40	1.53	<.05
Sessions × Time-Within-Sessions	27.22	16	1.24	n.s.
Subjects × Sessions × Time-Within-Sessions	21.84	160		

FIGURE 3
Mean Plasma Growth Hormone Values for All 11 Subjects[a]



[a]Flooding from 0–60 minutes in sessions 3 and 4.

DISCUSSION

Many factors have been proposed to explain the extreme lability of plasma growth hormone levels (18), but even in studies in which such factors as falling blood glucose levels (19, 20), exercise (21, 22), and venipuncture (23) have been controlled, there are still frequent unexplainable episodes of growth hormone secretion (21). These must be taken into account in psychophysiological studies. Women are usually found to show greater lability than men (19). This may account for the somewhat greater baseline lability in the present study than in several others in which men comprised a higher proportion of the subjects (3, 4, 6, 8).

Other factors are difficult to evaluate. Brown and Heninger's subjects (3, 6) were studied after an 8-hour fast, whereas those of Greene and associates (4) were given a light breakfast, a glucose infusion, 75 mg of sodium pentobarbital, and 25 mg of promethazine. Our patients were drug free, but there were no dietary restrictions; no such restrictions were described by Abplanalp and associates (8) or by Kurokawa and associates (5), although some of the subjects of the latter study underwent gastroscopy and were presumably fasting. Venipuncture or intravenous catheterization is reported to provoke growth hormone release in one-third or fewer of subjects and to do so on repetition in most of those who respond the first time (2, 3). This could account for the consistent and statistically significant elevations early in most sessions in 2 of our female subjects. Replacement of a nonfunctioning needle or catheter was too infrequent to evaluate its effect, although we occasionally observed both rises and nonrises following this type of event. Rises during the third hour in most sessions by 2 subjects may have been due to a postprandial fall in blood glucose (24).

The percentage of subjects with growth hormone responses to the psychological stimulus was somewhat higher in the present study—55% on both occasions, 73% on one occasion or the other—than the approximately 33% reported by others (2–4, 6, 8). This may be because the anxiety stimulus used in the present study was quite potent. Present findings support previous ones (3), in that responders on one occasion tended to remain responders on the next, and nonresponders tended to remain nonresponders. An elevated baseline level does not appear to affect the growth hormone response to flooding, since the probability of a response was the same (63%–64%) whether or not the baseline was elevated. Growth hormone responses were certainly not diminished, and may have been enhanced, on the second exposure to the same anxiety stimulus, even though the subjective and behavioral responses were diminished. If confirmed, this would define a difference from the behavior of cortisol, which tends to respond early, even before the encounter with the phobic stimulus, and to damp out rapidly (12, 13). This could be a further example of what behavior therapists call desynchrony of fear (25, 26). One interesting although tentative interpretation of this is that growth hormone responses may have been classically conditioned while cortisol and subjective responses were being extinguished. In further contrast to the case with cortisol (12, 13), these results give no reason to suspect differential responsiveness at different circadian phases.

Even intense anxiety may fail to stimulate any detectable growth hormone secretion. In the past it has often been tempting to use such a finding to challenge the reality of the anxiety. In the present instance, we can find no basis for such an inference.

Although there were only 2 men in the present study, both had low baselines with little variability, and both were nonresponders on both occasions of flooding. This is consistent with the frequent finding that women are more responsive than men. Three of the 9 women also failed to respond to one or both flooding hours, but of these, 2 had numerous peaks at other times. The responses of the 1 postmenopausal woman were not particularly remarkable; her values were somewhat less labile than those of some of the

younger women, but she did show a baseline elevation and a response to the first flooding session.

As explanations of growth hormone response and nonresponse, variations in manifest affect or in baseline growth hormone values become less attractive, thus enhancing the relative appeal of psychological factors other than affect. For example, nonresponders have been reported to score higher than responders on measures of social engagement (4, 5), field dependence (3), and Type A behavior (27) and lower on the Nowlis factor for egotism (3). These variables seem to warrant further investigation.

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court. However, our own experience as well as that of other investigators (17) suggests that although personal histories can be valuable clinically, they are notoriously unreliable regarding numbers of illnesses and dates of medical events, information essential to this study.

Data from other public and private medical facilities were unavailable because of issues of confidentiality; however, as far as we could determine, our two populations had equal access to other community resources, which makes it unlikely that one group made greater use of such clinics than the other. Furthermore, serious accidents and illnesses would have most likely been referred to the hospital. The choice of private medical care was relatively rare in both groups because of financial considerations. At the time of the study, the hospital selected tended to be used by the less affluent in the community as a source of primary care. Finally, case loss would create underestimates of the actual prevalence of disorders and use of medical facilities in both groups.

We assessed medical records in terms of numbers of visits to the hospital, timing of visits, use made of different hospital services (e.g., emergency room, clinics, and wards), and reasons for visits (e.g., accidents, head or face trauma, respiratory illnesses, child abuse, and psychiatric problems).

RESULTS

The numbers of hospital visits were similar in the two groups. Incarcerated delinquents averaged 15.818 visits, compared with 13.131 visits for nonincarcerated delinquents. There was a slight tendency for incarcerated delinquents to have made more emergency room visits than nonincarcerated delinquents; incarcerated children averaged 8.974 emergency room visits, compared with 5.714 emergency room visits for nonincarcerated delinquents ($t=1.949$, $p=.054$). The numbers of clinic visits and ward admissions were almost identical in the two groups.

The greatest differences between the medical histories of the two groups emerged from an analysis of the reasons for hospital visits and from a study of the symptomatology noted in hospital charts. Although similar percentages of children in each group had been treated at one time or another for accidents or injuries (72.7% of incarcerated delinquents versus 71.1% of nonincarcerated delinquents), incarcerated children were significantly more likely to have sustained a head or face injury (62.3% versus 44.6%, $\chi^2=4.371$, $p=.037$, Yates's correction applied). A measure of the severity of head injuries sustained by the two groups was reflected in the difference between the proportion of children who received skull X rays. In the incarcerated group, 33.8% of the children had received a skull X ray, compared with 13.1% of the nonincarcerated group ($\chi^2=8.566$, $p=.004$, Yates's correction applied). Numbers of visits for head and face injuries were also significantly greater in the incarcerated group (1.026 for incarcerated versus .607 for nonincarcerated children, $t=2.072$, $p=.04$). Of special note was the finding of a significant difference in numbers of head injuries before age 2 (.091 versus .012, $t=2.329$, $p=.022$).

Also, child abuse was noted more frequently in the medical charts of incarcerated children (10.4%) than in the charts of nonincarcerated children (3.6%). It was our impression from previous clinical and epidemiological studies that abuse was underrecognized in both groups.

Of particular note was the finding that perinatal difficulties were significantly more prevalent in the histories of incarcerated delinquents than in the records of nonincarcerated delinquents. Information about pregnancy, delivery, and the immediate postnatal period was available in the charts of 41 incarcerated delinquents and 50 nonincarcerated delinquents. Of the incarcerated delinquents for whom any information was available, 36.6% had histories of perinatal difficulties, compared with 8.0% of nonincarcerated delinquents on whom information existed ($\chi^2=9.480$, $p=.003$, Yates's correction applied). These perinatal difficulties ranged from maternal syphilis to postnatal apnea requiring intubation.

Psychiatric symptoms were found more commonly in the medical records of incarcerated delinquents (44.2% versus 26.2% of nonincarcerated children) ($\chi^2=4.951$, $p=.027$, Yates's correction applied).

In an effort to determine which combination of medical history variables investigated in this study best distinguished the incarcerated delinquent group from the nonincarcerated delinquent group, a multiple regression analysis was conducted, using membership in the incarcerated or nonincarcerated group as the dependent variable. Predictor variables were presence of perinatal difficulties, child abuse, psychiatric symptoms in the subjects, number of emergency room visits, clinic visits, ward admissions, accidents or injuries, head or face injuries, and visits for upper respiratory infections before age 16. (It should be noted that the results of a multiple regression analysis in this two-group situation are identical to those of a discriminant function analysis in a greater than two-group situation [18].) Two variables, presence of perinatal difficulties and number of head and face injuries, produced an R of .382, accounting for 14.6% of the total variance. Presence of perinatal difficulties accounted for 12% of the total variance and number of head and face injuries for another 2.6%.

We were also interested in determining whether there was a combination of variables that was especially effective in distinguishing the two groups relatively early in their lives. Consequently, another multiple regression analysis was conducted, using membership in the incarcerated or nonincarcerated group as the dependent variable and, as independent variables, presence of perinatal difficulties, number of emergency room visits, number of clinic visits, num-

ber of ward admissions, number of accidents and injuries, number of head and face injuries, and number of visits for upper respiratory infections, all before age four. The results of this analysis were remarkably similar to those reported above. The combination of presence of perinatal difficulties and number of head or face injuries before age four produced an R of .365, accounting for 13.3% of the total variance. Presence of perinatal difficulties accounted for 12% of the total variance and number of head and face injuries before age four for an additional 1.3%.

To summarize, perinatal difficulties and head and face injuries most clearly distinguished incarcerated from nonincarcerated delinquents, a pattern of findings evident from early childhood through adolescence. There were also tendencies for abuse to be more prevalent in the histories of incarcerated delinquents and for psychiatric symptoms to be noted more often in histories of incarcerated children. On the basis of our clinical observations we were surprised that the presence of child abuse failed to distinguish the two groups at conventional levels of statistical significance. When we reexamined the medical records, we became convinced that the presence of child abuse was significantly underrecognized in the histories of the incarcerated group. It is our clinical impression that the incarcerated children constituted an especially abused group.

ESPECIALLY VIOLENT DELINQUENTS

We wondered whether differences in medical histories existed between more and less violent children within the incarcerated delinquent population.

At the time of this study, a new "secure unit" for especially dangerous juveniles from all over Connecticut had been constructed at the correctional school where our study sample was incarcerated. Over a 9-month period, 11 children from the New Haven area with histories of particularly violent offenses had been placed on this secure unit. We decided to compare the medical histories of this especially aggressive group with the medical histories of the general correctional school population. Because of the small number of secure-unit children from the New Haven area, we did not attempt to match for age or race in our comparison sample. We decided instead to match only for sex and geographic area. That is, since all 11 secure-unit children were male, we compared them with a random sample of 56 male delinquents from the New Haven area who had been incarcerated at the school but had not been placed in secure custody. All of the 11 secure-unit children had committed serious offenses against persons (e.g., assault, rape, arson), compared with 51% of the regularly incarcerated boys.

A comparison of secure-unit boys and the regularly incarcerated boys indicated that the secure-unit population was significantly younger than the comparison group at the time of hospital record review (179 months versus 263 months, $t=5.654$, $p<.001$). Of the secure-unit boys, 40.0% were white, compared with 62.5% of the incarcerated group. This difference was not significant.

Although total numbers of hospital visits did not differ significantly between the groups, ward admissions, especially those before age 2, tended to be greater in number for secure-unit children (1.000 versus 0.577, 0.182 versus 0.038 before age 2). Secure-unit boys tended to have more accidents and injuries before age 14; they also tended to have abuse or neglect and head and face injuries noted in their hospital charts more often than regularly incarcerated boys, but the differences did not reach conventional levels of significance.

Of the 6 secure-unit boys for whom perinatal data were available, 5 (83.3%) had perinatal difficulties, compared with 9 of 32 regularly incarcerated boys (28.1%) for whom perinatal data were available ($p=.019$, Fisher's exact test). Thus, significantly more violent offenders had perinatal problems. Moreover, our sample of violent offenders tended to have more accidents and injuries, ward admissions, and abuse than their less violent incarcerated peers.

DISCUSSION

On the basis of our clinical and epidemiological findings, it would seem that serious delinquents had extremely serious medical histories. Head and face traumas were numerous and severe in serious delinquents.

We must modify our previous report that delinquent children were no more likely to suffer from perinatal trauma than nondelinquent children. Our previous study focused on children with minor delinquencies. Seriously delinquent children, particularly those who had committed violent acts, were significantly more likely to have sustained perinatal trauma than the average delinquent child. Furthermore, seriously delinquent incarcerated children were likely to have been physically abused and/or neglected, although such abuse was underrecognized by hospital personnel.

On the basis of our present study of medical histories and our past work on the relationship of delinquency and psychopathology, there would seem to be identifiable biopsychosocial factors that together contribute to violent delinquent behavior. The combination of trauma to the central nervous system, parental psychopathology (14) (often expressed through incredible physical and psychological abuse), and social deprivation (particularly as manifested by the failure of physicians to recognize and appropriately treat psychiatric illness and/or central nervous system dysfunction in lower socioeconomic group children) creates the kind of serious, often violent, delinquent acts so prevalent in our society today.

We found that trauma to the central nervous system occurred early, either in the form of perinatal diffi-

culties or accidents and injuries. This finding has possible implications for primary prevention of some serious delinquency.

We would suggest that a single factor (e.g., brain damage, social deprivation, vulnerability to psychosis) is insufficient to engender violent delinquency. Unfortunately, often the combination of familial vulnerability (e.g., as indexed by the presence of a schizophrenic parent), trauma to the central nervous system (e.g., perinatal trauma, head injury), physical and psychological abuse from a parent, and social deprivation (e.g., failure of a physician to diagnose and treat correctly, or failure of society to provide adequate support systems in the form of community programs or residential treatment) is sufficient to create the violent young offender, and this combination of factors occurs frequently.

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The Relationship of Early Maternal Separation to Borderline Personality in Children and Adolescents: A Pilot Study

BY SUSAN J. BRADLEY, M.D.

The author compared 14 child and adolescent borderline patients, diagnosed by the criteria of Gunderson and Singer (1), with a control population for history of separation from the mother or significant caretaker before age 10. The borderline patients experienced a statistically greater number of separations than the psychotic controls, the nonpsychotic psychiatric controls, and the non-psychiatrically-referred delinquent controls before age 5 but not between ages 5 and 10. The author concludes that the study provides statistical support for the theoretic role of disruption of the early infant-mother bond in borderline pathology.

THE GROUP OF individuals labeled "borderline" is a somewhat controversial population, about whom much has been written (see reference 1). However, until the paper by Gunderson and Singer (1) appeared, the literature lacked a base from which research into etiologic factors could proceed. These authors identified six criteria, generally accepted by other writers as well, to characterize borderline persons. They are 1) the presence of intense affect, usually depressive or hostile; 2) a history of impulsive behavior; 3) social adaptiveness; 4) brief psychotic experiences; 5) bizarre, dereistic, illogical, or primitive responses on unstructured psychological tests such as the Rorschach but not on more structured tests such as the Wechsler Adult Intelligence Scale; and 6) relationships that vacillate between transient superficiality and intense dependency.

Etiologic theories of the borderline personality (2, 3) have not been validated, partly because of the lack of acceptable diagnostic criteria but also because of the difficulty in defining and measuring some of the hypothesized variables. Although still partially theoretic, works such as those of Bowlby (4–6) on the process of attachment and the effects of separation in children and of Mahler and associates (7) on the separation-individuation process and its vicissitudes provide insight into variables of likely relevance, some of which are measurable.

Bowlby (4) has stressed the generally deleterious effect of early parent-child separation and supports his position with studies that demonstrate an increased incidence of early parental loss in psychiatric patients, depressed persons, and juvenile delinquents. Some studies (8, 9) have reported that the incidence of early parental loss in sociopaths is particularly high. These statistics, however, may be misleading because the term "sociopath" has frequently been used loosely to refer to anyone manifesting antisocial behavior. A 1961 paper by Earle and Earle (8), which demonstrated a higher incidence of sociopathy in a maternally deprived population than in non-maternally-deprived psychiatric controls, included no statement of how the authors defined sociopathy. As they did not include a borderline personality category, one might wonder whether borderline personalities were included in the sociopathy category—the distinction is sometimes difficult to make. To date, there has been to my knowledge no attempt to look specifically at the incidence of early maternal loss in a borderline population, although both Bowlby (4) and Mahler (7, chapter 16) have theorized that severe character defects such as those seen in borderline patients are often the outcome of early maternal deprivation or difficulties in resolution of the separation-individuation phase (which may be related to actual loss).[1]

Although the Gunderson and Singer paper was concerned largely with the adult borderline patient, I believe that their criteria can also apply to child and adolescent borderline patients. This paper reports an attempt to explore the history of single or repeated significant periods of separation from mothers or other significant caretakers in a population of child and ado-

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[1]Bowlby does not use the term "borderline," but in his paper on childhood mourning he repeatedly emphasized his belief that inability to deal with angry feelings over loss of the mother results in a variety of different pathologies including extreme and prolonged detachment, use of splitting as a defense mechanism, and severe depressive reactions, all of which are characteristic of the borderline personality.

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lescent borderline patients (defined by the Gunderson and Singer criteria). These data are compared with similar information from two control populations.

METHOD

Borderline patients were diagnosed by the six criteria employed by Gunderson and Singer (1). It is my opinion that a history of brief psychotic episodes and/or looseness in unstructured situations (e.g., projective testing) with intactness in structured situations are necessary criteria for the diagnosis of borderline personality. In young children, the intensity of affect is sometimes difficult to assess. However, the other criteria are generally applicable and become even more so as the children reach adolescence.

The patients were seen by me in routine clinical assessments in three different consultation settings: a child and adolescent department of a large psychiatric institute, a preschool day treatment center, and an assessment center for juvenile delinquents in the province of Ontario.

One of the two control populations consisted of nonborderline children and adolescents referred to me for assessment and day-care patients known to me. The charts of both of these groups were reviewed. This population was selected to control for severity of illness. The other control group consisted of delinquent adolescents who were not referred for psychiatric assessment—presumably a somewhat healthier group than those referred. These adolescents represent a nonspecific "character disordered" population. Information on this group was sought from the case managers who were most knowledgeable about each adolescent's background.

There were 14 patients diagnosed as borderline and a total of 68 controls. The subgroups of the control populations break down as follows: 1) 45 psychiatric controls (12 psychotic, 33 nonpsychotic) and 2) 23 nonpsychiatric (i.e., delinquent) controls. The mean age of the borderline patients was 10.9 years for boys (N=10) and 12 years for girls (N=4) (range=5–15 years). The mean age of boys in control samples (N=48) was 11.2 years and of girls (N=20), 11.1 years (range=3–16 years). Accurate placement of these families on a socioeconomic scale was not possible with the available data. My clinical estimate of the families I knew personally would put most in Hollingshead and Redlich (10) social classes III, IV, and V, with 1 borderline family and 3 control families in class II.

Data were gathered on the borderline and control populations with respect to history of separation from mothers or significant caretakers. Separation from fathers was not included unless the father was the significant caretaker. Separation was defined as removal of the child or lengthy absences (greater than 3–4 weeks) due to illness of the mother or caretaker. As there was difficulty in obtaining information about separation due to maternal illness, the bulk of the separations reflect removal of the child or loss of mother or caretaker. Adoption with placement within 1 month after birth was not considered a separation. Once this information was obtained, all separations were divided into those occurring from birth to age 5 and those from ages 5 to 10, reflecting Bowlby's theoretic view of more and less vulnerable periods.

RESULTS

The data, presented in table 1, were analyzed by means of chi-square tests. Those children who had not yet had their 10th birthdays were excluded from the analysis of separations between ages 5 and 10.

Comparison of the total borderline patient sample to the total control sample makes it evident that the history of early but not late separation is greater in the borderlines ($p<.01$). The same applies in comparing the borderlines to the psychiatric controls. Because of the possibility that the borderlines represent a less severely disturbed subgroup on a continuum from neurosis to psychosis (11), borderlines were also compared with psychotic controls. Again, a history of early separation was significantly more frequent in the borderlines.

The adolescent borderlines were compared separately with psychiatrically referred delinquents ($\chi^2=7.2$, $p<.01$) and non-psychiatrically-referred delinquents ($\chi^2=11.59$, $p<.1$), and the same preponderance of increased early separations appeared in the borderline group. Both of these control groups contain a significant number of characterologically disturbed individuals. The incidence of early separations in these delinquents is quite comparable to that found by Glueck and Glueck (11) in a population of delinquents.

The mean age at the time of the first separation was 9.6 months for the borderlines, with a range of 3–30 months, and 13.8 months for the controls, with a range of 1.5–36 months. These differences were not significant.

DISCUSSION

The finding of an increased incidence of early separations in borderline patients supports Bowlby's statements about the critical effect of early disruption of the primary bond. They also lend weight to Mahler's notions regarding the part played by failure to resolve the separation-individuation phase of development in the etiology of the borderline personality, if we may assume that actual separation interferes with the process.

These findings have not generally been noted in other writings on borderline patients, and one wonders if those studies included large numbers of character-disordered individuals who might not have fulfilled the Gunderson and Singer criteria. Many of the controls

TABLE 1
Comparison of Separation Experiences in Borderline Patients and Controls

Group	Birth to 5 Years Old				5–10 Years Old			
	Sample Size	One or More Separations			Sample Size	One or More Separations		
		N	%	χ^2		N	%	χ^2
Total borderlines	14	9	64	—	9	2	22	—
Total controls	68	8	12	19.49[a]	56	7	13	.614
Psychiatrically referred	45	6	13	14.62[a]	33	3	9	1.16
Psychotic	12	1	8	8.5[a]	7	0	0	.30
Nonpsychotic	33	5	15	11.34[a]	26	3	12	.62
Delinquents (non-psychiatrically-referred)	23	2	9	12.87[a]	23	4	17	.1

[a]$p<.01$.

fall into a group that will ultimately be designated character-disordered, and it is clear that the incidence of early separations in this group, although elevated, is not nearly as high as in the borderline population. In fact, there is a tendency for this group to experience more separations as they age, some of which are related to removal from their natural homes because of uncontrollable behavior.

Because many of the borderline children manifest hyperactivity and learning disabilities, one would suspect that genetic and constitutional factors might be important variables in the production of the borderline picture. In fact, Masterson (3) suggested that most of the mothers of his borderline subjects were themselves borderline personalities. Although Masterson sees the mother's pathology as contributing to her difficulty allowing separation, one could also postulate that genetic or constitutional factors in the child may produce a vulnerability that makes the child overreact to experiences of separation, thereby reinforcing the mother's tendency to fear separation from the child.

The fact that a significant number of the borderlines were adopted raises further questions about genetic predisposition. Some people have, in fact, suggested that a high percentage of all illegitimate children are the product of incestuous unions.

One wonders if children who had multiple placements were constitutionally very difficult to manage and, therefore, subject to more frequent foster home placements. Consistent with this possibility is the fact that many of the adopted children were not placed until several months after birth—a somewhat unusual situation and one which raises the possibility that, even as infants, they might have had either physical or behavioral anomalies.

An obvious weakness in this study is the lack of validation of independent diagnoses. Although the Gunderson and Singer criteria, if adhered to carefully, allow reasonable consistency in the selection of cases, there is still room for disagreement in the interpretation of some of the criteria. The small sample size is the other obvious defect and would point to the need for a replication of this study that takes both factors into account.

In conclusion, the present study provides experimental evidence for the theoretic role of disruption of the early infant-mother bond in borderline pathology.

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The Development of an Acute Short-Term Inpatient Child Psychiatric Setting: A Pediatric-Psychiatric Model

BY MOHAMMAD SHAFII, M.D., ANN MCCUE, R.N., M.S., JOHN F. ICE, M.D., AND JOHN J. SCHWAB, M.D.

The authors describe the establishment of a unit for children that emphasizes the integration of the pediatric model of acute, short-term inpatient care with the psychological and developmental perspective of the psychiatric model. Of the 145 children admitted during the first year, more than 33% manifested aggressive or hyperactive behavior and 25%, depression or suicidal behavior. Eighty-five percent were discharged to their homes or previous residences after an average length of stay of 24 days. The authors suggest that similar units established in children's hospitals or general hospitals could help meet the urgent need for acute inpatient psychiatric care of children in this country.

THE MENTAL HEALTH needs of children and youth in this country are in a stage of crisis. According to the 1969 report of the Joint Commission on Mental Health of Children, "We are providing care to only one-third of our children who are in serious need of attention" (1, p. 5). Since then, with the decline in federal funding for mental health services, this situation has deteriorated further. In 1972 the Group for the Advancement of Psychiatry reported, "When the community mental health centers were originally set up, it was stipulated that all ages be served. Nonetheless, most of the initial community mental health centers did not and do not provide facilities to serve children" (2, p. 112). Although 71 million people (33% of the total population) are under 18 years of age, only a disproportionately small percent of our mental health resources are used for this group. Studies by Berlin (3), Adams (4), and Rexford (5) all verify that mental health services for young people are grossly neglected.

From antiquity to the present time, in many societies children have been perceived and treated in ambivalent and distorted ways. De Mause (6) has described the hair-raising and heartbreaking historical trends in the neglect and maltreatment of children throughout the ages. Legitimate and illegitimate children have been killed for many reasons including crying too much or too little, not being perfect in appearance or size, and for being female. Child labor and other protective laws did not reach humanitarian standards even in the United States until recently (7). Although reforms have corrected many abuses, our ambivalence toward children is expressed overtly by neglect and child abuse and covertly by the incarceration of emotionally troubled children in county jails, their long-term hospitalization in dilapidated institutions, and negligence in creating humanistic, short-term inpatient psychiatric facilities for them.

INPATIENT PSYCHIATRIC CARE FOR CHILDREN

Residential Model

Some states do not have any inpatient psychiatric facilities for young people (1, p. 6). Most of the states with long-term psychiatric inpatient facilities for children do not have acute, short-term inpatient services. Under various names, often categorized as "residential settings," the orphanage model for long-term custodial care of children and youth is still the basis of psychiatric care in most public hospitals in this country (8, p. 2246). The residential model places emphasis on screening procedures to ensure that the new patient fits the milieu. It is not uncommon for a child to wait 3–6 months for admission screening, and according to the Joint Commission report only 1 out of 10 children screened is eventually admitted (1, p. 6). Many children stay in such facilities 1–3 years; the average stay is 1–18 months. The emotional impact of the child's long-term separation from family, neighborhood, and school needs careful scrutiny. After all, a 9 or 10-year-old child who is hospitalized for 1 year has spent about 10% of his or her life away from home. Following the child's discharge from the hospital, it may take

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a long time for him or her to find a place within the family again.

Pediatric-Psychiatric Model

The pediatric-psychiatric model emphasizes the integration of the pediatric model of efficient, acute, short-term inpatient care with the psychiatric model of intensive individual psychotherapy, family therapy, group therapy, milieu therapy, school, art, and activity therapies. The strength of such a program rests on its immediate responsiveness to a crisis situation and rapid admission of the child to the hospital—with preparation but without delay. There should be either no waiting list or a very short one. According to an NIMH survey, the median length of stay in state and county mental hospitals for young people under age 18 is "considerably longer" than the length of hospitalization for persons of all ages (72 days versus 41 days) (9, p. 4).

There is a 33% to 66% shortage of psychiatric beds for children and youth (reference 10 and an unpublished paper by J. R. Whittinghill), although "there are more than 100,000 unnecessary hospital beds across the country" (11, p. 2). Could some of this unused bed space be converted to acute, short-term inpatient psychiatric units for children and youth?

Psychiatry is moving into the mainstream of medical practice. The creation of pediatric-psychiatric units within children's hospitals or general hospitals along with other special units, such as the high-risk nursery, intensive care unit, burn unit, and the pediatric-adolescent unit, is essential for the total care of children. In a pediatric-psychiatric unit all of the necessary diagnostic assessment, therapeutic intervention, and discharge planning can be done concomitantly. The average stay should not exceed 3–4 weeks. From the beginning, the treatment plan should emphasize reintegration of the child with his or her family. The cost of such special units and the occupancy rate should be similar to those of other special units in the medical and pediatric settings.

THE LOUISVILLE PROGRAM

The Bingham Child Guidance Clinic, established in 1913, is the fourth oldest child guidance clinic in the nation. It is the outpatient facility of the Child Psychiatric Services, University of Louisville School of Medicine. For many years the staff of the clinic felt a need for an inpatient child psychiatric facility to provide care for emotionally disturbed children who could not be managed in the outpatient setting.

Setting

Norton-Children's Hospitals' futuristic structure encompasses Norton Hospital with beds for 300 adults, Children's Hospital with 156 beds, Norton Psychiatric Clinic with beds for 36 adults, and the Bingham Child Guidance Clinic. In 1975 one of the 8-bed pediatric units in Children's Hospital was designated for the care of emotionally disturbed children. This unit was named the Ackerly Child Psychiatric Service in honor of S. Spafford Ackerly, former chairman of the department of psychiatry and director of the Child Guidance Clinic for more than 30 years.

The 8-bed unit contains approximately 2,000 square feet and 7 rooms. Two large bedrooms accommodate 4 patients each, and 2 rooms have single beds. A multipurpose room functions as a recreation, dining, and family room. Another small room is used as a family visiting room; on occasion, for diagnostic and treatment purposes, the parents stay overnight in this room and participate in their child's daily activities. An additional room serves as the office for the medical records and as a staff conference and consultation room.

Staffing

To provide an efficient, economical inpatient unit for children, the inpatient and outpatient services were integrated. For example, the child psychiatrist in charge of the inpatient program also supervises and leads a team that provides outpatient services. Child psychiatric faculty, fellows and residents, psychologists, a clinical specialist nurse, and social workers provide services for both the inpatient and outpatient units. Nursing staff and child care workers not only take care of the children on the unit but also accompany them to the classroom and assist the teacher and art therapists.

A Child's Day in the Hospital

Children are seen regularly 3–5 times per week in individual, group, or family therapy. They spend 2 hours per day in a one-room country-style school located in the outpatient facility. A special-education teacher, paid by the county school system, evaluates the child's educational level, develops an individual learning plan using educational materials from the child's home school, and acts as a liaison person to facilitate the child's reentry into the home school. The children receive credit from their home school systems for attendance in the hospital school program. In addition, the daily schedule calls for 2 hours of creative and expressive art therapy in the art therapy studio adjacent to the classroom. The children use the adult psychiatric inpatient gymnasium for 1 hour of planned recreational activity per day. A Red Cross van takes them on weekly outings and to community activities.

In contrast to the practices of many other child psychiatric units, parents usually visit their children daily for 1 hour, at times in the presence of the staff. We encourage these frequent visits in an effort to involve the parents actively with the child's treatment program and to develop healthier attitudes toward the child and each other.

After discharge many of the children are followed by the same therapist for 3–6 months or longer on a week-

ly or biweekly basis. Continuity of care and active follow-up play a significant role in further stimulating the healing process within the child and the family.

Patient Population

During the first year 145 young people (approximately 20% of the outpatient population) were admitted to the inpatient unit. There was an almost equal number of boys and girls. The patients ranged in age from 2–16 years; 43% were 12–16 years old; 39%, 6–11; 14%, 4–5; and 4%, 2–3 years old.

Diagnoses

Thirty-six percent of the hospitalized children manifested unmanageable behavior such as aggression, uncontrollable and impulsive behavior, or hyperactivity. Seventeen percent had psychophysiologic disorders such as anorexia nervosa, asthma, cyclic vomiting, enuresis, encopresis, diabetes, and seizure disorders associated with behavioral disorders. Sixteen percent had psychoneurotic disorders; the majority of them were depressed. About 10% of the children were given the diagnosis of schizophrenia, infantile autism, or other forms of childhood psychosis. Ten percent were admitted because of suicide attempts. Six percent had organic brain syndromes, mental retardation, or other psychopathology; and 5% were suffering from child abuse.

Outcome

During the first full year of the unit's operation, 82% of the 145 children admitted were discharged to their homes or previous residence; most of them continued to receive outpatient follow-up care in the Bingham Child Guidance Clinic and other community agencies. Eighteen percent required placement outside of the home, but only 2 patients (1.5%) were placed in a long-term state psychiatric facility. Eleven patients (about 7.5%) were readmitted for further assessment or treatment on a short-term basis.

The occupancy rate was 122%; 2–3 extra beds were needed to help meet the demand. The average length of stay was 24 days; the median length of stay in state and county mental hospitals for patients under the age of 18 is 72 days (9, p. 4). Thus the patients in our acute treatment unit were removed from their homes for relatively brief periods of time. Further, this short period of hospitalization obviously reduces the cost. Many hospitals and departments of psychiatry have not developed child inpatient psychiatric services because they usually cost more than adult units. Our experience shows that the cost of the short-term inpatient child psychiatric unit was similar to that of adult inpatient psychiatric units and special pediatric units in this area.

CONCLUSIONS

In our experience the successful outcome of the acute inpatient child psychiatry program is based on the following:

1. Freedom from the traditional residential model.
2. Creation of a pediatric-psychiatric model with emphasis on the integration of the immediate response of pediatric practice with the careful psychological and developmental perspective of the psychiatric model.
3. Integration of outpatient and inpatient resources to provide continuity of care rather than the traditional division.
4. Recognition that inpatient care of children is only one small phase of the therapeutic process.
5. Effective and efficient emergency service in the outpatient setting to assess the child's need for immediate hospitalization.
6. Immediate response to the need for hospitalization rather than long and cumbersome screening procedures and waiting lists.
7. Active outpatient treatment and follow-up with the same therapist for consolidation and enhancement of therapeutic gains.

Similar pediatric-psychiatric models established within children's hospitals or general hospitals and managed by the child psychiatric staff could respond to the urgent need for acute inpatient psychiatric care of children and youth in this country.

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Adolescents' Adjustment to Chronic Hemodialysis

BY ATARA KAPLAN DE-NOUR, M.D.

The author compares the adjustment of 18 adolescents on chronic hemodialysis to that of 63 adult patients on hemodialysis. The adolescents tended to comply less well with the medical regimen, their vocational rehabilitation was significantly lower, and their social activities were more restricted. The adolescents suffered more from the changes in body functioning and appearances. They were more hostile and overtly aggressive but seemed to have fewer psychiatric complications and, especially, less suicidal ideation. The author suggests that chronic disease is especially difficult for adolescents to handle because it creates stress in areas already problematic for this age group.

THE LITERATURE on the reactions of adolescents to chronic disease is still limited. One problem in their adjustment relates to adolescents' narcissism, which makes them vulnerable to disfigurement and loss of function as well as fearful of injury and/or pain caused by the disease or by treatment. The second problem area is that of alienation. Disease and/or hospitalization causes the adolescents to feel different from others, and they become alienated from their peer group. Sick adolescents increase the anxiety of their peers, who often react with rejection, further increasing their alienation. The third problem area is the increase in adolescents' ambivalence about dependency-independence. Some adolescents solve this ambivalence by regressing to passive and often cooperative behavior. Others try to master their surroundings and fight the medical regimen to the extent of harming themselves.

The advance of chronic hemodialysis as a routine method of treatment enabled us to study a large population of chronically ill patients. In our studies, just under 20% of the patients were adolescents (aged 14 to 19). The aim of the present report is not to highlight the problems of chronic hemodialysis but to concentrate on the reactions of adolescents to chronic disease; the reactions of adolescents will be compared to those of adults undergoing identical treatment. Such a comparison will enable us to determine more clearly whether adolescent patients are indeed different from adult patients, and it may promote understanding of the sources of their special problems and delineate therapeutic interventions.

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METHOD

The adolescent group included 18 patients 14 to 19 years old, 10 girls and 8 boys. None of the adolescents was illiterate and about two-thirds had more than elementary school education. Two other age groups were taken as comparison groups: group 1 included 28 patients, 10 women and 18 men aged 20 to 34, and group 2 included 35 patients, 15 women and 20 men aged 35 to 49. The level of education in the two comparison groups was much lower: group 1 contained no illiterate patients but only 50% had more than an elementary school education. In group 2 this percentage dropped to 23%, and 29% of the patients in this group were illiterate.

All of the patients were observed as part of a predictive study on the influence of personality on adjustment to chronic hemodialysis. They were interviewed for the first time before dialysis began and were followed up 6, 12, 24, etc., months after commencement of dialysis. In each follow-up, patients' adjustment was assessed. Assessment of compliance with the diet was based on biochemical data gathered from the patients' charts. Vocational rehabilitation referred only to the number of hours per week of work or study; these data were gathered from patients, nurses, and social workers. Patients' psychological condition was assessed by clinical interviews. Details of the method of predialysis and follow-up interviews have been described previously (1, 2).

RESULTS

None of the adolescents or patients in the comparison groups refused the predialysis interview although they knew that psychiatric research was one of our aims. The interviews with the adolescents, however, were difficult; they were withdrawn, some shy and others sullen. They usually did not talk spontaneously and preferred not to discuss their disease and previous treatments or the upcoming treatment—only 2 girls were overtly anxious and 2 boys inquired about the mechanical aspects of dialysis. On the other hand, the majority of the older patients talked a lot and discussed their disease and previous medical treatments spontaneously and often at length and discussed the

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TABLE 1
Adjustment to Chronic Hemodialysis of Adolescents and Two Adult Comparison Groups

Aspect of Adjustment	Age Group			Analysis	
	14–19 (N=18)	20–34 (N=28)	35–49 (N=35)	χ^2	p
Nonfunctioning before hemodialysis (percent)	37.0	21.4	2.9		
Diet compliance					
Good	3	8	9		
Fair	6	9	17	4.044	n.s.
Poor	9	11	9		
Vocational rehabilitation					
Yes	3	17	23	12.48	<.01
No	15	11	12		
Psychiatric complications					
None	8	6	11		
Moderate	8	14	10	7.202	n.s.
Severe	2	8	14		
Suicidal ideation					
Yes	2	8	21	13.73	<.01
No	16	20	14		

prospects of chronic dialysis from many aspects. The behavior of the adolescents in the follow-up interviews also differed from that of the other patients: the adolescents were usually overtly angry, much more so than the other patients. They expressed anger at the medical staff, at their families, and often at fate.

Table I summarizes various aspects of adjustment to dialysis. The difference in compliance between the adolescents and the comparison groups did not reach statistical significance yet there was a tendency toward less diet abuse in older patients; half of the adolescent group and only a quarter of group 2 abused the diet.

The adolescents' level of functioning decreased much more than that of the comparison groups even before dialysis, and the vocational rehabilitation of the adolescents at follow-up was significantly poorer than that of the comparison groups. Those considered to have achieved vocational rehabilitation worked more than half-time (or were in school). The majority of patients who did not meet this criterion did not work at all.

Unlike the adults, the adolescents were inclined to discuss at length their problems in social activities. Thirteen of the 18 adolescents reported marked decrease in social activities, which they usually did not attribute to physical malbeing. As far as we could ascertain, only 1 of the adolescents had heterosexual social activities. The patients in the comparison groups talked much less about social activities and reported some but no great decrease, which did not seem to disturb them.

The information gathered in the follow-ups about patients' psychological condition is presented in three categories: "none," meaning that no psychiatric complications were observed at any of the follow-ups or that there were only minor symptoms; "moderate," meaning that moderate depression and/or anxiety was observed in at least one follow-up; and "severe," meaning that either moderate depression and/or anxiety was permanent or severe in at least 1 follow-up, and/or that psychotic symptoms were observed. The difference between the adolescents and the comparison groups did not reach statistical significance. However, a tendency was found—the percentage of patients with severe psychiatric complications increased with age. Suicidal ideation was significantly less frequent in the adolescent group and increased with age.

A few words should be added about the adolescents' problems in other areas. The adolescents often discussed bitterly the decrease in or lack of urination. They also complained more than the adult patients about their appearance, e.g., about the pale yellow tinge of their skin. While on dialysis their relations with other patients, including other adolescents, were limited. They talked little to other patients or to nurses, and they seemed to sleep or try to sleep more than the adult patients.

DISCUSSION

The adjustment of the 18 adolescents in our study to chronic hemodialysis was different from that of adult patients. In some aspects of adjustment the differences did not reach statistical significance, but trends could be observed; the adolescents' compliance with the diet was poorer than that of the adults, and the adolescents had fewer and less severe psychiatric complications. The adolescents also had a significantly lower frequency of suicidal ideation. We would like to suggest that the adolescents' tendency to have fewer psychiatric complications is related to the finding that they were overtly angry and often verbally aggressive.

The adolescents were found to do significantly less well than the comparison groups in vocational rehabilitation. This difference is striking considering that the

adolescents had more education and none of them were unskilled workers, factors which promote rehabilitation. Adolescents' social activities were also more limited than the adult patients'; their alienation from their peer group was severe, and they suffered greatly from these changes. Body image problems were much more prominent in the adolescent group.

In summary it can be said that the reactions of adolescents to chronic disease (chronic hemodialysis) are indeed different from the reactions of adult patients. We would like to suggest that these differences are caused by the fact that the stresses of chronic disease which adolescents have to handle lie in areas that are already problem areas because they involve the tasks of adolescence. Consideration of the psychological meaning of injury or illness (3) supports this suggestion. The ill person has a sense of responsibility for the damage, and, in addition, adolescents still have the remains of magical thinking and are prone to displace guilt and to feel guilty about their disease. Illness also means separation from family and friends. Adolescents' ambivalence about their families has often been described, and attaining emotional independence is regarded as a major task of adolescence. Furthermore, normal adolescents feel a strong need to belong to their peer group and not to feel different. Moore (4), who studied patients with malignant disease, expressed the opinion that feeling different is more stressful for adolescents than the threat of death. Disability affects future plans, and learning to plan is considered one of the main tasks of adolescence. Illness and disability cause a feeling of inadequacy, which is often a problem for normal adolescents, as well as the feeling of personal rejection that often accompanies disability. Weinberg (5), who studied suicidal thoughts in physically ill adolescents, found that in boys these thoughts were indeed directly connected to the physical disease and accompanying feelings of inadequacy. In girls, suicidal thoughts were connected to the interpretation of disease as rejection.

Illness and disability often mean or produce sexual problems, and developing one's sexual role and identity is a main task of adolescence. The last point Hamburg (3) mentioned is that disease leads to increased hostility, and the normal adolescent's problems in handling increased aggression have been mentioned by all writers.

All of the problems that have been mentioned in relation to the psychological meaning of disease and disability are even more extreme in the chronic dialysis situation where the frustration of drives and their derivatives is prominent, body image problems are extreme, and life is machine dependent.

One of the requirements for achieving good adjustment to chronic hemodialysis is the capacity for partial regression (6). We believe that adolescents, who have to handle their own ambivalence and struggle about dependency-independence in a situation that is overwhelmingly dependency producing, cannot achieve this delicate balance of partial regression. They use total regression, which results in poor vocational rehabilitation. The inability to handle the normal task of planning for the future while disabled increases the tendency to regress.

A combination of factors seems to be responsible for the abuse of the medical regimen: total regression leading to lowered frustration tolerance, acting out of independence against the adult world, including the medical team, and the need to deny being different from the peer group may all lead to noncompliance.

It seems, therefore, that the regression found in the adolescents is one of the factors responsible for their poor compliance and poor rehabilitation. At the same time, it is believed to be the reason for the comparative absence of psychiatric complications—adolescents express their aggression directly instead of handling it by projection or introjection.

The present findings raise the question of what can be done to improve adolescents' adjustment. It seems that nephrologists also believe that chronic hemodialysis is no way of life for adolescents and prefer kidney transplant as treatment for these younger patients. Transplants were performed on 14 of the 18 adolescent patients (78%) and only 19 of the 63 adult patients (30%). Transplantation is by no means a cure, but some of the stresses are reduced, especially the problems of dependency.

We doubt whether psychotherapy can be of great help to the adolescents. We have found psychotherapy with chronic dialysis patients extremely difficult (7). The prospects seem gloomy indeed, in light of the difficulties of psychotherapy with adolescents, as stated, for example, by Lecker and associates (8): "Psychotherapy with adolescents has come to be known as being difficult, frustrating and unsatisfactory for both the adolescent and the therapist." We would like, however, to suggest that the set-up of treatment could and should be modified to decrease the stressfulness. Any change that would decrease the dependency and increase the adolescent's feeling of mastery and adequacy would reduce the stress and might thus prevent total regression. We therefore agree wholeheartedly with those (9) who recommend special units with staff specially skilled for handling young people.

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Juvenile Firesetters: Do the Agencies Help?

BY STUART FINE, M.B., AND DON LOUIE, M.D.

In an attempt to find out what was being done for juvenile firesetters in their metropolitan community the authors reviewed the records of the local fire marshal, juvenile court, and psychiatric clinic. They found that the various agencies involved with 69 juveniles over a 3-year period were unable to coordinate their efforts and were largely unsuccessful in controlling the firesetting. Because the families of firesetters seem to have many other social problems, the authors recommend early inpatient assessment and possible referral to foster homes or residential treatment settings for juvenile firesetters.

IN RESPONSE TO the Provincial Minister of Health's expression of renewed concern about fires set by juveniles, we attempted to find out what was being done for firesetters in our community, a metropolitan area with a juvenile population of about 653,000. We started out with the impression that working with the child in his or her family setting was not effective. Several studies (1–3) have found that children who often set fires and who are assessed by psychiatrists show evidence of a great deal of psychopathology, that other members of their families are often also disturbed, and that firesetting is seldom the only or the presenting complaint.

Much has been written about the psychodynamics of firesetting (4), but very little has been written about management. Although it is not a frequent presenting symptom for referral to child psychiatry clinics, firesetting can cause great economic loss as well as morbidity and mortality.

In a survey of 20 cases Vandersall and Wiener (1) described three subgroups. In the largest subgroup, composed of 9 children, many were infantile, impulsive, and clearly deviant; several of these children also demonstrated severe retardation and developmental deviations. Macht and Mack (5) noted intrapsychic turmoil and chaotic functioning in the families of 4 adolescent firesetters. These studies cited the unavailability of fathers, especially at the time of the firesetting.

In a large survey of 60 juvenile firesetters, Lewis and Yarnell (2) stressed the aggressive component in firesetting, but much of the earlier literature stressed the erotic component. Lewis and Yarnell also pointed out the frequency with which sexually immature adolescent boys working in pairs are found among firesetters. This group seems to be trying to find social acceptance through such exploits. Older adolescents, however, seem to act alone and seem to be even more difficult to treat. In a survey of the attitudes of 300 urban adults toward children who set fires, Winget and Whitman (6) showed that only one-third thought that mental health professionals might be of use to firesetters. Several suggested punishments like "burning him so he'd know better," "calling in a policeman to give him a good scare," and "whipping him and talking to my minister about him."

METHOD

To find out how juvenile firesetters were assessed and managed in our community we met with representatives from the fire marshal's office and from the juvenile court. They stated that these children and their families were very difficult to involve in programs to change their behavior. We noted that there was partic-

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ularly poor coordination of services for the child who sets fires and his or her family among the different agencies. The approach of the fire marshal's office was to "scare the children so that they would stop."

At the fire marshal's office we examined the records of all reported cases of firesetters aged 5–16 years from 1971 to 1973. We found 78 cases, 26 of which had been referred to the juvenile court. We obtained permission to go to the court to examine the records of those cases, which showed that 11 youngsters had been referred from the juvenile court for psychiatric opinion. We then obtained permission to look at the psychiatric reports.

DATA

Sixty-five fires were set by 36 juvenile firesetters in 1971; 18 fires were set by 11 firesetters in 1972; and 87 were set by 31 firesetters in 1973. The population of metropolitan Vancouver included 636,600 juveniles aged 5–16 years in 1971 and 652,900 in 1973. No census figures are available for 1972.

The 78 firesetters included 9 who set fires in more than 1 year. We thus have data for 69 firesetters. Four of these were girls. Twenty-seven of the juveniles set fires on their own and 44 operated in groups (2 did both solo and group firesetting). Fifteen were over 12 years of age. Twenty-six (37.7%) were referred to the juvenile court, and 11 (15.9%) had psychiatric assessments. Two were referred to a residential treatment center. Twenty-three firesetters were involved in school fires; the others were involved in home, car, railroad car, and lumber fires.

Of the 26 adolescents referred to the juvenile court, 9 were placed on probation, 5 had a "stay of proceedings," and 2 were not brought for a court hearing at all. The remaining 10 were not followed by the juvenile court at all. The reason for referral to the court was seldom the firesetting alone but complaints such as running away from home and "incorrigibility."

According to the psychiatric histories of 11 of the juveniles, 7 were from broken homes and 6 had parents with severe psychopathology, including suicide, homicide, psychoses, and alcoholism. The children themselves were suffering from learning disabilities manifested in school failure, depressive reactions, and family adjustment problems. Three were mentally retarded. Referral for psychiatric assessment was related to such behavioral difficulties as running away from home and to school difficulties rather than primarily to firesetting. All 11 psychiatric referrals had been previously admitted to a general hospital for at least one accident, such as a fracture or accidental ingestion of poison.

Two case histories may help to emphasize the diversity of the symptoms associated with firesetting and the difficulties psychiatrists and other helping professionals may experience in trying to help these children and their families. Both of these cases were reported to the fire marshal's office and were known to probation services.

CASE REPORTS

Case 1. A, an 11-year-old boy, was referred for psychiatric evaluation after setting fire to a car outside the school from which he had recently been expelled. He had been to the psychiatric clinic 2 years previously because he was making slow progress at school and his parents had difficulty in disciplining him. He had difficulties with reading and writing and for 3 years had been in a special class for children with learning disabilities. He also suffered from primary enuresis. He had been evaluated by several agencies and professionals and had been found to have an overall IQ on the WISC of 76 (verbal, 71; performance, 86).

A was the third-born in a family with 4 children; he had 2 older brothers and a younger sister. His father had been unemployed for more than a year at the time of the assessment and was known to drink heavily. A's mother was talkative and circumstantial but well-meaning. A's siblings also had had academic and behavioral problems.

A's developmental milestones were all delayed. According to his parents he was a happy and contented infant and toddler, but he became difficult to manage when he went to school.

The clinic's records showed that A was always pleasant and cooperative when seen individually but that in a group setting he became disruptive and aggressive. His parents were ineffectual in controlling his behavior, and his father was often out of the home because he went drinking at the local pub. All of the members of the family expressed anger by hitting, by developing somatic symptoms, or by running away (one brother had run away twice).

A tutor, a worker from a social welfare agency, a family service agency worker, and staff from the psychiatry clinic had all been involved in A's treatment. Despite all the intervention, A had been expelled from two schools in quick succession, and he set fires. His mother had initially resisted suggestions about residential treatment but finally agreed to let him go to a residential treatment center. We heard that he stopped setting fires while there.

Case 2. B, a 14-year-old boy, was referred for psychiatric evaluation because he threatened to assault his mother and had broken some of the furniture in the house. He was suspected of setting about 10 fires in his neighborhood over a period of 9 months. Before his referral he had refused to go to school for a year. He had been taking a correspondence course for 3 months.

B had been adopted at the age of 10 days. He had an adopted sister 2 years younger than he. His adoptive father was a taciturn man who had irregular hours at his job. His mother was a tense woman who had difficulty in controlling the behavior of her very tall 14-year-old son and rather large 12-year-old daughter. There were no members of the extended family in the province.

B was described as a difficult baby from the time of adoption. He became particularly difficult at 21 months when his sister was adopted. He showed quite marked separation anxiety when sent to kindergarten. At the age of 9 he was referred to a child psychiatrist and was given dextroamphetamine for "minimal brain dysfunction." There was little response to amphetamines. He was then referred to a special

education clinic, which he attended once a week for 4 months. His behavior improved. After 3 months in high school he stopped attending school and spent long periods of time in his room. He refused to leave the house.

The psychiatric record stated that B was a tall (6 feet) 14-year-old who was withdrawn and suspicious of the interviewer. He seemed to have a poor understanding of the interview situation. However, on intelligence tests he scored in the average range.

B and his family were seen weekly over a period of 4 months. Thioridazine, 25 mg, t.i.d., was prescribed. He admitted to setting several fires but would not elaborate on how he felt before, during, or after the setting of these fires. He was suspicious throughout his contact with the therapist. Two residential facilities refused to consider him because he had set fires and they felt they could not "take the chance." He was eventually placed in a facility that placed great emphasis on outdoor activities. The therapist thought B had borderline personality integration, and at times he was frankly psychotic.

The people who dealt with B seemed to have doubted that this well-meaning youth had set so many fires. The fire marshal's office and the patient's family did not inform personnel at the child psychiatry clinic about further firesetting during treatment. B's firesetting ceased in the residential treatment center.

DISCUSSION

The two cases cited illustrate the long history of difficulties that seem to be typical of juveniles who repeatedly set fires. They also illustrate that in some families removal of the child from the home environment for at least a short period of time seems to be necessary to stop the maladaptive behavior, even though removal of the child from the home setting goes against prevailing social work policies. The parents in most of these families were found to be inconsistent in disciplining their children.

The psychiatric reports of the 11 cases reviewed confirmed the findings of other workers that many complaints and symptoms other than firesetting were reported. For example, several children had frequent accidents, which might reflect a lack of supervision in the home.

The agencies that dealt with these children were unable to coordinate their efforts. Not all of the firesetting reported to the fire marshal's office was recorded there. There was minimal intervention when a child firesetter referred to the juvenile court was placed on probation. At best, the probation officer, would see the child once a month, and very little change would occur in a child's behavior and interpersonal relationships.

These children are difficult to assess and treat, and their families seem to be difficult to reach. Unless active efforts are made to intervene, repeated firesetting occurs and tragedies ensue. Fras (7) suggested very directive instructions to parents. If the parents are unable to control the firesetting after obtaining help, other caretakers should be considered. Six months of unsuccessful therapy should be the most allowed. Family therapy for "loosely linked" families may be difficult (8), and individual therapy in these cases seems to be of very limited value. An inpatient evaluation should be obtained and subsequent foster home placement or residential treatment should be seriously considered. "Treatment" of this behavior disorder in the community seems to be particularly unsuccessful.

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BRIEF COMMUNICATIONS

The Mini-Mental State Test and Computerized Tomography

BY LUKE TSAI, M.D., AND MING T. TSUANG, M.D., PH.D.

In order to evaluate the clinical usefulness of the Mini-Mental State (MMS) test, the authors had 63 patients complete the test after undergoing computerized tomographic (CT) scans of the brain. Patients with positive CT scans scored significantly lower than those with negative CT scans. Further analysis showed that cerebral atrophy was more closely related to cognitive impairment than focal cerebral lesions only. The authors conclude that the MMS can discriminate organicity in some instances but that further refinement and modification of its subtests are needed for maximum usefulness.

COMPUTERIZED TOMOGRAPHIC (CT) scan of the brain is a noninvasive neuroradiological technique for diagnosing intracranial lesions and dementia (1-3). However, this technique is costly and generally follows a clinical diagnosis of suspected organic disorder of the brain. The clinical diagnosis of organic brain syndrome basically depends on the findings of the mental status examination regarding impairment in orientation, memory, and other intellectual functions (4).

There are standard psychometric tests to measure cognitive ability; the most widely used are the Clinical Test of Sensorium, the Weschler Adult Intelligence Scale, and the Bender Gestalt Test. However thorough these tests may be, they all require a great deal of time to administer, making them impractical in certain clinical situations, such as testing elderly patients or patients with dementia syndrome (5). Thus there is need for an expedient clinical method of measuring cognitive capability that can identify those patients who are likely to have organic brain syndrome.

The Mini-Mental State (MMS) Test, developed by Folstein and McHugh (6), is a short series of questions testing orientation, registration, attention and calculation, recall, and language. Advocates attest to its high degree of validity and reliability in identifying three distinct psychiatric disorders: organic dementia, uncomplicated affective disorders, and depression with cognitive impairment (7).

We have undertaken to evaluate the usefulness of this test by comparing the results of the MMS with findings from CT scans of the brain. We hope to determine whether the MMS can predict organicity and also discriminate among types of organic conditions of the brain.

METHOD

Subjects

The 63 patients in our study group were selected from patients consecutively referred to the radiology department of the University of Iowa Hospitals from November 30 to December 15, 1976. These patients were referred for CT scans of the brain from the departments of neurology and psychiatry of the same medical center. In most cases suspected organic brain syndrome prompted the referral.

Because the MMS involves reading and writing, patients with poor cooperation, motor-sensory aphasia, paralysis of the arm on the dominant side, severe impairment of vision and hearing, or impairment of consciousness due to stupor and coma were excluded. Ac-

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L.C. Chiu, M.D., Department of Radiology, University of Iowa College of Medicine, assessed the computerized tomographic findings. Jerome A. Fleming, M.S., provided assistance in performing the statistical analysis of data.

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cording to these criteria, 20 patients were excluded before we concluded the selection process with 63 subjects.

Procedure

The CT scans were performed using the Ambrose method (8). The determination of ventricular size and degree of enlargement followed methods established by Huckman and associates (9). The radiologist who assessed the CT findings was aware of the purpose of our study but had no knowledge of the individual cases of patients participating in the study.

Within 24 hours of the CT scans one of us (L.T.) administered the MMS to each of the 63 patients. He had no knowledge of the CT findings and scored the MMS tests independently.

Although the entire test contains 11 items and generally requires no more than 10 minutes, the test is not timed. Detailed instructions for administering the test have been provided by Folstein and McHugh (6). The maximum possible score is 30, and according to Folstein and McHugh, who used a selected group of patients to study the validity and reliability of the MMS, the mean score for patients with dementia was 9.7; depression with cognitive impairment, 19.0; and uncomplicated affective disorder, depressed, 25.1. The mean score for a control group of normal elderly persons was 27.6.

After the CT scans were completed and the MMS administered, the 63 subjects were divided into 2 groups based on the results of the CT scans. Group 1 included the 32 patients with negative CT scans and group 2, the 31 patients with positive CT scans. In order to be judged positive, the CT printout had to show evidence of cerebral atrophy and/or focal lesions. Focal lesions include tumor, hematoma, abscess, infarction, and subarachnoid hemorrhage. According to the criteria of Huckman and associates (9), cerebral atrophy is diagnosed if 1) the ventricles are larger than 15 mm but cortical sulci are 5 mm or less, 2) the ventricles are larger than 15 mm and cortical sulci are greater than 5 mm, or 3) the ventricles are 15 mm or less but the cortical sulci are greater than 5 mm.

Table 1 shows the distribution of the 63 patients according to CT findings and clinical diagnosis.

Group 2 was further subdivided into 2A and 2B. Subgroup 2B included the 18 patients whose positive CT scans resulted from cerebral atrophy with or without focal lesions, while subgroup 2A included the 10 patients with focal cerebral lesions only. Three subjects originally in group 2 were excluded because the ventricular size could not be measured due to compression of the ventricles by tumors.

Comparisons between groups 1 and 2 and between subgroups 2A and 2B were carried out using a t test for two means, Pearson correlation coefficient, and chi-square with Yates correction; $p<.05$ was considered to be statistically significant. The mean total scores of each group were compared, as were the mean scores on individual subtests.

TABLE 1
Clinical Diagnosis and CT Findings in 63 Patients

Diagnosis	Group 1[a] (N=32)	Group 2[b] (N=31)
Psychiatric disorders		
Organic brain syndrome	2	7
Alcoholism and drug dependence	2	1
Other	2	0
Neurological disorders		
Intracranial neoplasm	0	8
Cerebrovascular accident	1	7
Epilepsy	7	2
Headache	2	0
Other diseases of the CNS	5	2
Other	6	3
Other systemic disorders	5	1

[a]Negative CT scans.
[b]Positive CT scans.

TABLE 2
Mean MMS Test Scores for Groups 1 and 2

	Group 1 (N=32)		Group 2 (N=31)		
Item	Mean	SD	Mean	SD	p
Total MMS score	26.4	5.6	20.4	8.3	<.01
Orientation	8.7	1.8	6.1	3.2	<.01
Registration	2.8	0.6	2.7	0.7	n.s.
Attention and calculation	4.2	1.5	3.1	2.1	<.02
Recall	2.3	1.0	1.1	1.2	<.01
Language	8.5	1.5	7.5	2.4	<.05

TABLE 3
Mean MMS Test Scores for Subgroups 2A and 2B

	Group 2A[a] (N=10)		Group 2B[b] (N=18)		
Item	Mean	SD	Mean	SD	p
Total MMS score	25.3	5.4	18.0	8.6	<.05
Orientation	8.1	2.1	5.2	3.3	<.02
Registration	3.0	0.0	2.7	0.8	n.s.
Attention and calculation	3.6	1.8	2.9	2.2	n.s.
Recall	2.0	0.8	0.6	1.0	<.01
Language	8.6	1.0	6.8	2.8	n.s.

[a]Focal cerebral lesions only.
[b]Cerebral atrophy with or without focal cerebral lesions.

RESULTS

The difference between mean total scores reached a statistically significant level ($p<.01$) when group 1 was compared with group 2 (26.4 ± 5.6 versus 20.4 ± 8.3). Group 2 scored lower on all of the subtests, but there was not a statistically significant difference between the 2 groups on the registration subtest. The tests of orientation, attention and calculation, recall, and language showed significant differences between the 2 groups (see table 2).

There was a significant difference in mean total scores ($p<.05$) between patients in subgroups 2A and 2B (25.3 ± 5.4 versus 18.0 ± 8.6) (see table 3). Patients in subgroup 2B scored significantly lower than those in

subgroup 2A on the subtests of orientation and recall, but the subtests of registration, attention and calculation, and language did not discriminate between the 2 subgroups.

COMMENT

The MMS test offers clinicians a quick, easy-to-administer measurement of cognitive functions. We have tried to determine its value and efficiency as a screening process for CT scans. Definite trends were observable. Group 1, the patients with negative CT scans, had significantly higher mean total scores and significantly higher mean scores on all subtests except registration than did group 2, the patients with positive CT scans. The mean total score for subgroup 2B, patients with cerebral atrophy (with or without focal lesions), was significantly lower than that for subgroup 2A, patients with focal lesions only. On the subtests of orientation and recall, the mean scores of subgroup 2B were significantly lower than those of subgroup 2A.

In order to determine whether findings of cognitive impairment were primarily caused by cerebral atrophy rather than focal lesions alone, we compared the results from group 1 with those from subgroup 2A. The comparison showed that there was no statistically significant difference between the mean total scores or between the mean scores of any of the subtests. That is, there appeared to be no significant difference in performance between patients with negative CT scans and patients with only focal lesions. These results strongly suggest that diagnostically subgroups 2A and 2B are two distinct groups.

Shapiro and associates (10) found that in testing attention and calculation, the Serial Sevens Test could not differentiate organic from functional mental disorder. Hinton and Withers (11), on the other hand, found that brain syndrome did significantly reduce patients' scores on this test. In our investigation there was a significant difference between the mean scores of group 1 and group 2 only when the scores of the Serial Sevens Test were combined with the scores from the Spell "World" Backwards Test. These results suggest the need for further investigation of the clinical merits of this subtest.

Within the subtests of language, 100% of the patients from group 1 and 90% from group 2 could correctly name a watch and a pencil; 94% of all patients could perform the reading task correctly (reading and obeying the statement, "Close your eyes"). The value of these items is dubious.

The Copy-Design Test, a modification of the Bender-Gestalt Copy Design Test, has a reputation for detecting organic brain syndrome (12). In our study, this was the only test question within the language subtest that differentiated between groups (correct copying in 94% of group 1 versus 55% in group 2; $p<.01$).

Patients with negative CT scans performed better than those with positive scans on the Sentence Repetition Test, although there was no significant difference between mean scores. The other two questions testing language faculties (writing a sentence and following a three-stage command) elicited no significant differences between groups. The value of these subtests must also be questioned.

Hinton and Withers (11) reported that the correlation between age and scores on clinical tests of sensorium was not high. Our investigation revealed that the correlation between age and total MMS score was not significant in group 1, whereas there was a significant negative correlation in group 2 ($r=-.426$, $p<.02$). Further analysis of group 2 showed that there was no statistically significant negative correlation in either subgroup 2A or 2B. However, in subgroup 2B there was a trend toward a negative correlation ($r=-.402$, $p<.10$). These results indicate that in patients with negative CT scans or focal cerebral lesions alone, the total MMS score will not be affected by age; however, in patients with cerebral atrophy with or without focal lesions, there is a trend toward a negative correlation between total MMS score and age. Although Folstein and McHugh (6) reported that the total MMS score difference was not due to age, the influence of age on MMS scores needs further investigation.

Hinton and Withers (11) also noted that there was a direct or indirect relationship between the scores of clinical tests of sensorium and the length of schooling but that this relationship had little predictive value. In our study there appeared to be a significant difference ($p<.05$) in length of schooling between groups 1 and 2 (50% versus 23% had more than high school education). However, further analysis comparing group 1 and subgroup 2A with subgroup 2B showed no significant differences. Our study appears to support Hinton and Withers' observation.

In summary, it appears that the Mini-Mental State Test has some clinical value for diagnosing organic brain syndrome. We found that the total MMS score and the subtests of orientation and recall were useful for discriminating between cerebral atrophy and focal cerebral lesions. However, on the basis of our findings, we recommend that future study seek to further refine and modify various test questions.

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Urinary Excretion of Dimethyltryptamine in Liver Disease

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The urinary excretion of N,N-*dimethyltryptamine (DMT) was higher in patients with severe liver disease than in normal subjects. This difference remained significant when patients with all grades of hepatic encephalopathy were excluded. Patients with liver disease whose mental states were normal excreted amounts of DMT similar to those of patients with a hospital diagnosis of schizophrenia.*

N,N-DIMETHYLTRYPTAMINE (DMT) is a hallucinogen that occurs normally in human urine in very small quantities (1). We have twice reported that its excretion is increased in some patients with psychotic illnesses (2, 3), although these observations are disputed (4). The meaning of any increased excretion of DMT in psychotic illness is uncertain, although superficially it lends credence to the hypothesis that DMT is involved in the pathogenesis of some psychotic symptoms (5, 6). To examine the significance of any increased excretion of DMT in mental illness, a search has been made for other conditions in which the excretion of DMT might be similarly raised. The liver contains enzymes sufficient for the synthesis of DMT (7) and for its degradation. It might be expected therefore that in liver disease the urinary excretion of DMT would be raised, partly because its metabolism might be impaired and partly because freshly synthesized DMT might be released from damaged liver cells. If endogenously formed DMT of extracerebral origin causes psychotic symptoms, then patients excreting abnormal quantities of DMT should experience psychotic phenomena, provided that the patients are sufficiently alert.

The recognized psychiatric effects of acute liver disease are those of an acute organic reaction (8). The severity of this reaction may be measured by a clinical rating of the level of consciousness on a 5-point scale that has been validated against EEG findings (9). This grading system will indicate those patients who are too drowsy to describe any mental effects of altered metabolism of DMT. The central question raised in this paper is whether patients with liver disease who have normal mental states excrete abnormally large quantities of DMT.

METHOD

Urine collections (24-hour) were made from patients admitted to a specialized liver unit. The patients had a wide range of liver diseases, most of which were both severe and acute. Patients with a raised blood urea concentration were excluded.

Similar collections were made from a group of normal volunteers who have been described previously (1) and from a group of patients with a Maudsley Hospital diagnosis of schizophrenia, made by using the British Glossary of Mental Disorders (10). We derived standardized diagnoses from the Present State Exami-

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nation (11) using the Catego program: S+ (N=14), P+ (N=2), and P? (N=1) and D+ (N=1). These patients form part of a larger clinical study that will be reported separately (3). Urinary DMT was determined by using a specific gas chromatographic method (12).

The Mann-Whitney Statistic and the Spearman Rank Correlation Coefficient were used for statistical analysis, as individual values for each biochemical measure were not normally distributed.

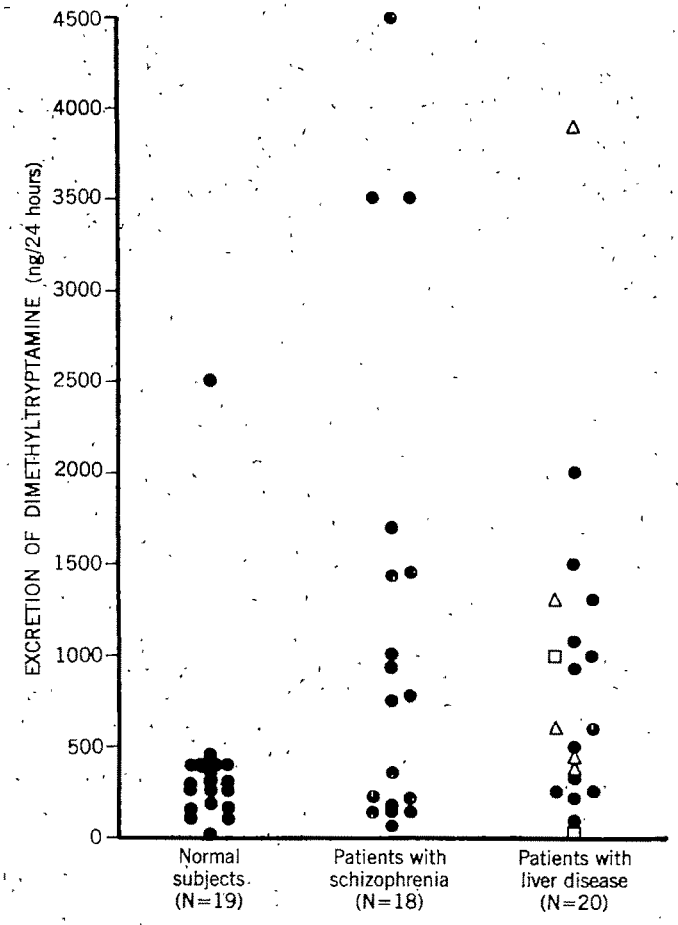
RESULTS[1]

The patients with liver disease (N=20) were well matched with the schizophrenic patients (N=18) and the normal subjects (N=19) for age and for renal function. The fact that they had a different sex ratio should not have affected the results, since there was no relationship between sex and the excretion of DMT within the control groups.

The excretion of DMT was higher in the patients with liver disease than in the normal subjects ($p<.01$). This difference remained significant ($p<.05$) after exclusion of patients with all grades of hepatic encephalopathy. The excretion of DMT was similar in the patients with all grades of hepatic encephalopathy. The excretion of DMT was similar in the patients with liver disease and schizophrenia (figure 1). Although the difference between schizophrenic patients and the normal patients failed to reach statistical significance, the present finding that 10 out of 18 patients with a diagnosis of schizophrenia excreted more than 500 ng of DMT in a day closely replicates our previous report that 20 out of 42 patients with a diagnosis of schizophrenia excreted detectable DMT (2). The detection limit of that method was 500 ng/24-hour urine collection.

Results of the routine liver function tests demonstrate the severity of the illnesses that were studied. Mean plasma concentrations for albumin, total bilirubin, alkaline phosphatase, and aspatate transaminase were 29 g/liter (range, 15–41), 203 nmol/liter (range, 37–480), 577 IU/liter (range, 94–2750), and 402 IU/liter (range, 61–2500), respectively. No significant relationships were found between the excretion of DMT and values for individual liver function tests; rather it seems that the excretion of DMT is raised across a wide range of different forms of acute liver disease. The raised excretion of DMT cannot be attributed to drugs since the difference between patients with liver disease and the normal subjects remained significant ($p<.05$) after we excluded patients who were receiving drugs that might affect the excretion of DMT. These drugs included steroids, other immunosuppressive therapy, and insulin but not antibiotics or vitamins. Changes of diet and sterilization of the gut have already been shown to have no effect upon the excretion of DMT (1). Three of the patients studied had taken paracetamol overdoses. Independent psychiatric evaluation in each case of overdose excluded the possibility of a functional psychosis. In view of the hallucinogenic properties of DMT it is interesting that no patient in the present series described either hallucinations or illusions.

FIGURE 1
Median Urinary Excretion of Dimethyltryptamine[a]



[a]Grade of encephalopathy of patients with liver disease: ●, grade 0; △, grades I and II; and □, grade IV.

DISCUSSION

We have shown that the excretion of DMT was higher in a group of patients with liver disease than in a group of normal subjects. This difference remained significant after exclusion of patients with hepatic encephalopathy. The increased excretion of DMT in the patients with liver disease was similar to that seen in patients with a hospital diagnosis of schizophrenia.

It seems most likely that the raised excretion of DMT in these patients reflects raised blood concentrations and not merely an increased renal clearance. DMT readily crosses the blood brain barrier—euphoria has been reported within minutes of an intravenous injection of DMT (13). If the patients with liver disease have raised concentrations of DMT in their

[1]Tables are available on request from Dr. Rodnight.

blood, it is likely that they will also have raised concentrations of DMT in their brains; however, their mental states appeared unaffected by such concentrations of a known hallucinogen. None reported hallucinations, and although some showed features of hepatic encephalopathy, there was no relationship between the excretion of DMT and the grade of encephalopathy. The present series suggests that an increased extracerebral production of DMT has no detectable effect on the mental state of a patient with liver disease; however, there may be an interaction between the effect of DMT and the neurochemical effects of liver disease.

The increased excretion of DMT in patients with liver disease and a normal mental state has to be compared with the observation that the parenteral administration of DMT causes hallucinations. It may be that DMT is only hallucinogenic when present in larger quantities than are found in patients with liver disease and schizophrenia. This explanation is supported by the observation that the excretion of DMT in subjects receiving hallucinogenic doses of DMT (13) is approximately 100 times greater than that found in our psychotic patients (2).

Although the present study suggests that the extracerebral production of DMT in these quantities has no effect on a patient's mental state, the possibility remains that DMT is formed within the brain and influences mental phenomena. One experimental strategy for manipulating the metabolism of any DMT that is formed within the brain is the administration of monoamine oxidase inhibitors. These drugs increase the excretion of DMT (1), probably by inhibiting its metabolism. It is therefore of interest that when these drugs have been given to drug-free schizophrenic patients under double-blind controlled conditions, the patients have reported no symptomatic change (14–17). We conclude that it may be useful to study the relationship between DMT and psychosis by examining other situations in which the production of endogenous hallucinogens may be increased.

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Adjustment of a Group of Vietnamese People to the United States

BY A. JOE VIGNES, M.D., AND RICHARD C.W. HALL, M.D.

The authors collected data on 50 Vietnamese families living in Baton Rouge, Louisiana, by individual and group interview questionnaire. They also used interviews of all of the Vietnamese people who came to the local community mental health center and studied the records of the center and other local social agencies for sociodemographic data. They define a number of specific sociological stresses encountered by Vietnamese refugees but conclude that basically these people are adjusting well without losing their cultural identity.

A GREAT DEAL has appeared in the lay press concerning the adjustment of Vietnamese people to the United States. The scientific literature remains largely silent on issues of this transcultural adjustment. We undertook the prospective study described here in an attempt to evaluate the psychiatric and social factors affecting the adjustment of Vietnamese refugees in the United States.

METHOD

We randomly selected 50 Vietnamese families, representing a total population of 114 individuals, from a list of newly settled Vietnamese in Baton Rouge, Louisiana. All 50 of the families initially surveyed agreed to participate in the study after the nature of the study was explained. The study design consisted of four elements.

Social Adaptation Questionnaire

One individual from each of the 50 families participated in a two-hour structured interview followed by a one-hour informal interview. All of the data thus obtained were used to develop a formal questionnaire to rate the overall adjustment to American society of all the subjects (N=114).

The seven investigators of the survey team (three Vietnamese and four Americans) initially chose 300 questions. These 300 items were presented to an individual from each of the 50 families. The responses were analyzed for clarity of meaning when translated into Vietnamese, subject acceptance, and lucidity of the translated concept. One hundred items were chosen for inclusion in the final questionnaire. Fourteen percent of the questions involved the area of self-image; 14%, religion; 12%, community relations; 12%, future expectations; 10%, relationship to authority; 9%, family relations; 7%, adjustment while in Viet Nam; 6%, finances; 6%, political orientation; 5%, marital relations; and 5%, sexual adjustment.

A 7-point discriminative scale ranging from positive adjustment (+3) through 0 to a negative adjustment (−3) was devised. The survey team subjectively rated each question. Questions felt to have the highest positive discriminatory adjustment value were rated +3 (i.e., "I feel happy in the United States"), and neutral items were rated 0 ("I approve of people who drink socially"). Items indicating the poorest adjustment were rated −3 ("I do not find it easy to make friends with Americans"). Items between these extremes were rated +2, +1, −1, and −2. Twenty-four percent of the questions had a value of +3 or −3; 23%, +2 or −2; and 23%, +1 or −1. Thirty percent of the questions were considered neutral and had a value of 0.

Social Adjustment Scale Questionnaire

Five Vietnamese college students administered a bilingual (Vietnamese-English) questionnaire to the 50 families in their homes. The questions were asked and answered in Vietnamese. The data from these questionnaires were analyzed by computer, and total adjustment scores were assigned to each respondent. These scores were then correlated with sociodemographic variables.

Social Survey Data

Sociodemographic data were collected from local social agencies, such as the community mental health center, state and private psychiatric institutions, schools, and law enforcement agencies. Agency utilization data, arrest records, and medical and psychiatric information were gathered for all of the Vietnamese in the Baton Rouge area.

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Psychiatric Evaluation

One of the authors (A.J.V.) interviewed all of the Vietnamese people who came to the local community mental health center during a one-year period.

DATA

Twenty-three percent (N=114) of the total Vietnamese population of Baton Rouge participated in the study. Sixty percent of these individuals were men; 58% were married. Ages ranged from 19 to 77 years with a mean age of 35.6 and a median age of 37. Eighty-three percent of the group had completed secondary school, and 60% had received at least a year of university training. Forty percent of the population stated that they were currently involved in some type of educational program. Twenty-two percent of the men interviewed had practiced a profession when they were in Viet Nam, but only 6% functioned in a professional capacity in the United States.

Four percent of all of the Vietnamese families in the area were receiving welfare (compared with 9% of all families in the area). During the study 6 minor traffic violations involving a Vietnamese were recorded. No Vietnamese had been arrested for any of the 6 categories of crime surveyed, which included rape, homicide, driving while intoxicated, theft, assault, and disturbing the peace. School officials reported language barriers as the major area of educational concern. The children presented no behavior problem in the schools. Educators felt the initial passivity and withdrawal of the Vietnamese children had abated markedly during their first year in school. No peer problems or difficulties with school authorities were reported for the Vietnamese children.

Scores on the social adjustment questionnaire for our sample of 50 families ranged from a low of −47 to a high of +70, with a mean score of 25.5 and a median of 24. Subjects scoring +25 or above were placed in the high adjustment group, and those scoring +24 or below were placed in the low adjustment group. Eight percent of the respondents received negative adjustment scores (mean negative rating, −17.2). Ninety-two percent of the respondents received positive adjustment scores (mean positive rating, +29.2). High and low adjustment group data were analyzed using a two-tailed chi-square test for significance of differences. Data were analyzed for differences of adjustment, determined by age, religion, income, education, and marital status. No significant differences were defined except for income. The high-income group in the United States was significantly less well adjusted than the low-income group. All of the subjects showing negative adjustment scores (N=10) had been high-income professionals in Viet Nam (income greater than $10,000) and also fell into the high-income United States group (income greater than $10,000).

The records and interviews at the local community mental health center and the records of state hospitals and private psychiatrists in the Baton Rouge area revealed 5 cases of schizophrenia during the study year. Three of these patients had been diagnosed as schizophrenic while in Viet Nam. All of the patients were men and were diagnosed as having paranoid schizophrenia. Two patients, 1 male and 1 female, had had an acute nonschizophrenic psychotic reaction that responded within 10 days to phenothiazine treatment. Two women who made suicide attempts were diagnosed as having acute psychoneurotic depressive reactions. Two men were diagnosed as having psychotic organic brain syndrome. One of these patients had viral pneumonia, and the other had acute hepatitis. The confirmed diagnosis and treated incidence and prevalence data are shown in table 1.

TABLE 1
Treated Incidence and Prevalence of Mental Illness in a Population of 488 Vietnamese Living in Baton Rouge, Louisiana, During 1976[a]

Diagnosis	Number of Cases	Treated Incidence[b]	Treated Prevalence[b]
Schizophrenia, paranoid type	5	.4	1.02
Acute reactive psychosis	2	.4	.4
Organic brain syndrome	2	.4	.4
Reactive depression	2	.4	.4

[a]Total treated incidence=1.64/1,000 individuals/year; total treated prevalence=2.25/1,000 individuals/year.
[b]Cases/1,000 individuals/year.

DISCUSSION

In 1975 Segal and Lourie reported to Rear Admiral S.G. Morrison that psychological shock and depression were the major psychiatric effects among Vietnamese relocated to Guam. They suggested a five-part program to meet the psychological and social needs of these people. Segal (1) reported that this program was followed, for the most part, by the authorities responsible for resettlement.

Our study suggests that the Vietnamese have adjusted well in the United States. Their treated incidence and prevalence of major psychiatric disorders was no higher than for the population at large. The 10 subjects in this study exhibiting poorest adjustment (8%), who were free of major psychiatric disorders, were in the highest socioeconomic strata in both Viet Nam and the United States. Loss of professional role and identity seemed to be major factors complicating their adjustment. In each case, the individual perceived extensive loss in both social and professional status. As a group they found it difficult to associate with other Vietnamese and were unwilling to socialize with the newly developing "upper class" of Vietnamese-Americans. This group reflected more on past losses and were more uncertain about the future than any other subgroup; they were also the least able to accept a socially forced change of occupation.

The difficulties predicted by the lay press for Vietnamese refugees adjusting to the American lifestyle have not materialized. Vietnamese children are doing well in school, the refugees have accepted American social norms, and the Vietnamese community has been able to maintain its cultural identity. The major problems reported by our subjects related to unemployment and underemployment. The need to supplement family income has forced many women to seek employment and to change their role in the family structure. The women reported favorably on their new "freedom," but it remains a source of consternation for men, who see it as an erosion of the patriarchal family structure. Recently developing marital discord also represented an important problem.

The major sociological stresses identified in this population were 1) loss of role identity, 2) loss of self-esteem, 3) social isolation secondary to language barriers, 4) local prejudice directed toward the Vietnamese, 5) Vietnamese prejudices directed toward the local community, 6) suspicion of helping agencies, and 7) suspicion of the U.S. government's motivation and intent.

The initial response of the Vietnamese families studied to resettlement in America was one of passivity followed in two to three months by expressions of anger and disappointment, which lasted at peak intensity for another four to six months. Following this phase, adjustment and acceptance increased dramatically. We noted no substantial deculturalization of Vietnamese traditions except for a change in the role of women. The women saw this as a positive change and were anxious to become more assertive.

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Peer Review of Group Therapy: Washington, D.C., 1972–1977

BY MARTIN G. ALLEN, M.D.

The author discusses the evolution of psychiatric peer review in Washington, D.C. In its first six years (1972–1977) the Washington Psychiatric Society Peer Review Committee evaluated 146 cases, 23 (16%) of which focused on group therapy. Eight of these cases (35%) were partially or fully denied. Most of the cases involved outpatients with neuroses or character disorders who were treated in combined group and individual therapy. The author presents specific cases illustrating group therapy issues and peer review decisions. He discusses the benefits and liabilities of peer review and emphasizes its importance as a flexible, clinically sensitive, confidential, and meaningful process.

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THE CONCEPT OF peer review in psychiatry and its actual implementation come at a time of increasing involvement by insurance companies and the federal government in the delivery of psychiatric services as well as greater consumerism in our society. In addition, there is the possibility of national health insurance, with psychiatric coverage. These developments have led to the medical audit as an effort to maintain cost control. The review of psychiatric services by peers is a result of all of these factors.

The APA Task Force on Peer Review has presented a model of peer review for psychiatry (1), including guidelines and methods of implementation. Its statement delineates the objectives of peer review as education of the physician and evaluation of quality, cost, and quantity/utilization. In addition, Sullivan (2) has emphasized the importance of confidentiality.

In Washington, D.C., psychiatric peer review first developed in 1972, probably due to a large population with insurance for psychiatric services and to the federal government's emphasis on accountability and audit procedures. Questions about cases usually come from insurance companies after screening by a psychi-

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atric consultant. Each case is reviewed by qualified peers who are experienced and understand the nuances and details of specific, specialized clinical problems. A decision is made in each case to accept, deny, or partially accept.

Chodoff and Santora (3) have discussed the Washington, D.C., method of operation and the overall experience with psychiatric peer review. The present paper presents a summary of peer review of all cases involving group psychotherapy in Washington, D.C., in the six years from 1972–1977. Specific cases illustrating group therapy issues and peer review decisions will be presented.

SUMMARY OF GROUP THERAPY CASES

From 1972–1977, 146 cases were referred to the Washington Psychiatric Society Peer Review Committee. Group psychotherapy was involved in 23 of these cases (16%). Of the 23 cases, all but 1 (from a psychiatrist) were referred by insurance companies. There were questions about 19 individual cases, 1 question about billing procedure from a psychiatrist, and 3 cases questioning patterns of care (in which several cases involving a single provider of care are submitted to peer review, with questions raised about a pattern of treatment).

A total of 60 questions were asked regarding the 23 cases, with an average of 2.6 questions per case. Twenty of the cases involved a combination of group and individual psychotherapy. The 3 cases in which group therapy was the only modality involved outpatients, and all of these were fully accepted by the Peer Review Committee.

Eight of the cases (35%) were either partially denied (6 cases) or completely denied (2 cases). A partial denial/approval occurred when some questions about the case were approved but others were denied. In Chodoff and Santora's report of the Washington, D.C., peer review experience from 1972 to 1975, 37% of all questions asked were denied. Thus the percent of denials for group therapy cases in the present study was similar to the overall Washington, D.C., experience. Three of the 8 denials were appealed, but the committee did not find any reason or further evidence to reverse its decisions.

Three of the cases involved inpatient treatment; 18 cases involved outpatients; 1, partial hospitalization (day care); and 1, inpatient and outpatient care. The patients' ages ranged from 8 to 69. Information about the diagnosis showed 12 cases of neurosis or personality disorder, 5 cases of psychosis, 3 cases of adolescent adjustment reaction, and 1 case of childhood adjustment problem.

Over the years the experience of the peer review committee has included a range of issues. There have been questions about patient selection, frequency of sessions, duration of treatment, and billing practices. Some of the issues specific to group therapy involved marathon sessions, rotating therapists, and multiple therapists.

CASE ILLUSTRATIONS

Several cases will be described in order to illustrate the types of questions asked of the peer review committee and the committee's response.

In one case the questions were: Is this marathon therapy? What benefits can be provided? The committee's response was as follows.

> The psychiatrist involved in this case has made clear this is not a "marathon session" in the usual sense of the word where most of the members may be strangers or at least not part of an on-going group therapy, but, in fact, are members of a regular group undergoing psychotherapy.
>
> There are a few well-known Washington area psychiatrists who feel that this procedure of "extended group psychotherapy" is a valuable adjunct to regular group psychotherapy. There are certain constraints which they feel should be maintained. They feel that there should be no more than four such sessions per year and that patients should be in at least once-a-week therapy the rest of the time. The "extended group sessions" should be conducted by the same person (or persons if a co-therapist is involved) who usually conducts the group.
>
> Since the group cannot possibly receive the same degree of attention normally exercised in a group session the hourly fee should be less, probably one-half, and never more than $100 for a weekend type session. There is no evidence to support the idea that extending a session to the point of breakdown of human endurance is therapeutic. The Committee feels that eight hours on a given day is the maximum for which one can expect useful work of this type.

Subsequent cases are often decided by precedents or criteria established in previous cases. The following case is an example.

> This case is concerned with "extended group psychotherapy sessions." The physician in this case documents the psychiatric condition for which he was treating the patient. He also gives clear documentation as to his reasons for the use of extended sessions. He meets the criteria we set forth in a previous case:
>
> 1. The patient was in regular group psychotherapy and in this case also in individual psychotherapy.
> 2. The physician held these extended sessions no more than four times per year.
> 3. On each of the successive days in which the physician held extended sessions, the total number of hours did not exceed 8.

The following is another example of an approved case.

> The problem presented in this case was "Is this an acceptable type of therapy?" The services rendered were described as "Network Intervention Therapy-Marathon Therapy." The therapy referred to is that of a network

treatment of a family in which the two children were diagnosed and billed as patients. The physician conducted five 50-minute sessions on two evenings approximately 2 weeks apart. In these sessions were the patients, all the family members, and all people with whom the patients had contact. The psychiatrist and his staff members conducted several strategy sessions on their own separate from these group network sessions. The psychiatrist charged at the rate of $8.75 per hour per patient. We feel that in this particular case the treatment seemed to be acceptable and also had useful end results in terms of the patients' treatment. We feel that the charges to the patients and their family were reasonable.

The following statement by the committee illustrates a decision of partial denial.

The patient is a 50-year-old married female with a long history of psychiatric care with diagnoses of schizo-affective schizophrenia and/or manic depression. The history supports the provider's summary that this patient is fragile, passive, socially isolated, anxious, and a difficult management problem.

Group therapy was begun to help this patient with her social isolation, feelings of alienation from her family, and as a supportive modality. However, in addition, the patient has been seen, weekly, in one 90-minute or two 45-minute psychotherapy sessions on the same day.

Our answers to the questions are:

1. A 90-minute or two 45-minute sessions in one day are not appropriate in this case. Our recommendation would be for individual sessions of no more than 50 minutes. Sessions more than once a week might be warranted during periods of crisis but once per week sessions should be sufficient for long-term treatment.
2. The committee does feel that weekly group psychotherapy sessions are appropriate. A once-weekly group therapy session would be appropriate, in addition to a once-weekly individual therapy session.

The last two examples are classified as complete denials.

Two child psychiatrists were conducting group psychotherapy with adolescents and each psychiatrist billed the patient separately. Each charged $20 per session. The question was asked: Is this appropriate utilization and appropriate cost?

Group psychotherapy with a co-therapist was found to be an appropriate treatment modality for these adolescents. However, it is not the usual and customary practice in this area for co-therapists to charge full fee, regardless of their training. A single charge is to be made for group therapy with or without a co-therapist. Although this can be billed singly or separately, the total should be within the range of the usual and customary fee for group psychotherapy in the area.

This patient was depressed, mistrustful, and ambivalent in close interpersonal situations and unable to make important decisions. Antidepressant medication was not successful . . . . Individual psychotherapy sessions were then conducted alternately with the psychiatrist and the psychologist. The patient was seen by both therapists individually, by the psychiatrist in group psychotherapy, and by the psychologist in group psychotherapy. In April, for example, the patient was seen weekly by the psychologist in group and in individual sessions and by the psychiatrist in group and individual sessions for a total of sixteen visits. On April 16, the patient was seen four times—once in each of the treatment modalities.

The peer review committee felt the patient was in need of psychiatric treatment. However, the pattern of therapy involving the two therapists and both individual and group sessions did not conform to any recognized treatment pattern and appeared to provide no continuity. This treatment program was not appropriate for this case.

DISCUSSION

Most of the cases with questions about group therapy were fully accepted by the peer review committee; only 2 were completely denied. The percent of complete denials is low, especially considering the fact that insurance companies have a screening procedure wherein questionable cases are reviewed by a nurse, and, if still questioned, they are sent to a psychiatric consultant. Many questioned claims are approved; only those not resolved at an earlier stage are referred to peer review.

How can we interpret the fact that few cases were denied? Is it reflective of loose standards, a lack of understanding, or a lack of clear standards and guidelines? It should be noted that 35% of the cases were completely or partially denied; this is a sizable percentage. In addition, we on the committee found that when clinicians communicated directly to us (and the chairman maintained confidentiality by deleting all names of persons and places), they felt more free to give complete details, knowing that the information would not be in the files of a bureaucracy. That helped us to understand a case more fully and to appreciate specific clinical decisions. Finally, when the peer review process began, standards and guidelines were not clear. As a result of our experience with peer review, we developed guidelines combining our experiences as clinicians, our experience with the peer review process, surveys of local practice, and recommendations from the APA guidelines. Currently our standards are being used by local insurance companies, and fewer cases have been referred for peer review.

Consequently, case review was not the only activity of the peer review committee. It also worked to develop standards for peer review, length-of-stay criteria for inpatient hospitalization (based on current local practice), and to review a collection of cases when questions arose about patterns of care. Obviously, all of these activities are interrelated.

The peer review process can have beneficial and detrimental results. Peers can review, and sometimes censure, questionable practices. The very existence of peer review serves as a deterrent to questionable clinical or billing practices. It also helps maintain cost control. However, at the same time peer review may inhibit innovation and experimentation with new approaches, combinations, and techniques. It also

becomes a factor in therapy, threatens confidentiality, and may interfere with treatment, especially with paranoid patients, patients involved in divorce cases or other legal proceedings, or patients in highly visible positions of power or prestige.

In conclusion, the three *A*s of modern society—automation, auditing, and accountability—are upon us. It is our responsibility to develop and implement a meaningful peer review process that is sensitive, flexible, and understanding of the realities of clinical issues. Through its decisions, peer review establishes standards of patient care. It is the clinician, not the bureaucrat, who must develop a realistic and viable peer review process—one that demonstrates confidentiality, accountability, and the highest quality of professional care.

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Medicaid Audit: Crisis in Confidentiality and the Patient-Psychiatrist Relationship

BY HARVEY J. SHWED, M.D., SEYMOUR F. KUVIN, M.D., AND RAVI K. BALIGA, M.D.

The authors describe their experience with a routine Medicaid audit and discuss the damage such a procedure can do to the trust between psychiatrists and patients, confidentiality, doctors' reputations, transference reactions, and patients' own emotional states. The authors point out that these crucial issues have broad implications because of the possibility of passage of national health insurance legislation.

CONFIDENTIALITY is undergoing intensive scrutiny because of the major upheaval in traditional concepts of the patient-doctor relationship in the past decade (1–3). Third-party insurers, whether private or government, armed with computers and data banks, have seemingly insatiable appetites for detailed information about what transpires between doctor and patient in order, they say, to justify the benefits they pay and to be assured that competent medical service has been rendered (4–9). What are the boundaries between the right to know and the doctor's time-honored pledge to patients of confidentiality?

Nowhere is confidentiality more basic to a discipline than in psychiatry and psychotherapy. Our experience suggests the possibilities for destruction of this patient-psychiatrist trust in a routine Medicaid audit of a psychiatrist's practice. (Medicaid is the federal-state program that pays for the health care of 28 million low-income Americans who meet eligibility guidelines for participation.)

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MEDICAID AUDIT PROCEDURE

In New Jersey, a Medicaid audit is an investigation of a physician and his or her practice. It is for the most part a routine check of doctors who treat significant numbers of Medicaid-sponsored patients. Medicaid staff limitations prevent the staff from scrutinizing all doctors who see Medicaid patients. They therefore give priority attention to doctors who see a large number of Medicaid patients or whose practice has raised the suspicion of fraud or abuse. We were audited because Medicaid patients comprise approximately 10%–15% of our private group psychiatric practice, which is located in a medical school-affiliated teaching hospital in Newark, N.J.

The Medicaid audit as typified by our experience includes the following:

1. The psychiatrist's office is informed of the impending audit and a mutually convenient appointment

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is made. The doctor is told that 25 or 30 patient charts will be reviewed the day of the audit and that preselected patients will also be interviewed by the audit team either before or after the formal visit to the psychiatrist's office. The identity of the patients is not revealed until the day of the audit. Members of the audit team who interview physicians in the field are usually not physicians.

2. The team interviews the psychiatrists and asks about such items as Board certification or eligibility, medical school affiliations, and names of other physicians and staff members within the practice.

3. The team asks for the charts of the preselected patients.

4. The team views and reproduces the charts. This procedure is especially important to them because a Medicaid physician at the central office ultimately reviews these patient records. In cases where a doctor is suspected of fraud or abuse, such charts and records serve as evidence for the Attorney General's office.

5. The team also reviews office bookkeeping procedures. Because New Jersey Medicaid will not reimburse psychiatrists for unkept appointments, the audit team searches for deviations from this standard when examining a psychiatrist's records. The audit team asks to examine the doctor's appointment books, presumably to assess the time the psychiatrist devotes to each patient.

6. The auditors, according to what our patients have told us, ask questions relating to a) quality of care patients feel they have received, b) how they like their doctor, c) how much time the psychiatrist spends with them, d) whether they were "prepped" for the interview by their doctor, and e) if they have any complaints about the doctor or his or her practice.

7. The team collects any information doctors offer as documentation of the kind of service rendered to their Medicaid patients. We supplied to the audit team copies of an introductory and orientation brochure we give each new Medicaid patient. The brochure explains our practice policies, provides emergency numbers to call when our office is closed, and indicates our "on call" availability. It also attempts to deal with the issue of unkept appointments, a serious problem in a psychiatric practice and, unfortunately, a characteristic pattern for many Medicaid-sponsored patients. We also supplied our weekly statistics of unkept Medicaid appointments to the audit team.

To document our contention that Medicaid patients receive extraordinary amounts of extra session service and time, we asked every member of our staff to keep a log for one month of the time and effort devoted to our Medicaid patients other than face-to-face psychotherapy sessions.

THE PSYCHIATRIST-PATIENT TRUST

It is difficult for us to conceive of meaningful psychotherapy taking place without implicit and explicit guarantees to the patient that confidentiality will be maintained (10, 11). Under the best of circumstances, patients enter psychotherapy with varying degrees of suspiciousness, resistance, and mistrust, based on their earlier life experiences.

Guarantees of confidentiality are crucial to the special trust between patient and psychiatrist that permits the patient to share increasingly intimate and difficult material with the therapist. For the overly sensitive or frankly paranoid patient any hint that confidentiality has been violated will destroy the therapeutic endeavor. So fragile and yet important to psychotherapy is the concept of confidentiality that, in our opinion, merely the idea that someone might have access to intimate material precludes meaningful psychotherapy from taking place.

On the first day of contact with the psychiatrist, the Medicaid patient signs a form permitting the psychiatrist to receive reimbursement for his or her services, which also gives Medicaid permission to examine and request any and all charts.

> Patient Certification. Authorization to release information, and payment request. I certify that the service(s) covered by this claim has been received, and I request that payment be made on my behalf. I authorize any holder of medical or other information about me to release to the Division of Medical Assistance and Health Services or its authorized agents any information needed for this or a related claim.

It is our belief that the nature of the Medicaid form brings greater pressure to bear on the Medicaid patient than on other patients. Middle-class patients do not have to sign such a form, and, in fact, many of them decide not to sign any insurance form because of the issue of confidentiality. Regrettably, they end up paying for psychotherapy on their own.

Medicaid's "Right to Know"

In New Jersey, 600,000 residents receive Medicaid funds, and the state and federal governments spent $429,577,145 for their health care in 1976. Across the nation, 28 million Americans depend on Medicaid for their health care, at a total cost of $14.7 billion. A program encompassing so many people and such large sums of public money may justly argue that it has not only a right but a responsibility to American citizens to monitor the medical care that they pay for. The executive and legislative branches of government expect it and the general public, whose tax dollars pay for the program, demands it.

Violations of Confidentiality

The act of reading psychiatric patient records by the staff of a geographically distant bureaucratic agency makes a sham of any pretense of confidentiality between a psychiatrist and his or her Medicaid patients. The Medicaid team who conducted our audit were not physicians. They were polite, deferential, and tactful

as they went about their business; nevertheless we shared a deep feeling of having been violated and having in turn betrayed our Medicaid patients.

Our sense of outrage was intensified by the knowledge that the selected patient charts were to be copied and transferred to central Medicaid offices in Trenton where they would be reviewed by the "psychiatric consultant." What would ultimately happen to these copies of our patient records was not made clear.

Medicaid's own policy of guaranteeing confidentiality and promising serious penalties to any staff member found guilty of violating such confidentiality is inadequate for the psychiatrist and his or her patient. In our opinion, merely the act of sharing psychiatric material with "benign" strangers is detrimental to the patient-doctor relationship.

In addition to the symbolic impact that Medicaid's "right to know" has upon the therapeutic alliance, there are more realistic and practical issues. We are less concerned with nefarious deeds perpetrated by "Big Brother" governmental agencies (although the Watergate-related Daniel Ellsberg case comes to mind) than with the inadvertent foul-ups that seem likely to occur when any large bureaucratic organization has confidential data at its disposal. The recent disclosure that names of 48,000 New York State women who had undergone abortions had not been deleted from data transmitted to the National Institutes of Health for a research project is a case in point (12).

PATIENT-PSYCHIATRIST RELATIONSHIPS

Psychiatrist and Staff Anxieties

An audit or examination, no matter how routine and from whatever the source, evokes anxiety in the psychiatrist and his or her staff. In an age of increasing government involvement (e.g., potential national health insurance, PSROs, and mental health advocacy), psychiatrists and their physician colleagues should be prepared for the onslaught of those carrying "right to know" passes.

The nightmare of every ethical physician is that a government investigating team, which consciously or unconsciously must justify its existence, will be too rigid and literal in interpreting rules or regulations and will fault doctors for not "dotting their i's or crossing their t's." In the case where an investigation leads to an actual indictment, ultimate acquittal is little consolation to the doctor who in the meantime has suffered irreparable damage to his or her professional reputation, days and months of emotional stress, and income lost from his practice and from legal fees.

The Effect of Patient Interviews

One of the charges leveled against a doctor in our state who was ultimately indicted for Medicaid abuse was that he attempted to "prep" his patients for their interview with the team. The Medicaid team, naive about psychotherapy issues and techniques, might therefore view any pre-interview discussions with our patients as an attempt to influence the patient's honest and frank responses to their questions, so when we were informed of the Medicaid audit, we felt constrained not to discuss it with our Medicaid patients. This violates a basic tenet of psychotherapy: that anything and everything relevant to the patient's psychotherapy can and should be discussed.

One psychiatrist, not in our group, told us that after a Medicaid audit interview patients called to warn the therapist that he was "in trouble." No matter how benign the investigation may be or how nonpejoratively it may be presented to the patient, the therapeutic relationship has been injured and, by innuendo, the psychiatrist's reputation has been harmed.

Furthermore, the type, intensity, and stage of transference reactions between the patient and psychiatrist may be interfered with by the audit interview. Likewise, transference may significantly distort the patient's replies to the investigator's questions; patients' responses may be unrealistically glowing and warm when positive or even erotic transference is occurring and unfairly damaging if the patient is working through issues of negative transference at the time of the audit.

The selection of patients who will be interviewed by the audit team does not, to our knowledge, consider the patient's psychological ability to take part in the interview or how the dialogue might be emotionally harmful to the patient. What is the effect of the audit interview on the patient who is fragile and overly anxious, paranoid, withdrawn, reclusive, passive-aggressive, or sociopathic? How do these characteristics influence and distort the replies that are given, especially to a nonmedical, nonpsychiatric investigator? What is the ultimate effect on the nature and future of the therapy and the patient-psychiatrist relationship?

COMMENT

As America moves closer to a national health insurance program, health planners, politicians, and government officials are gaining experience from the current health payment program for the 28 million Medicaid-sponsored citizens in the United States. The majority of psychiatrists in private practice who do not see Medicaid patients will face similar issues if and when they are blanketed into a national health insurance program serving a more universal population.

The overriding philosophical issue, with many diverse practical ramifications, is whether a government-administered health program, which spends large sums of public tax money and insists that it has the right to know in order to monitor quality of care and insure fiscal integrity, can accommodate the need to maintain a meaningful patient-psychiatrist relationship and confidentiality.

The task of dealing with and reconciling these conflicting needs is awesome.

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Special Issue of *AJP* to Feature Depression and Alcoholism

A special issue of the *American Journal of Psychiatry* will be published in mid-April 1979. This issue, which will be distributed to all subscribers and is in addition to the regular April issue, will consist of a collection of articles on the subjects of alcoholism, depression, or both. Articles will deal with such topics as the treatment of depression with imipramine and ECT; bipolar illness: factors in drinking behavior; physical activity and plasma cyclic AMP in manic-depressive patients; childhood depression; life events at the onset of bipolar affective illness; psychiatric problems in women at alcoholic detoxification; lithium maintenance; efficacy of drugs and psychotherapy in the treatment of acute depressive episodes; and life events, coping, stress, and depressive symptomatology.

Authors include David Avery, M.D., Jesse O. Cavenar, Jr., M.D., David L. Dunner, M.D., Ronald R. Fieve, M.D., Eric W. Fine, M.D., Evelyn L. Goldberg, Sc.D., John Hamm, M.D., Shirley Hill, Ph.D., Eleftherios Lykouras, M.D., Anthony Marsella, Ph.D., Roy Mathew, M.D., Jerome Motto, M.D., Irving Philips, M.D., Timothy Rivinus, M.D., Fred Steinberg, Ph.D., Marc Schuckit, M.D., Robert Belmaker, M.D., Martin Keeler, M.D., Paul Garfinkel, M.D., John Helzer, M.D., James Kocsis, M.D., Myrna Weissman, Ph.D., Alberto DiMascio, Ph.D., Gerald Klerman, M.D., Larry Ereshefsky, Pharm.D., Michael R. O'Leary, Ph.D., Steven J. Wolin, M.D., George J. Warheit, Ph.D., and Walter A. Brown, M.D.

Extra copies of the special issue will be available from the APA Circulation Department at a cost of $3.50 for domestic orders and $4.25 for foreign orders. Payment must be included with order.

CLINICAL AND RESEARCH REPORTS

This section contains 1) new research findings, including preliminary data from pilot studies, either clinical or laboratory; 2) worthwhile replication studies; 3) case reports that describe a truly new syndrome or cast new light on established ones; and 4) case reports that indicate a new therapeutic procedure of potential value or call attention to adverse effects of drugs or previously unreported complications of therapeutic interventions. Program descriptions and literature reviews cannot be printed in this section. Criteria for format are listed in "Information for Contributors" in each issue; papers that do not adhere to these criteria will be returned to the author.

Excretion of Imipramine and Desipramine in Human Breast Milk

BY ROBERT SOVNER, M.D., AND PAUL J. ORSULAK, PH.D.

Little information is available concerning the excretion of psychotropic drugs, including the tricyclic antidepressants, in breast milk (1, 2). Knowles (3) attributed this lack of data to difficulties in doing studies on nursing women, the complexity of the chemical analyses, and a general lack of interest in undertaking them. In this paper we will present a case report documenting the excretion of both imipramine and desipramine in human breast milk.

Case Report

Ms. A, a 32-year-old mother of two children, presented with the complaint, "I'm afraid of getting depressed again." She had had a depression when she was 26, following the birth of her first child, and at that time required imipramine in dosage of 350 mg/day for a therapeutic response. Once in remission, she continued to take imipramine, 200 mg/day, for 6 years until she became pregnant for the second time.

At 1 month postpartum Ms. A began to experience mild depressive symptoms and sought treatment. She also had a history of hypothyroidism and takes levothyroxine, 0.3 mg/day. She was euthyroid at the time of her reevaluation. She was started on imipramine at bedtime and was taking 200 mg h.s. on the 14th treatment day. She had been nursing her child before the initiation of drug therapy and decided to continue doing so after treatment began. Imipramine therapy was initiated with the collaboration of the child's pediatrician, who examined the infant during the time Ms. A was breast feeding and taking imipramine. No physical or behavioral changes were detected by either Ms. A or the pediatrician. However, on the 16th day of treatment, she agreed to discontinue breast feeding. During these first 3 weeks of drug therapy, Ms. A showed no clinically significant response to imipramine.

TABLE 1
Imipramine and Desipramine Levels in Plasma and Breast Milk[a]

Treatment Day	Hours Since Drug Ingestion	Concentrations (ng/ml)	
		Imipramine	Desipramine
Milk			
16	23	18	29
16	1	29	30
17	9	24	35
17	23	4	17
17	1	12	20
18	10	12	26
Plasma			
22	13	21	41

[a]The patient's imipramine dosage was 200 mg h.s. She discontinued breast feeding on day 16.

On the 16th treatment day Ms. A began to collect milk samples for imipramine and desipramine assays. She collected six samples over a 34-hour period and placed them in her freezer until her blood was drawn. Plasma samples for determination of imipramine and desipramine concentrations were obtained on the 22nd treatment day, 13 hours after the last drug dose, from blood drawn into heparinized Venoject vacuum blood collection tubes. Plasma was separated by centrifugation and stored at −10° C until analyzed. Plasma concentrations of imipramine and desipramine were determined by a modification of the method of Cooper and associ-

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ates (4) using a gas chromatograph equipped with a nitrogen detector. Analyses of imipramine and desipramine in breast milk were carried out in a manner identical to that used for the plasma assays. Ms. A gave informed consent to participate in this study.

As shown in table 1, the blood samples revealed antidepressant levels below the therapeutic range of 150–250 ng/ml for imipramine plus desipramine (5, 6). However, quantifiable levels of both imipramine and desipramine were found in the breast milk specimens and the concentrations in breast milk were similar to those found in plasma.

Discussion

In a comprehensive review, Ayd (1) noted only two reports on the excretion of tricyclic antidepressants in human breast milk. In one (7), detectable levels of imipramine were found in the milk of a nursing mother, and concentrations were related to drug dosage. In the other, Knowles (3) reported no detectable drug levels in milk from nursing mothers who had been taking imipramine for up to 5 days. More recently, Ananth (2) reviewed side effects of psychotropic agents excreted in breast milk and concluded that imipramine is present in much lower concentrations in breast milk than in plasma.

Our paper is, to our knowledge, the second report of imipramine excretion in human breast milk and the first to indicate that the demethylated metabolite desipramine is also found in breast milk, both in concentrations similar to those found in plasma.

With respect to other tricyclic antidepressants, Eschenof and Rieder (7) assayed the milk of six normal postpartum women who had received a single oral dose of amitriptyline within 8 days after delivery. Five of these women received 50-mg doses and the sixth received 25 mg. No detectable drug levels could be found in any of the milk samples. Ayd (1), based on results supplied by Eli Lilly & Company, manufacturers of nortriptyline, reported negative findings in serial milk samples from a lactating woman who had been given a single 25-mg oral dose or nortriptyline. We could find no reports concerning the other tricyclic antidepressants, doxepin and protriptyline.

It is most likely that imipramine and desipramine are passively transported into milk along with lipid substances because both drugs are highly lipophilic. This is a common mechanism for the transport of drugs into breast milk (8). Since other tricyclic antidepressants are also lipophilic, they should be transported into breast milk by the same mechanism as imipramine and desipramine. It may be that these drugs appear in milk only after chronic ingestion and that the negative findings for amitriptyline and nortriptyline resulted from the use of single, relatively small drug doses. Knowles' failure (3) to find imipramine may indicate that a period of 5 days of drug ingestion is not sufficient. (Our subject had been taking imipramine for 16 days before milk samples were collected.)

Ms. A's child did not appear to be affected by the ingestion of imipramine and desipramine. However, the levels of these drugs in the mother's plasma were well below therapeutic levels. Since similar concentrations were present in the milk samples, it is possible that even higher concentrations of these drugs may be found in the milk of women who achieve therapeutic levels of imipramine. At these higher concentrations the drugs might have clinically significant effects on nursing infants. In addition, we do not know whether there would have been physiologic or behavioral effects if the infant had been exposed to imipramine for a longer period of time.

Given the current state of knowledge regarding the excretion of tricyclic antidepressants in human milk, it would be prudent to advise nursing women to discontinue breast feeding if they require antidepressant drug therapy. As Rivera-Calimlim (9) noted, even small drug doses may have significant effect on infants because of their greater gastrointestinal permeability (compared to adults) and the immaturity of their drug-metabolizing enzymes, which may result in enhanced drug accumulation.

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Multiple ECT: Morphologic Effects

BY MATTHEW MENKEN, M.D., JAN SAFER, M.D., CHARLES GOLDFARB, M.D., AND ERVIN VARGA, M.D.

Memory loss and other intellectual changes may be a prominent side effect of ECT (1). Since it was introduced into clinical practice in 1937, ECT has been recognized by most psychiatrists as an effective therapy for psychotic depressions, catatonic stupor, and other selected clinical conditions. However, because of the possibility of post-ECT amnestic-confusional syndrome and the introduction of psychotropic drugs, ECT has been used less widely in recent years, undoubtedly due in part to the potential medicolegal exposure. Indeed, certain techniques, such as unilateral nondominant hemisphere ECT, have been employed to reduce memory loss. Because subjective memory complaints may persist after ECT, a literature has developed suggesting that ECT is effective because it produces memory loss, i.e., the treatment works by creating brain damage (2). However, the majority of clinicians have opposed this view of ECT, except to the extent that it is analogous to treatment modalities such as pharmacotherapy, which act by inducing biochemical changes that could be labeled "damage" as a matter of rhetoric.

Computerized tomography (CT) is a new neuroradiologic tool that is an extremely sensitive and noninvasive procedure for investigating many neurologic disorders. Essentially, it measures alterations in tissue density and associated displacement and distortion. It is extemely sensitive in detecting hemorrhage and edema, the two pathologic changes most often ascribed to ECT. The administration of 10 ECTs over 45 minutes to a suicidal patient afforded us the opportunity to perform a CT scan before and after ECT in a clinical situation that would be particularly likely to produce an abnormality if such changes were common after ECT.

Case Report

Ms. A, a 30-year-old woman, was admitted to the hospital on Oct. 26, 1977, with the diagnosis of unipolar depression. She had had recurrent depressions for the past 7 years. She had never received ECT. During her current depressive episode she had received imipramine, amitriptyline, and doxepin sequentially in dosages to a maximum of 300 mg/day without benefit, and she became suicidal. The patient said her father was "always depressed" and her mother had been depressed during menopause. An EEG and neurologic examination were normal. All tricyclic antidepressants were discontinued. On Oct. 27, a CT scan was performed (scan time 2 minutes, 10 seconds).

Apart from sulcal prominence, the CT scan was normal. On the next day, the patient was medicated with sodium theopentol and succinylcholine and given 10 ECTs during a 45-minute period by one of us (C.G.).

Three hours after the multiple ECT, a repeat CT scan was performed. No change was observed when the scan was compared with the earlier study. At this time, the patient was amnestic for events during the day before ECT and disoriented to place and time. She also showed restlessness and confusion that resulted in some motion artifact on the second CT scan. This disturbance of the higher intellectual functions gradually resolved, and on Nov. 9 (12 days after ECT) no confusion or objective impairment of memory was detected. The patient's depression had improved markedly, and she was discharged from the hospital.

Comment

Status epilepticus has been shown in approximately one-third of cases to produce on CT scan the succession of brain edema and subsequent brain atrophy (3). Since the edema and atrophy were unilateral on the CT scan when the clinical seizures were unilateral, it is apparent that the pathologic changes depend on the epileptic process itself rather than the secondary effects of the convulsions, such as an hypoxic/ischemic encephalopathy. Thus, it seems appropriate to infer that if the effectiveness of ECT depended on the production of morphologic changes in the brain, the CT scan would be able to detect such changes.

Thus, the absence of any CT changes following administration of multiple ECT in a 45-minute period to a patient who demonstrated the typical post-ECT amnestic syndrome suggests that the basis of the amnestic-confusional syndrome depends on functional subcellular or synaptic changes, and that ECT does not produce hemorrhages or edema in the brain. It is also appropriate to note that if an autopsy showed prominent sulci, this might be mistakenly ascribed to ECT. The possibility that local alterations in vascular permeability contribute to amnesia and confusion is not excluded by the present study; we thought it was not justifiable to subject this patient to an intravenous injection of iodinated contrast material to measure alterations of cerebral blood flow patterns.

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Lithium in Chronic Schizophrenia

BY GLENN A. GROWE, PH.D., JOHN W. CRAYTON, M.D., DAVID B. KLASS, M.D., HENRY EVANS, M.D., AND MICHAEL STRIZICH

Recent research indicates that in addition to its well-established effect on manic-depressive psychosis, lithium is helpful for patients with a variety of other disorders, including hyperaggressiveness, depression, and epilepsy (1). Still uncertain is lithium's place in the treatment of schizophrenia. Controlled studies of lithium given alone to schizophrenics have generally found it to be ineffective (2, 3). Anecdotal reports suggest that lithium can occasionally lessen hyperactivity and aggression without modifying the core symptoms of schizophrenia (4). That schizophrenic patients react to lithium differently than affectively ill patients is suggested by reports of increased neurotoxicity in lithium-treated schizophrenics. Shopsin and associates (5) found confusion, memory impairment, disorientation, and reduced comprehension in 11 of 17 schizophrenic patients, even though their blood levels of lithium were maintained at about 0.75 mEq/liter. A general clinical worsening accompanied this toxicity. Cohen and Cohen (6) reported irreversible brain damage in excited schizophrenic patients mistakenly diagnosed as manic who received a combination of lithium and haloperidol. On the other hand, Small and associates (7) obtained favorable results by adding lithium to the regular neuroleptic drug regimen of a group of schizophrenics. Ratings of patients on several variables, including a global rating of severity of illness, showed significant improvement. A toxic reaction to lithium was observed in only 1 of 22 subjects. In view of these conflicting data on lithium's effects in schizophrenia and the uncertainty about its role in treating schizophrenia, we studied the effects of combining lithium with neuroleptic medication administered to a group of chronic schizophrenics. Care was taken to monitor blood levels of lithium and to assess both diagnosis and behavioral ratings.

Method

The subjects were eight inpatients (two men, six women) on the Clinical Investigation Unit of the Manteno Mental Health Center. Six subjects fulfilled the Research Diagnostic Criteria standards for the diagnosis of schizophrenia and two met the criteria for schizo-affective schizophrenia (8). The subjects had a median number of three previous admissions and a median age of 25 years. Subjects gave their informed consent to participate in the study. Patients were admitted to the study if they had failed to respond significantly to medication. Either lithium or placebo was added to the treatment regime in a 16-week double-blind placebo-controlled crossover study. Each treatment period lasted 4 weeks. Neuroleptic medications, including haloperidol, chlorpromazine, and trifluoperazine, were maintained at a constant dosage throughout the course of the experiment.

Incorporated in the design were two lithium and two placebo periods; each lithium period alternated with a placebo period. Lithium carbonate was administered in divided doses totalling 900–2400 mg/day in order to maintain serum lithium levels of 0.5–1.0 mEq/liter. Serum lithium determinations were made twice weekly. Patients were also rated biweekly on the eight manifest behavior scales of the Psychotic Inpatient Profile (9). Ratings were done by staff members who were familiar with the patient. The data analysis included 72 observation days for which both behavioral ratings and lithium determinations were available.

Results

Behavior ratings done on patients while their serum lithium levels were within the normal therapeutic range were compared with those done while their levels were subtherapeutic. A repeated-measures analysis of variance for the various subscales of the Psychotic Inpatient Profile revealed significant improvement in only one of the eight scales, Psychotic Excitement, $F(8, 195)=2.66$, $p<.01$. Ratings of patients on this scale averaged 5.99 for subtherapeutic periods compared with 3.77 for therapeutic-range periods. There were trends toward less seclusiveness and reduced retardation on therapeutic-range ratings. No significant changes occurred in the other scales, including Hostile Belligerence, Paranoid Projection, Anxious Depression, Care Needed, and Psychotic Disorganization. None of these patients showed significant lithium toxicity.

Discussion

The results suggest that adding lithium to a neuroleptic regimen in schizophrenic patients produces a decrease in psychotic excitement. This result raises the question about the presumed specificity of lithium for mania (10), since it does appear to have an effect on certain aspects of schizophrenic behavior, and a

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suggests that its effect in schizophrenia may be on one aspect of schizophrenic behavior that is closely related to mania, namely, excitement.

Those scales which tapped behavior more specifically symptomatic of schizophrenia were not affected by lithium. This result is congruent with the anecdotal reports of lithium modifying activity levels but not the primary symptoms in schizophrenia. Lithium may be a useful adjunct to the phenothiazines in the treatment of schizophrenics whose clinical picture includes psychotic excitement.

No significant toxic reactions were seen in the present study. This finding supports the published report (7) that lithium can be combined with neuroleptics and does not pose the increased danger of side effects seen with lithium alone. Further studies may clarify which patient groups are most responsive to a lithium-neuroleptic combination.

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Group Psychotherapy as an Adjunct to Lithium Maintenance

BY SAAD A. SHAKIR, M.D., FRED R. VOLKMAR, M.D., SANDRA BACON, M.S.W., AND ADOLF PFEFFERBAUM, M.D.

Traditionally, patients with bipolar affective illness have been considered poor candidates for group psychotherapy; the presence of a manic-depressive member in a heterogeneous outpatient therapy group has been termed "one of the worst calamities that can befall a therapy group" (1, p. 393). There has been general agreement that the character of manic-depressive patients, including those on lithium prophylaxis, makes psychotherapy difficult, even during the euthymic phase of the illness (2). A substantial number of patients will relapse, even those receiving lithium, and a significant minority of patients will remain chronically ill (3, 4). There have been reports of conjoint family and couples group psychotherapy as an adjunct to lithium maintenance (5, 6). In this article we report our experience with a long-term ongoing psychotherapy group composed exclusively of patients with bipolar affective illness who are receiving maintenance lithium therapy.

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Group Composition and Format

Fifteen patients (13 men and 2 women, mean age=43.3 years, range=19–63 years) have participated in the group. Diagnosis was established by at least two psychiatrists on the basis of history and mental status examination and was supported by psychological testing. All patients had responded acutely to lithium treatment before participating in the group. The average duration of lithium prophylaxis before entry into the group was 21 months.

Three of the members corresponded to the bipolar type II description of Fieve and associates (7), i.e., they had been hospitalized for depression and had a history of hypomania. The remainder were bipolar type I, i.e., they had a history of hospitalizations for mania and mild to severe depressions. Ten patients had histories of poor adherence to lithium maintenance. Prior to their participation in the group patients were evaluated by the two cotherapists (F.R.V. and S.B., or S.A.S. and S.B.), and the general theory and purpose of group psychotherapy and group format were discussed with them.

The group met for 75 minutes weekly and was con-

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ducted primarily with an interpersonal, interactional "here and now" approach as described by Yalom (1). Lithium carbonate was dispensed monthly during the final 30 minutes of the group meeting. Lithium levels were maintained in the range of .8–1.2 mEq/liter. Group members were free to discuss specific problems or concerns regarding lithium treatment at any time. The average duration of group treatment was 51 weeks; the mean number of participants per session was 8.2. Generally, members left the group after obtaining full-time employment but continued to attend the group monthly for support and medication. Three members left the group after about 12 sessions; they were given medication follow-up outside the group.

Several common themes emerged during the course of patients' participation in the group. Initially they were skeptical of both the long-term efficacy of lithium prophylaxis and the benefits of group psychotherapy. This seemed to reflect their previous experience with group therapy and their ambivalence about the loss of a sense of well-being that lithium use sometimes entails (8). Participants typically exhibited massive denial of their problems on entering the group and projected responsibility for the efficacy of lithium maintenance onto the psychiatrist while remaining aloof from other group members. After approximately 10 sessions patients became more identified with the group and more committed to it. During this period members became more interpersonally revealing and actively solicited the advice and support of other members. Common problems included concerns about the chronicity of their illness, the threat of recurrence, societal acceptance, and marital and occupational adjustment. Less time was spent discussing medications and more time in interpersonal learning, instilling hope, and imparting information. If a group member began to exhibit signs of either mania or depression, other members would discuss this openly and provide support and encouragement while medications were adjusted as necessary. Individual members gradually began seeking to return to full-time employment and after doing so would return monthly to the group for medication and support.

Outcome

In the 2-year period prior to participation in the group, 10 of the members had been hospitalized for manic or depressive episodes. The group as a whole spent an average of 16.2 weeks a year in the hospital during this period, with mean lithium levels of .53 mEq/liter. Only 5 members were continuously employed during this period. During the 2 years the group has functioned, 3 of the participants have been rehospitalized for affective episodes. The group has averaged 3.2 weeks of hospitalization a year in this time, with mean lithium levels of .94 mEq/liter. Ten members have now been continuously employed for over 6 months. Participants and their families have indicated a more satisfactory interpersonal and social adjustment.

Discussion

Despite the fact that 10 of the participants had histories of poor adherence to maintenance lithium treatment, the rate of recurrence compares very favorably with those reported in follow-up studies of lithium maintenance treatment (3). As in other studies of lithium maintenance, the most common reason for lithium prophylaxis failure was discontinuation of lithium (8); two of the patients who required rehospitalization had stopped taking their lithium several months after terminating their participation in the group. It is difficult to determine whether the relatively high rate of adherence to lithium maintenance we observed reflects the effects of group therapy per se or the close supervision patients received each week; certainly, the modeling of lithium compliance and effectiveness provided by the occasional attendance of employed group members enhanced lithium compliance.

We have been impressed that, contrary to expectation, our group of manic-depressive outpatients could work therapeutically in a homogeneous group. The shared experience of manic-depressive illness and lithium prophylaxis made the occasional episode of hypomania or depression much less disruptive than would have been the case in a heterogeneous therapy group. The same curative factors at work in other therapy groups were seen in this group (1). Such groups may serve as a simple, efficient, and highly cost-effective adjunct to lithium maintenance treatment, especially for patients with a history of poor compliance, and may provide the benefits of psychotherapy to a group of patients generally viewed as poor candidates for traditional therapeutic modalities.

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Withdrawal Symptoms After Abrupt Cessation of Amitriptyline in an Eight-Year-Old Boy

BY C. THOMAS GUALTIERI, M.D., AND JEANNETTE STAYE

We recently treated a child who seemed to suffer an amitriptyline withdrawal reaction. The patient developed severe gastrointestinal symptoms when amitriptyline was stopped abruptly, and there was no evidence to suggest a toxic or infectious origin for his nausea and vomiting. This case report suggests that a withdrawal syndrome may occur in children treated with amitriptyline when the drug is stopped abruptly.

Case Report

The patient, an 8-year-old boy, was referred to our clinic because of hyperactivity and problems with drug management. He had been treated with methylphenidate from age 2½ (an unusually early age for such treatment) to 6, apparently with some success. When he was 6, he was placed in a highly structured classroom and did well, so the drug was stopped. When the next school year started, however, he began to have the same problems—hyperactivity, distractibility, disruptive behavior, emotional lability, and sleeping difficulties. Methylphenidate was tried again but only increased his symptoms.

In November, amitriptyline was begun. He appeared to do well on 30 mg at bedtime, and serum levels in December showed 30 ng/ml of amitriptyline and 10 ng/ml of nortriptyline. In early spring, a number of new behavior problems arose—he was irritable and seemed depressed—and the sleep problems recurred. The dose of amitriptyline was raised to 50 mg q.h.s., with no apparent response. He was referred to our clinic at this point.

The boy had no severe medical problems, and his development was normal. His IQ was well above average, and he had no specific learning disabilities. His mother had had preeclampsia, but the pregnancy and delivery were otherwise normal. There was a family history of hyperactivity and of alcoholism.

When we first saw the child, he did not seem to be hyperactive or excessively distractible. Results of physical and neurologic examination, including an EEG, were normal. His height was 130 cm (75th centile) and his weight was 24 kg (45th centile).

We wanted to get a baseline and suggested to the child's mother that she stop giving him the medication because of the curious symptom pattern and the equivocal nature of his drug response. The drug was stopped after 7 months of treatment.

Two days later, the child began to have severe nausea, vomiting, and abdominal cramps. He could keep no liquids down, and on Tuesday he was admitted to a community hospital, where he was found to be almost 10% dehydrated, acidotic, and ketonuric. A CBC and a WBC count were normal. He did not have a fever, and there were no similar illnesses at that time among his family, neighbors, friends, or peers. He was treated with intravenuous fluids and electrolytes and discharged 2 days later. For 2 weeks after his discharge, he vomited whenever he ate solid foods.

He has done well since that time, and has had no recurrence of gastrointestinal problems. His behavior problems have not returned, and he is currently doing well in a third-grade class with no medications.

Discussion

Of all the tricyclic antidepressants, only imipramine is approved for use in children for the temporary relief of symptoms of nocturnal enuresis (1). However, over the years, imipramine, amitriptyline, other tricyclics, and even the monoamine oxidase inhibitors have been used in children for a variety of problems, ranging from hyperkinesis to petit mal seizures (1). There is no evidence that imipramine, the most commonly used tricyclic among children, is superior to stimulant drugs in the treatment of hyperkinesis (2). There are few studies on the use of amitriptyline in childhood disorders (1). Yepes and associates (3) found that the drug was comparable to methylphenidate among "behaviorally disordered" children in some of its effects but tended to be sedating.

Nausea and vomiting have been described in connection with withdrawal from neuroleptic drugs (4–6). Children appear to be particularly sensitive to the effects of neuroleptic withdrawal, and a syndrome characterized by gastrointestinal symptoms, peculiar movements, agitation, and autonomic disturbances has been described (7).

Withdrawal symptoms, including gastrointestinal complaints, have been described among patients treated with imipramine (8, 9). To our knowledge, however, an amitriptyline withdrawal syndrome similar to the case described above has never been reported, and tricyclic withdrawal symptoms have not been described in the pediatric age group.

It is a common belief among clinicians that the abrupt discontinuation of any strongly anticholinergic drug may provoke withdrawal symptoms. Amitriptyline is the most strongly anticholinergic of all the tricyclics (10), and it seems likely that this child's severe reaction was induced by the abrupt withdrawal of an anticholinergic influence.

Although this case report does not make an ironclad case for the existence of an amitriptyline withdrawal syndrome in children, it does suggest certain cautions. First, amitriptyline should not be used in the

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treatment of hyperkinesis when other more familiar drugs are available. Second, children who are treated with amitriptyline probably should not be abruptly withdrawn from the drug.

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Chronic Amitriptyline Toxicity

BY EARL L. GILLER, JR., M.D., PH.D., DONALD S. BIALOS, M.D., JOHN P. DOCHERTY, M.D., PETER JATLOW, M.D., AND LAURIE HARKNESS, M.S.W.

Recent work has demonstrated that the therapeutic response of depressed patients to amitriptyline occurs at total tricyclic plasma concentrations within a range of approximately 75-200 ng/ml (1). Severe, acute overdosage, with total tricyclic plasma levels greater than 1000 ng/ml, is generally recognizable by such clinical signs as decreased consciousness, marked anticholinergic manifestations (tachycardia, mydriasis, blurred vision, dry mouth, urinary retention, decreased bowel activity, agitation) (2), and ECG changes (prolonged PR and QRS intervals, diffuse ST-T wave changes), with QRS widening being the best indicator of high drug plasma levels (3).

Chronic excessive levels, however, may be associated with milder toxic symptoms that are difficult to distinguish from depressive symptoms. Plasma nortriptyline levels over 140 ng/ml are associated with poor antidepressant response (4) that may be confused with undermedication. Thus the clinical state of patients with chronic total antidepressant plasma levels between 200 and 1000 ng/ml can help to clarify when toxic effects begin and what those effects are. This report describes a patient with total antidepressant levels of over 700 ng/ml who had received a daily dose of 250 mg of amitriptyline for 3 years and who, in retrospect, appears to have shown specific symptoms of toxicity.

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Case Report

Mr. A., a 54-year-old married man, had been hospitalized six times since 1943 for depressive episodes and suicide attempts. He became alcoholic in 1955, and a liver biopsy in 1962 showed Lannec's cirrhosis. In 1973 he was hospitalized for depression and treated with 150 mg/day of amitriptyline for 4 months. He then took an overdose and was thought to have a subendocardial infarction with enzyme and ECG changes (QRS widening, diffuse ST and T abnormalities). Subsequent ECGs were normal. After discharge, he was restarted on amitriptyline, which was increased to 250 mg/day in 1975 for continued feelings of depression, anergia, and anxiety. This dose was continued until he began to participate in an antidepressant withdrawal study in June 1978. On baseline evaluation he was found to be mildly confused, agitated, and nervous. Anticholinergic symptoms of dry mouth, blurred vision, constipation, and urinary hesitancy were mild to moderate. One week later, after his daily dose had been tapered to 175 mg, he was more coherent and less nervous but coincidentally was hospitalized and treated surgically for a perforated duodenal ulcer. Amitriptyline was stopped on hospitalization. An ECG showed QRS and PR interval increases and diffuse ST and T changes. Steady-state baseline plasma antidepressant levels drawn 1 and 2 weeks earlier and assayed (5) at this time showed total levels of 744 ng/ml (amitriptyline=492, nortriptyline=252) and 720 ng/ml (amitriptyline=443, nortriptyline=277). At discharge, he was not confused and was more relaxed, communicative, and cheerful than he had been in years. When questioned at discharge, Mr. A denied excessive drug ingestion. He subsequently became depressed and was again started on 50 mg of amitriptyline, which he independently increased to 150

mg/day. After 1 month, plasma levels at this dose were 294 ng/ml of amitriptyline and 91 ng/ml of nortriptyline. His dose was reduced.

Discussion

This patient had chronically toxic plasma levels of amitriptyline and nortriptyline, possibly as a result of impaired liver metabolism of the drug secondary to his cirrhosis. The almost identical drug concentrations obtained 1 week apart and the very high nortriptyline levels are more compatible with steady-state excessive levels than acute overdose (6). Equivalently high levels were seen after the patient was given a lower daily dose. The assessment of his myocardial infarction in 1973 was, in retrospect, complicated by amitriptyline treatment, since the ECG changes were similar to those seen with high antidepressant plasma levels. Similar ECG changes were seen in 1978 when Mr. A had greatly elevated antidepressant plasma levels. His mild confusion and agitation are consistent with the more severe symptoms of agitation, restlessness, and even delirium seen with higher plasma levels (1).

When tricyclic antidepressants were introduced into clinical use, insufficient dosage was the most common cause of poor medication response. Clinicians learned to increase the upper limit for daily dosages from 150 mg to 300 mg. This case illustrates the problem that, as in neuroleptic treatment (7), symptoms resulting from excessive drug blood levels may be interpreted as reflecting insufficient levels. This dose is then increased instead of decreased, which causes further toxicity. Thus patients taking tricyclic antidepressants who have impaired drug clearance, including geriatric patients (8) and alcoholics, and who show signs of confusion, agitation, or nervousness, even without prominent anticholinergic symptoms, should have plasma drug level determinations. If this is not possible, a trial of dosage reduction should be considered.

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Trihexyphenidyl Abuse for Euphorigenic Effect

BY DANIEL A. GOGGIN, M.D., AND GEORGE F. SOLOMON, M.D.

Synthetic anticholinergics are used to treat Parkinson's disease and extrapyramidal side effects of psychotropic medication. The clinical and pharmacologic literature contains references to various responses to anticholinergic CNS stimulation, particularly toxic psychosis. However, there is little information about the affective aspects of CNS responses to anticholinergics. We will describe the case of a patient who abused trihexyphenidyl for a prolonged period to obtain a euphorigenic effect.

Case Report

Mr. A, a 40-year-old chemist with administrative and scientific duties, was seen initially with the chief complaint, "I think I'm going to pieces." He had become seriously depressed as a result of his suspicions, which had been intensifying for 2 years, that his wife was having an affair. He stated that his wife had done nothing to validate his fears. Five days after she left for a vacation with relatives the obsessive thoughts had become so anxiety-provoking that Mr. A had not eaten or slept, and he sought treatment. He reported a weight loss of 4.6–5.4 kg in the past year, decreased interest in routine activities, intermittent and terminal sleep problems, and recent onset of feelings of futility about life. He had no known allergies or history of drug abuse.

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The initial diagnosis was depressive neurosis of moderate severity with psychotic trends and obsessive character. He was started on thiothixene, 5 mg t.i.d., and began long-term psychotherapy. The choice of thiothixene was based on clinical reports of its efficacy in treating depression associated with anxiety, agitation, and obsessive features (1–3). Two days later he was given trihexyphenidyl (Artane), 2 mg t.i.d., to treat symptoms of akathisia. The trihexyphenidyl dosage was increased to 4 mg. t.i.d. 2 weeks later for recurring akathisia, which resolved within 72 hours. After 2 months, his anxiety and ruminations had decreased and his sleep pattern had improved, so his dosages were reduced to 5 mg b.i.d. of both thiothixene and trihexyphenidyl. After he had been stabilized in treatment for 4 months, he was given a prescription for a 1-month supply with two refills of both medications. He became more despondent in the ensuing month but withheld this information and treated himself, first with increased doses of thiothixene and then with 35–40 mg of trihexyphenidyl daily. He told the therapist what he was doing after he depleted his prescription. He felt that the trihexyphenidyl had a definite antidepressant effect that thiothixene lacked. He denied adverse side effects or other drug abuse during that period. Hospitalization was considered, but Mr. A responded to doxepin, up to 250 mg/day, and to intensive therapy.

His depressive symptoms diminished during the next 3 months, and the doxepin dosage was gradually reduced to 100 mg/day. However, shortly after this period he synthesized trihexyphenidyl and ingested approximately 25 mg of the chemically impure compound over a 6-hour period. He experienced acute tachycardia, nausea, weakness, restlessness, and faintness, which led to a brief hospitalization (72 hours). He denied other drug abuse. He stated that he wanted the trihexyphenidyl because "I was just tired of looking at people who appeared to not be depressed, and I wanted to feel good that way again." Subsequently, doxepin was stopped and amitriptyline, 75 mg h.s., was started. After several sessions Mr. A stopped therapy, but he continued to take amitriptyline for 6 months. When he was seen in follow-up a month later, he felt recovered from his depression and denied further abuse of trihexyphenidyl "because of the possible consequences to my health and job."

In one session, Mr. A described his subjective feelings during his first instance of trihexyphenidyl abuse as follows: "I really felt so bad, I felt nothing was worthwhile. Everything was very bad, dark, I wanted to crawl in a hole. I took two Navane [5 mg] capsules. They didn't do anything for me; I just kept sinking. Then I took two Artane [5 mg]. That brought me up to feel okay or normal. After the response I could think about my problem, know I had it, but I didn't feel depressed about it. But I was extremely depressed before I took them [Artane] . . . sick."

Mood-Altering Effects of Other Anticholinergics

It has been our experience that jail inmates hoard and trade trihexyphenidyl, which they use to get high, but do not generally abuse benztropine. However, a colleague related two cases of benztropine abuse for mood-altering effect, in one case to relieve anxiety and in the other to obtain a high (S. Castro, M.D., personal communication). Jellinek (4) reported on eight individuals who rejected discontinuation of trihexyphenidyl and biperiden, prescribed for dyskinetic side effects of phenothiazines, because they "did not feel as well" without the drugs, which had an energizing, mood-elevating effect. Another colleague recently noted that five of his patients had taken 3–4 times the prescribed dosage of biperiden to obtain a euphorigenic effect (W. Bernstein, M.D., personal communication). Fink and associates suggested in one study that a chlorpromazine-procyclidine regimen might more effectively treat depression than chlorpromazine alone (5).

Discussion

MacVicar (6) and Rubinstein (7, 8) have emphasized the abuse potential of anticholinergics, particularly by patients with histories of multiple drug abuse, and have cautioned against excessive or lax prescription of these drugs. The use of an anticholinergic specifically to relieve a depressed mood is minimally documented. Bolin (9) reported that a 32-year-old woman was treated for spastic torticollis with trihexyphenidyl, which induced a sense of well-being that counteracted depressive feelings. She took higher doses (eventually reaching 24–30 mg/day) as life stresses accumulated, and developed a toxic psychosis.

In the case we have described the patient clearly abused trihexyphenidyl to alleviate his depressed state and to "feel good" again. He discriminated between his responses to trihexyphenidyl and to thiothixene. His sustained use of trihexyphenidyl without intolerable side effects, in daily doses that usually result in toxic psychosis, implies individual differences in CNS response to anticholinergics and indicates the need for further research on the role of cholinergic pathways in affective states.

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LETTERS TO THE EDITOR

This section contains reactions to Journal *articles, statements of opinion, comments on Association activities, requests for information, etc. Letters should be submitted in duplicate to the Editor, who makes all decisions regarding publication. Letters must be typed double spaced throughout and should not contain more than 500 words and 5 pertinent references. Criticisms of published articles will automatically be sent to the author(s) for response. Letters will be edited for clarity and conformance with* Journal *style. We regret that we cannot inform writers of the disposition of letters to return those which are not printed.*

Sleep Architecture in Psychotropic-Induced Somnambulism

SIR: In "Filicide During Psychotropic-Induced Somnambulism: A Case Report" (November 1978 issue) Daniel J. Luchins, M.D., and colleagues stated that they were unaware of reports documenting frequent movement and awakening from stage 4 sleep in patients with somnambulism or the onset of somnambulism after at least one REM period.

In fact, there have been papers which address both of these issues. Broughton (1), in his classic paper on somnambulism, night terrors, and enuresis as disorders of arousal, cited four references which demonstrated that sleepwalkers exhibit many movements during stage 4 sleep independent of the somnambulism itself. Additionally, Fisher and associates (2) reported that patients with night terrors (also a stage 4 phenomenon) experienced significantly more awakenings from stages 3 and 4 sleep than a control group. Indeed, Broughton hypothesized that it is a constellation of physiological factors, of which increased awakening from stage 4 sleep is a part, that predisposes a patient to a particular disorder of arousal such as somnambulism or night terrors.

Several authors have also noted that it is not uncommon for episodes of somnambulism and night terrors to occur after the first REM period (2–4). The finding in thioridazine-induced somnambulism that the episodes occurred after at least one REM period had elapsed may be related to that drug's ability to increase stage 4 sleep. This increase probably occurs after the first REM period when drug plasma levels begin to rise following the evening dose. One could postulate that in patients who experience neuroleptic-induced somnambulism the drugs induce a physiological state during stage 4 sleep similar to that in patients who have endogenous somnambulism. Therefore, it would be of interest to know if their patient's sleep architecture, especially in regard to movement and awakenings from stage 4 sleep, was different on the nights she walked in her sleep from the nights when she did not.

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Dr. Luchins and Associates Reply

SIR: We found interesting Dr. Charney's suggestion that thioridazine might induce a physiological state in stage 4 sleep (increased movements and awakening) similar to what is found in "endogenous" somnambulism. However, we do not believe it applies to our patient. She showed frequent movement and awakening from stage 4 sleep when she was both on and off medication. In fact, there were nights when she was not taking medication and would sit up in bed during stage 4 sleep. On nights when she was taking medication and did not sleepwalk, there was no increase in movements or awakening as compared to nights without medication. It seems to us that the thioridazine (particularly in combination with chloral hydrate) "disinhibited" a mechanism that normally suppressed full-blown somnambulism in a predisposed individual.

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Family Planning for the Mentally Ill

SIR: The excellent article by Ming T. Tsuang, M.D., Ph.D., "Genetic Counseling for Psychiatric Patients and Their Families" (December 1978 issue), makes it appropriate to reiterate the urgent need of mentally ill patients and their spouses for family planning services and that this is a responsibility of the psychiatric profession.

The birth rate among mentally ill women has risen substantially within the past two decades. By virtue of their social and psychological problems, these women have difficulty obtaining and utilizing family planning services. This fact, coupled with growing use of community and child-care services, coincides with a more general concern for popu-

lation control and family planning programs. Because pregnancy, child-birth, and child-care may contribute to recurrences of psychotic or depressive episodes, and because the children of mental patients are often unwanted and tend to be disadvantaged psychologically, socially, and educationally, this sector of the population needs adequate family planning services.

Both the need and desire for such services have been documented by the patients themselves. Lack of knowledge about and access to services, fear and ignorance about methods, and poor communication with sexual partners and therapists prevent these women from getting the care they need and want. When asked if they would like such services offered in conjunction with hospital care, the hospitalized psychiatric patients we surveyed unanimously answered yes (1). The prevalence of unplanned and unwanted pregnancies and the concomitant emotional distress further document this need (2). If the pregnancy is carried to term, the unwanted child is often blamed for his or her mother's distress and is consequently deprived of maternal care and attention. Since the child may become a ward of the state or be adopted, he is likely to incorporate the blame and perceive himself as worthless. In addition, the associated experiential and potential genetic deficits may, as Dr. Tsuang has documented, decrease the child's potential for normal development.

There are three major arguments favoring the integration of family planning services with other mental health services. First, the mental patient's welfare requires an amount of time and special attention that standard community family planning programs cannot provide in order to overcome the psychological and informational barriers to effective contraception. Second, the potential handicaps of an unwanted pregnancy—which would affect the child as well as his family—make it ethically necessary to inform these women about family planning and allow them to opt for an alternative. Third, this type of planning is an integral part of primary prevention in mental illness and thus belongs in every mental health care setting throughout the country. Deinstitutionalization and community maintenance make it imperative to provide screening, counseling, follow-up, and any additional services to ensure adequate education and use of contraceptives for mentally ill women.

Over the past five years, in collaboration with both the national and local Offices of Family Planning, Department of Health, Education, and Welfare, we have developed family planning services in three state mental hospitals and one community general hospital that has a psychiatric ward and have provided consultation to two private hospitals with psychiatric wards as well as to a community mental health guidance center. The patients and staff welcomed these services, which were delivered in a variety of psychiatric settings. Follow-up studies have demonstrated that a significant proportion of these patients continued using contraceptives after leaving the hospital.

I have attempted to point out the need of psychiatric patients not only for genetic counseling but for family planning services as well. Based on our experience, these services can be effectively delivered in the psychiatric hospital. We wish to express our willingness to consult with any psychiatric hospital in the nation that may wish to provide these services as part of every woman's right to good medical care.

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HENRY GRUNEBAUM, M.D.
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Imipramine and Delusional Depressions

SIR: In "Imipramine Response in Deluded Depressive Patients" (July 1978 issue) Frederic Quitkin, M.D., and associates reported that delusional depressed patients respond to imipramine as well as depressed patients without delusions, which contradicts our observation that delusional depressed patients do not respond to imipramine (1). We feel there are major differences in the way the data were collected and in the populations studied that preclude any meaningful comparisons between these two studies.

Dr. Quitkin and associates' retrospective analysis used scale scores from the LORR multidimensional features, and the authors noted that psychotic features were generally "slight to mild" and that "delusions and hallucinations were not prominent features of the symptoms." In our study, a clinical evaluation was used to determine whether the patient was delusional, and patients about whom there was uncertainty were excluded. In marked contrast to the Quitkin and associates study, our patients' delusions were of primary importance and were often their chief complaint.

Dr. Quitkin and associates stated that we did not present the criteria we used to define a delusional depression. In fact, that definition was included in our original report and elaborated in a subsequent paper that dealt with the history of the delusional concept in depression (2). It has been our impression that profound feelings of guilt, worthlessness, and pessimism are common in severe depressions and that even when held with a quasi-delusional conviction they do not constitute delusional ideation. We considered a patient who felt his life had been wasted (with abundant evidence to the contrary) as nondelusional. However, a patient who thought he had committed a crime and was convinced he should be punished was considered delusional, as was a woman who was convinced the FBI wanted to deport her because she was not worthy of citizenship. We are perplexed as to how one defines a "slight to mild" delusion. Certainly our population was not mildly delusional.

There are other significant differences between the two patient groups. In the 60 patients we studied (3), 28% were delusional by our criteria; Dr. Quitkin and associates found 75% of their 36 patients delusional. The mean age of their patients was 38, whereas the mean age of our 13 delusional patients was 61 years. Before participating in the study, their patients "had failed to respond to nonsomatic hospital treatments," presumably a prolonged period of hospitalization. In contrast, our delusional patients were so sick that an active drug trial of more than 4 weeks could not be justified. Thus, our patients would seem to have been more severely ill, older, and, most importantly, more clearly delusional than theirs. These are large and, we think, clinically significant differences. Dr. Quitkin and associates suggested that delusional patients may have required higher doses. Of our 13 patients, the 2 who responded had plasma levels of 50 and 100 ng/ml, respectively, and the 11 nonresponders had a mean level of 208 ng/ml, with a range of 65–535 ng/ml. Among nondelusional patients we have found approximately

200 ng/ml to be a therapeutic antidepressant plasma level. Thus we do not feel that nonresponsiveness among the delusional depressives was related to an inadequate concentration of drugs.

We think that because of major differences in the patient population studied, the ways in which data were collected, and the criteria used for defining delusional patients, the results obtained by Dr. Quitkin and associates cannot be compared with our data. However, it is also important to note that both of these studies are retrospective observations and involve only imipramine. As a result they are limited, and both suffer the pitfalls so common to retrospective observation. We continue to think that delusional patients do poorly on imipramine and very well with ECT. We do not know that other treatments do not work—we simply have no information. Delusional depressions are a regular part of clinical practice, and we heartily agree with Dr. Quitkin and associates that this issue deserves a careful prospective evaluation.

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ALEXANDER H. GLASSMAN, M.D.
MICHAEL SHOSTAK, M.D.
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Dr. Quitkin and Associates Reply

SIR: It may very well be that our results are different from those of Dr. Glassman and associates because of different characteristics of our samples. However, there remains the possibility that if they had used a higher dose their results would have resembled ours. The question is, which is the better predictor of therapeutic effect—blood level or daily dose? One would, of course, like more data, but an appraisal of the present literature, which includes two double-blind prospective controlled studies showing that 300 mg/day was superior to 150 mg/day (1, 2), leads us to believe that a trial of imipramine should not be considered adequate unless a dosage of 300 mg/day has been reached, and we believe such a dose should be tried unless adverse reactions supravene.

In the study by Glassman and associates (3) a fixed dose of 3.5 mg/kg per day was used, which, given the usual weight of women, would result in a dose of approximately 200 mg/day.

We all agree that this important question should be resolved by future research. We have, in the course of a study of desipramine, 300 mg/day, in depression, treated four patients with prominent delusions who met the criteria of psychotic depression of the Research Diagnostic Criteria (4). Three had a marked clinical improvement with remissions of both delusions and depression; the fourth refused to continue in the study.

This anecdotal experience adds to our conviction that it is premature to conclude that psychotic depressives should be treated with ECT without a trial of tricyclic antidepressants first.

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Successful Treatment of Hypertension—Compliance or Marital Therapy?

SIR: The title of the paper by Frank Summers, Ph.D., "Severe Hypertension Treated Successfully by Marital Psychotherapy" (August 1978 issue) was provocative but misleading. The patient described in the paper was treated not only by marital psychotherapy but by large doses of five effective antihypertensive drugs. Although psychological factors probably play a role in the etiology of hypertension (1), I doubt that the decreased stress and tension perceived by the patient caused the blood-pressure-lowering effect of psychotherapy. A much more likely explanation is that the patient did not respond initially to medication because she did not take it as prescribed. Then psychotherapy decreased blood pressure by improving the patient's compliance.

Inadequate compliance of patients with the therapeutic regimen is the most important factor in hypertension treatment (2). The compliance decreases as the number of pills prescribed increases, and it is very doubtful that the patient was taking all of her 17 daily pills. Dr. Summers describes the patient as deeply depressed, and this could also contribute to low compliance. Even without suicidal tendencies, a depressed patient is hardly concerned about his or her physical well-being and therefore is not motivated to take medication. Finally, large doses of antihypertensive medications usually cause some side effects. The lack of these in this case also indicates poor compliance.

Compliance of hypertensive patients is not affected by increased knowledge about the disease, but it is improved by a behaviorally oriented approach (3). Summers' paper suggests that psychotherapy might be an effective strategy to improve blood pressure control in a noncomplying patient.

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LADISLAV VOLICER, M.D., PH.D.
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Dr. Summers Replies

SIR: I disagree with Dr. Volicer's contention that my paper was misleading. The fact that he did not cite any misleading statements in my paper supports this objection. The body of his letter attempted to defend his speculation that my patient was helped by a change in her medication compliance habits rather than a reduction in tension brought about by psychotherapy. This implies that I had proposed the latter as her route to improvement. Of course I made no such statement. The limitations imposed by the Clinical and Research Reports section left me no space to speculate about the vehicle of my patient's rather dramatic change. I reported the facts as I saw them, and the fact is that the reduction in her blood pressure began after a particularly poignant marital therapy session. Had I not reported this sequence, I would have been guilty of misleading my readers.

I do agree that Dr. Volicer's speculation regarding the means by which psychotherapy helped effect a lowering of the patient's blood pressure is plausible. But I also entertain the hypothesis of decreased stress, especially since the patient's relief was quite apparent. However, I simply do not have the data to make a definitive judgment. The patient did not discuss medication compliance during our sessions, nor did she talk about it with her physician. Dr. Volicer's certainty about the implications of my paper seems unwarranted. Had I had the space, I would have presented alternative speculations of possible reasons for the effects of psychotherapy on the patient's reduced blood pressure; any statement in this regard is speculation.

FRANK SUMMERS, PH.D.
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More on Trimipramine and Alcohol

SIR: The issue of possible idiosyncratic response to trimipramine in middle-aged men suffering from intermittent bipolar affective disorder compounded by dipsomania was discussed in my letter, "Trimipramine and Relapses of Symptomatic Drinking," and Dr. Akiskal's reply in the October 1978 issue of the *Journal*.

Trimipramine enjoys an exceptional position among the tricyclic antidepressants, having an anxiolytic effect second only to chlorprothixene (1) and failing to suppress REM sleep (2), as most compounds of the group do. Moreover, it is the only tricyclic antidepressant not explicitly contraindicated in combination with MAO inhibitors (3).

These oddities of trimipramine could perhaps explain in some way the fact that one of the patients I described in my previous letter was able to stabilize emotionally and reduce considerably his habitual ingestion of alcohol on 150 mg/day of amitriptyline, whereas trimipramine seems to have led to exceptionally heavy drinking that culminated in a predelirious condition.

Another alcoholic patient, aged 35, who was seemingly dependent on thioridazine, diazepam, and methyprylon and had a long history of impulsive behavior and pronounced mood swings, developed a typical picture of delirium tremens within a few days of introduction of trimipramine, 100 mg h.s.

A 52-year-old salesman who had recently separated from his wife was diagnosed as suffering from a depressive reaction against a background of cyclothymic personality. He failed to improve with various combinations of antidepressants, minor tranquilizers, and hypnotics for several months. He was then given trimipramine, 50 mg t.i.d., which led to his being practically recovered within about 2 months. About 6 months later he required urgent attention for the first time in his life for a bout of heavy drinking of over 2 weeks' duration that was accompanied by reckless spending and extensive traveling, a definite flight of ideas, and suicidal impulses. He had been taking 200 mg/day of trimipramine for about 6 weeks upon the prescription of another physician for a well-documented depressive relapse.

All these cases would favor the possibility of trimipramine triggering a switch to hypomania; this is warned against by the manufacturer (4) as manifested chiefly by an excessive use of ethanol.

This does not seem to be a problem unless there is a background of intermittent bipolar affective disorder. I have followed during three consecutive episodes of agitated depression, compounded by heavy drinking and occurring apparently around the anniversary of her husband's death, a 62-year-old woman who had become helpless and lonely after she was widowed. Each time she recovered entirely within a fortnight of the introduction of trimipramine at the dose of 75–100 mg h.s. She had no history of periodic swings of mood and activity. Is the difference in gender related to the opposite, beneficial response to trimipramine in regard to excessive drinking in this case?

Another patient's case seems to militate against such a conclusion. An anxious 57-year-old man with a 25-year history of alcohol abuse who was prone to depressive spells had been abstinent for 6 years. He was seen in a state of agitated depression and was given clomipramine, 100 mg/day and then amitriptyline, 50–150 mg, with no improvement. Within 10 days he improved on 200 mg/day of trimipramine and remained well for the next 5 months of follow-up care, with maintenance doses of approximately 75–100 mg/day. He has never returned to drinking.

Although the number of these clinical observations is insufficient to establish as a well-founded fact this particular untoward response to trimipramine in a subgroup of bipolar affective patients, the subject seems to merit further investigation. Discounting the above incidents as merely returns to pretreatment (drinking) behavior would not do justice to the issue.

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More on Bioequivalence of Tricyclics

SIR: The case report by Robert B. Ostroff, M.D., and John P. Docherty, M.D., on "Tricyclics, Bioequivalence, and Clinical Response" (December 1978 issue) prompts me to

share recent experiences in which four outpatients had substitutions of tricyclic antidepressant by the hospital pharmacy.

The patients, two men and two women ages 38–54, had been taking one brand of amitriptyline in dosages ranging from 100 to 450 mg/day over 6 months for depression. Each patient participates in supportive psychotherapy and is seen for one hour every two to four weeks; there have been no recent changes in scheduling. All four patients had experienced an excellent clinical response, with improved sleep pattern, appetite, energy, pleasure, and sense of well-being.

I routinely prescribe no more than a 4-week supply of medication, and all of these patients happened to obtain refills during a 3-week period after the hospital pharmacy began to substitute another brand of amitriptyline. (Illinois law allows pharmacists to substitute generic drugs or other name brands unless the physician indicates "no substitution" on the prescription.) Each patient called me approximately 10 days after the substitution and complained of feeling worse, particularly in terms of decreased energy and disturbed sleeping patterns. Interestingly, three patients noted that the pharmacist had discussed the substitution and reassured them; they wondered aloud, "Is my mind playing a trick on me with the new colored pill, although I definitely do feel worse?" I initially reassured the patients regarding the substitution, but their discomfort persisted and I offered them new prescriptions for the original brand, marked "no substitutions." I have started to do this with other patients for whom I prescribe tricyclic antidepressants. This experience led me to wonder if there were bioequivalence differences among the tricyclic antidepressants, as is the case with digitalis.

Dr. Ostroff's report and a recent letter by Ayd (1) add confirming emphasis to the possibility of important bioequivalence differences. Physicians prescribing antidepressant tricyclics should indicate a brand name with which they are experienced and mark "no substitutions" on the prescription until this problem is better researched.

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Double Standards or Different Standards?

SIR: In his letter, "Editorial Double Standards?" (December 1978 issue), Michael A. Taylor, M.D. unfairly attacked *Journal* editors for using what he described as a double standard in accepting manuscripts. I do not believe he has proven a double standard at all, and I wish the Editor had not accepted culpability so quickly.

Dr. Taylor's implicit premise is that journals should accept only one type of manuscript, which I shall label statistic-based. This is the type Dr. Taylor prefers. Writers of this sort of article should maintain a certain standard by using control groups and unbiased raters. Dr. Taylor complains that his articles must live up to this standard, while Dr. Arieti's do not.

Why must a journal editor subscribe to Dr. Taylor's notion that all manuscripts be of the statistic-based type? An editor may believe there is a place for articles of a purely descriptive, theoretical, or even a musing-humorous nature. If the editor sets a high, but different, standard for each type of article, would Dr. Taylor deny him this prerogative?

If an editor does what I have just described, could one say (as Dr. Taylor does) that the editor has a double (or triple, or quadruple) standard—or that he has two to four categories of manuscripts he accepts, with a single high standard of excellence for each? The latter seems a more accurate statement.

In setting the standard for descriptive articles, criteria worth considering are the number of patients the observer has worked with intensively over a period of time (i.e., his experience); the perceptiveness of his observations (best demonstrated by his prior writings or lectures); and his skills at exposition.

I do not wish to imply that description is nonscientific. The scientific method begins with observation, and in psychiatry this means a careful, accurate observation of clinical phenomena in patients one knows rather well. (Observing the patient and being certain of one's interpretations are vastly underrated abilities, achieved only by a skilled clinician.) Such work generates many of the basic data that are later correlated, chi-squared, and generally chewed on by statistic-minded psychiatrists. This is not intended pejoratively, because I recognize their work as comprising a distinct part of the total scientific enquiry.

But the scientific method does not begin with collected data; it begins with a first-hand accurate observation of reality. Hypotheses may then be formed. Once formulated, they will eventually be subjected to verification by skilled clinicians like Dr. Arieti, who apply the hypotheses to patients they have worked with firsthand. Interpreting the results accurately depends upon correct observations and correct controls and analysis.

There are several types of scientific enquiry, each involving different phases, and I think a psychiatric journal should encompass them all. I do not see any double standard, merely single standards applied to several categories of manuscripts.

ARTHUR S. MODE, M.D.
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A Quotation Problem

SIR: In the article "Adult Minimal Brain Dysfunction and Schizophrenia: A Case Report" (December 1978 issue), Leighton Y. Huey, M.D., and associates quoted our work (1) incorrectly. We did not say that imipramine is helpful in schizophrenics characterized by "soft neurologic signs." In fact, the subgroup of schizophrenics who are most prone to soft neurologic signs are those who have very poor premorbid sociality and are not only poor responders to antipsychotics but are most likely to do poorly when given tricyclic antidepressants, i.e., to develop an exacerbation of their psychotic symptoms (2, 3).

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Propranolol in Breast Milk

SIR: In his article "Side Effects in the Neonate from Psychotropic Agents Excreted Through Breast-Feeding" (July 1978 issue), Jambur Ananth, M.D., M.B.B.S., D.P.M., stated that one paper (1) indicated propranolol is excreted freely in breast milk and the actual plasma-to-milk ratio is greater than 1:1.

I believe Dr. Ananth's interpretation of the propranolol data is incorrect. Two papers have reported propranolol levels in human milk (1, 2). The maximum milk level reported was about 120 ng/ml three hours after a maternal dose of 160 mg p.o. (1). The second paper contained a laboratory reporting error, which when corrected (3) indicated a maximum milk level of 30 ng/ml two hours after a maternal dose of 40 mg p.o. The two papers are therefore in agreement. The second paper measured sequential plasma and milk levels in one patient. Maximum milk levels were only about half of peak plasma levels. If milk levels do exceed plasma levels, it is unlikely that they exceed them by very much or for longer than a brief period (4).

One can estimate an infant's theoretic maximum drug intake by assuming that an infant takes in 165 ml/kg of milk per day (4). If the infant nurses at the time of peak propranolol levels (which is unlikely) and the mother is taking the rather high dose of 160 mg, the infant would receive only 20 μg/kg per day. This is about three orders of magnitude less than the reported therapeutic dose in infants of 14.4 mg/kg per day (2).

There appears to be no basis for Dr. Ananth's dogmatic statement that "under no circumstances should this drug be administered to a nursing mother, and no mother taking this medication should be permitted to breast-feed her infant."

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Dysorexia and Self-Mutilation

SIR: Several of us who have studied varieties of self-mutilating behavior (1–5) have noted an incidence of dysorexia—bulimia, binge-eating, and vomiting—and periods of anorexia in such patients; the figures were over 62% in the series done by Rosenthal and associates (4) and 75% in my study (2). Japanese researchers investigating binge-eating and dysorexia have described the significant occurrence of self-mutilation. However, the majority of the Western literature on anorexia nervosa fails to mention such behavior, and researchers in this area do not seem to look for, inquire about, or record such mutilations. In none of the self-mutilating anorexic patients I have seen have the mutilations been recorded in the official case summaries, and they seem often to be omitted from the medical records altogether.

There are some interesting similarities between self-mutilating and anorexic patients, and an association, if one exists, could suggest further useful means of exploring both conditions. Anorexic and self-mutilating patients share characteristics such as an altered body image and self-control problems, difficulty in expressing or responding to emotions verbally rather than physically (by not eating or by cutting), difficulties in accepting adult sexual roles, and themes of loss and separation and reactions to loss.

I would be interested to know whether others have noticed this tentative relationship.

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Heat and Neuroleptics: A Deadly Combination

SIR: I would like to congratulate Stephan C. Mann, M.D., and William P. Boger, M.D., on their timely and much needed article, "Psychotropic Drugs, Summer Heat and Humidity, and Hyperpyrexia: a Danger Restated" (September 1978 issue), and add the following case report to the literature. This tragic case involved a robust young man who had been stabilized on the same medications for many months and whose DIC (disseminated intravascular coagulation) syndrome proved fatal.

Mr. A, a healthy 25-year-old single man, had been in psychiatric treatment for a number of years following a lengthy inpatient hospitalization. In spite of a diagnosis of chronic paranoid schizophrenia, he managed to graduate from a prestigious college, obtain gainful employment, and live in a cooperative boarding house. For 4 months he had been maintained without side effects and with good results on 64 mg of perphenazine, 150 mg of imipramine, and 10 mg of trihexyphenidyl HCl. The latter had been instituted because of persistent signs and symptoms of akathisia. Attempts to decrease the medication led to sleeplessness, agitation, and depression.

In August 1975, during a severe summer heat wave, coincidentally while his therapist was on vacation, the patient became progressively confused and agitated. Earlier in the day he had walked through the streets of his home town

distributing political leaflets. Unable to contact the therapist's substitute, Mr. A wandered into the unventilated, windowless washroom of his rooming house. Six to eight hours later he was discovered dying in the washroom, where the temperature was about 105°F (40°C).

Rushed to the emergency room of the local hospital, Mr. A remained unresponsive, with fixed, dilated, and unresponsive pupils. His skin was hot and dry. Temperature was 111.2°F (44°C) rectally, pulse 150, respiration 15, blood pressure 60/0. Shortly thereafter he became apneic and was intubated. His extremities were flaccid, reflexes were absent, and there were no spontaneous movements. Complete laboratory studies of blood, urine, and spinal fluid were within normal limits. Coagulation studies were also normal.

Mr. A never regained consciousness in spite of emergency treatment with an ice bath and later a cooling blanket, steroids, multiple pressor agents, plasma expanders, and antibiotics. Finally, he developed DIC syndrome with acute gastrointestinal bleeding, as well as a metabolic acidosis and focal seizures. In spite of the addition of fresh frozen plasma, packed red blood cells, cryoprecipitate, and albumin to the regimen, Mr. A died about 3 hours after admission.

Postmortem findings confirmed the diagnosis of heat stroke and specifically revealed diffuse hemorrhagic processes involving the upper respiratory tract, upper gastrointestinal tract and bladder, acute peritonitis and mesenteric lymphadenitis, splenic hemorrhage, hepatic necrosis, cerebral edema, and hemorrhagic pleural and peritoneal effusions. Toxicologic examination of multiple body fluids and tissue specimens was negative.

All physicians, particularly psychiatrists, should warn patients and their families, especially those on combinations of neuroleptics, of the dangers of summer heat and physical exertion. They can urge patients to take less drug, limit exercise, drink adequate fluids, and remain in an air-conditioned room during heat waves. Such a warning might have prevented this patient's tragic demise.

I, too, have been impressed both with the drug manufacturers' reluctance to publicize this serious complication and a general unawareness of this problem among psychiatrists and mental health workers.

RICHARD A. COOPER, M.D.
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SSI, the Sick Role, and Economics

SIR: In "Supplemental Security Income and the Sick Role" (October 1978 issue), H. Richard Lamb, M.D., and Alexander Rogawski, M.D., grapple with important but neglected issues. I agree that SSI as now provided tends to lock people into a disabled role. The recipient suffers but accepts this in return for secondary gain and a feeling of security. The authors' recommended solutions would be an improvement. However, there is an omission of the factor of world economic conditions.

There may not be enough U.S. jobs to go around. Perhaps we are obliged to consider with such patients the alternative of accepting the fate of the position in which they find themselves and focus their energies upon nonreimbursed leisure and good works.

JAMES N. HOOD, PH.D., M.D.
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Drs. Lamb and Rogawski Reply

SIR: Dr. Hood correctly points out that it is difficult to obtain income-producing opportunities for handicapped people in an economy which cannot offer full employment for able-minded and able-bodied people. Although this is a problem that must be addressed, it must also be recognized that the relevant social and economic problems are outside the competence and influence of mental health professionals.

Dr. Hood seems to agree with us that more than economic considerations are involved when a person becomes a recipient of SSI. We owe our patients constant attention to questions such as How much "help" is required to compensate for disabilities? When does "help" itself become an injurious influence?

In our article we hope to arouse interest in the possible damaging effects of SSI and we make suggestions for reducing untoward consequences.

H. RICHARD LAMB, M.D.
ALEXANDER S. ROGAWSKI, M.D.
Los Angeles, Calif.

Eye Size Discrepancy in Schizophrenia

SIR: In the past years, I have observed and documented a new physical symptom of schizophrenia, which, to my knowledge, has not yet been described. This ocular symptom consists of discrepancy in eye size, i.e., one eye looks larger than the other ("psychic unilateral exophthalmos", or PUE).

The study was conducted during 1977–1978 at Northville Regional Psychiatric Hospital (NRPH), Northville, Mich. The sample consisted of 336 schizophrenic patients, 16 of whom were diagnosed on admission as having acute schizophrenic episodes and had not taken any psychotropic medication. The diagnostic criteria for schizophrenia included formal thought disorder, delusions and/or hallucinations, flatness of affect, absence of gross medical pathology, and no history of medical conditions that might cause psychiatric symptoms. I selected consecutive admissions for the pathological sample. I then matched the randomly chosen control group of 336 normal individuals with the schizophrenic sample for race, age, and sex.

The technique of detecting difference in eye size was purely subjective and clinical, i.e., no measurements were done. However, I relied on three independent observers to contest or support my subjective impressions about the discrepancy in eye size. These observers were not told the subject of my study; they were asked only to look at the face of the subject and make a written report concerning any peculiar finding. They also did not know to which sample the subject belonged. Interrater reliability was found to be 90%.[1]

Of the 336 schizophrenics in this study, 159 (47.3%) showed clear evidence of discrepancy in eye size. Ninety-five patients in this group had a right eye larger than the left one, and the opposite was true for the 64 other patients. The magnitude of the ocular symptom was described as marked in only 4 cases, as moderate in 130 cases, and as mild in 23 cases. The eye that looks markedly or moderately larger actually protrudes from the orbital cavity. This finding justifies

[1]The data were analyzed by John B. Moir, Director of Psychology at NRPH, and Barbara Robertson.

the use of the term PUE. In 16 of the 23 cases in which the symptom was considered mild, the clinical impression was that there seemed to be a reduction of the palpebral fissure.

Ten of the 336 normal people in the control group showed the same type of ocular symptom observed in the schizophrenic sample in moderate or mild degree. In 7 of 10 cases, the impression was of a reduced palpebral fissure.

The 47.3% incidence of discrepant eye size in the schizophrenic patients compared to the 2.9% incidence in the control group (chi-square significant at .001 level) indicates that the ocular symptom and schizophrenia correlate statistically at a very high level of confidence. Such a correlation was not found between the ocular symptom and several other variables such as race, age, sex, dominance of the left hemisphere of the brain, severity of psychopathology, and medication administered.

The data reported in this study indicate it may be possible to predict the presence of schizophrenia from the discrepancy in eye size. Thus, it may be possible to predict schizophrenia or a borderline mental disorder or even a latent psychic deviance in people showing the ocular symptom but not the psychiatric syndrome.

Regarding the possible etio-pathogenesis of the observed ocular symptom in schizophrenics, it may be 1) an epiphenomenon of a general metabolic and/or endocrine disorder analogous to Graves' disease; 2) myogenic in nature due to a disorder of the neuromuscular system; 3) an interaction of the two; or 4) the result of numbers 1 and 2 but actually affecting each eye differentially.

Psychic unilateral exophthalmos (PUE) may prove significant in developing new research studies of schizophrenia. I plan to replicate this initial research using objective measurements. I also plan to apply the same method of investigation to other psychiatric syndromes. I believe that organic factors of a neurologic nature may be the basic, primary determinants of mental illness in general, of schizophrenia in particular, and of the observed ocular symptom specifically (1).

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ELIO MAGGIO, M.D.
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Questioning Combat Neurosis in Teachers

SIR: I wish to add some perspective to the paper "Combat Neurosis in Inner-City Schools," by Alfred M. Bloch, M.D., Ph.D. (October 1978 issue). Although Dr. Bloch presents the physical complaints of 253 teachers who had been referred for psychiatric evaluation, there was no control sample of physical complaints among teachers not seeking psychiatric evaluation. Considering that the ages in his patient population ranged up to 61, one would expect a significant number of teachers with some physical complaints, particularly when such categories as bronchitis and atherosclerosis are included. This patient population is hardly comparable to young men in their teens and 20s who have been described as having "combat neurosis."

Although he does not clarify what percentage of these teachers were involved in litigation, he mentions among his sources of referral union representatives and attorneys who represented teachers in Workers' Compensation applications.

He fails to consider the effect that the Workers' Compensation system, which introduces such variables as paid sick leave, fully paid lifetime medical care for various medical problems, disability retirement, and a chance to "get back at the system," would have on the presentation of his patients. Some teachers have had serious injuries, and clearly each case needs to be examined on an individual basis, keeping in mind this reminiscence of Dr. Leo Rangell (1): "In military psychiatry of World War II psychoanalytic psychiatrists confronted with the differential diagnosis between neurosis and malingering, underplayed or even ignored the latter, as if it did not exist."

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JOHN HOCHMAN, M.D.
Encino, Calif.

Dr. Bloch Replies

SIR: I acknowledge there was no formal control sample and that this is relevant. A subsequent study is under way that will include controls.

Dr. Hochman's description of sources of referral is misleading. Most of the referrals (85%) were initiated or recommended by other physicians. Years of well-documented chronological medical records antedated the patient's filing any claim. Although the psychiatric evaluation could be construed, in part, as a chance "to get back at the system," the symptoms preceded (often by years) anything that could be so construed.

The extensive medical substantiation also speaks out against this being the product of malingering, although subjective magnification undoubtedly occurred.

I certainly addressed the issue of secondary gain, which is not limited to the factors mentioned by Dr. Hochman. What I learned was that the most significant (conscious and unconscious) aspect of secondary gain is the fact that the disabling illness allows the individual to withdraw from an extremely stressful situation with a minimum of guilt and failure. I determined this, more than "paid benefits," to be a major factor. Any economic benefits that these people receive, almost without exception, do not equal the salary they would have received had they been able to continue teaching.

Since the original study I have evaluated a number of other teachers. My findings, as well as the findings of other physicians who have communicated with me, substantiate the original data and conclusions.

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Am J Psychiatry 136:4A, April 1979

BOOK REVIEWS

A Festering Sweetness: Poems of the American People, *by Robert Coles. Pittsburgh, Pa., University of Pittsburgh Press, 1978, 51 pp., $6.95; $3.95 (paper).*

This little volume could have been written only by Robert Coles. Its unusual name was suggested by a remark of William Carlos Williams, whom Dr. Coles had written about in a thesis as an undergraduate. Dr. Coles and Dr. Williams became fast friends, and Dr. Williams took the young man on his medical rounds on occasion. In a letter describing what he heard from day to day Dr. Williams hoped that the aspiring young doctor would not feel completely removed in future years from the moments of poetry in everyday life. He wrote, "Every day I hear these poor souls . . . talk poems to me. Sometimes I run to my car and write a few of their words down. Not every day, but on a lucky day, two or three times." Dr. Coles remembered these comments about working-class patients and saw that Dr. Williams both loved and disliked, needed and resented, them. Dr. Williams took heartily to their robust, hard-laboring life, "knew many of them to be utterly decent and thoughtful and kind—but also saw the scars, the warts, the mean and callous and hurt and sad side of their condition, the mix he referred to once as a 'festering sweetness.' "

These words fell on receptive ears as Dr. Coles, a young child psychiatrist working as a physician in the Air Force and based in Mississippi, saw the start of "the troubles" and the growing violence. He decided to stay in the South to try to find out how children who come face to face with a serious social and political crisis managed to get along psychologically, managed really to initiate, in the course of their everyday lives, what others have called a moment of historical change.

Although he started out asking "psychiatric questions" of the families he dealt with, Dr. Coles changed because he saw he was fitting their lives into his psychiatric categories and setting severe limits on what he would allow himself to see and to try to comprehend. It was finally a six-year-old black child, he says, who brought him to his senses: "I don't worry, no sir, I don't. I just go ahead past the crowd of people. They're the ones who are worried about me. I'm only worried about them—they must have bad dreams when they go to bed. When I go to bed I say my prayers with my grandmother and she tells me how lucky I am."

What Dr. Coles writes in his book are a number of "rough diamonds . . . not given the important company of symbol, allusion, and metaphor that Dr. Williams could provide. . . . All I have done is listen [and] keep track of what I have heard." Many of the people Dr. Coles worked with have been not only poor but hungry, wretchedly sick, and subject to many indignities, including rats, flies, viruses, and the absence of electricity.

The poems, culled from the even more distressing words Dr. Coles heard, are poignant. They range from the plaint of migrant workers to the productions of little girls to one titled "Growing Up with Love" written by a black mother in a Chicago tenement in 1970. All were heard by a compassionate physician with a feeling for people of all ages who hurt now and who have been hurt. I will not quote from the book—read for yourself. The whole work is only 51 pages long.

F.J.B.

Social Origins of Depression: A Study of Psychiatric Disorder in Women, *by George W. Brown and Tirril Harris. New York, N.Y., Free Press (Macmillan Publishing Co.), 1978, 389 pp., $19.95.*

This important work on social factors in depression appears at a time when most of us are impressed with the role of genetic factors in certain types of depression, the search for biological predictors, and the unquestionable efficacy of psychotropic drugs in the reduction of the acute symptoms of depression and as maintenance treatment. This book alerts us to the fact that depressions, like most medical disorders, occur in a psychosocial context. Using sophisticated methodology, the authors decipher those social factors which lead to increased risk of depression and develop links between social, economic, and cultural systems and their eventual impact on the individual.

The book is based on a study of about 100 inpatients and outpatients and more than 400 women in the communities of the Camberwell area of London and the Outer Hebrides in Scotland. Using various versions of the Present State Examination, Brown and Harris study the development of depressive disorders and their relationship to stress, provoking agents, and vulnerability factors.

This highly readable book integrates intensive clinical studies with an epidemiological approach, thus avoiding the scholarly but sterile content that often characterizes studies of this type. Although the text is filled with tables, statistics, and appendixes, the authors are careful to help the reader translate sociological concepts into clinical realities.

The major findings of the book follow:

1. Depression is extraordinarily common among working-class women in Camberwell, especially those with children at home, but is less common in rural Scotland.
2. Provoking agents influence when depression occurs.
3. Vulnerability factors determine whether the provoking agent will have an effect. These vulnerability factors include absence of a confiding, trusting relationship, particularly with the husband; presence of three or more children living at home; and death of the mother (but not the father) before the age of 11.
4. The effect of the vulnerability factors is additive in the face of a provoking agent (i.e., more vulnerability factors increase the risk of depression).

5. An outside job may reduce the risk of depression in women in the presence of vulnerability factors.

6. Working-class women are at increased risk because they have more vulnerability factors.

The findings of this book will not be accepted without debate and criticism. The high rate of depression found (i.e., 15% prevalence in Camberwell) raises a question about the looseness of the diagnostic criteria. The firmness of the evidence for the distinction between different etiological factors for psychotic and neurotic depression (i.e., psychotic depression is presumably linked to early parental death and neurotic depression to death and separations) must be replicated before it can be accepted. The broadness of the definition of loss or disappointment and, especially, the emphasis on the meaning of the event to the person will raise a question about reliability.

In fact, the controversy has already begun. Soon after the appearance of this book in London, it was the object of an editorial review and a letter in the prestigious British medical journal *Lancet* (1, 2), and soon the equally prestigious *Psychological Medicine* will be publishing a criticism. The full attention of an editorial in a medical journal for a book written by a sociologist and psychotherapist on a psychiatric topic is unprecedented.

The major finding, one that will be remembered, challenged, and studied, is the importance of intimacy as a protection against adversity in the lives of women. Although the link is not developed by the authors, this finding links this epidemiological study to the work of Bowlby, the animal ethologists, and all investigators interested in the importance of emotional attachments and bonds in the lives of people.

Mental health practitioners and sociologists who have always been interested in these issues will want to read this book. However, it should not be avoided by geneticists, biologists, and psychopharmacologists who search for an integrated understanding of human emotions.

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MYRNA M. WEISSMAN, PH.D.
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Affective Disorders: Drug Actions in Animals and Man. Handbook of Psychopharmacology, Vol. 14, *edited by Leslie L. Iversen, Susan D. Iversen, and Solomon H. Snyder. New York, N.Y., Plenum Press, 1978, 369 pp., $29.50.*

This volume is the fourteenth publication in a remarkable series devoted to neuropsychopharmacology and biological psychiatry. Previous volumes dealt with various aspects of basic sciences, such as neurochemistry and animal pharmacology. The present work is mainly concerned with human and animal psychopharmacology as well as applied clinical research in the area of affective disorders. This book successfully presents new progress in basic research in neuropsychopharmacology and important developments in the pathogenesis and biological treatment of affective disorders.

The volume contains a comprehensive review of the pharmacology of antidepressant drugs, including papers on monoamine oxidase (MAO) inhibitors, tricyclics, and lithium salts. There are two outstanding chapters: "MAO Inhibitors: Animal Pharmacology" by Richard Squires and "Lithium Pharmacology and Physiology" by Nelson Hendler. These are undoubtedly the most valuable contributions of the book. The chapters are well written and offer the reader an updated and comprehensive review of the MAO field.

The chapter on the biochemistry and metabolism of tricyclic drugs by F. Sulser is a good synthesis of current knowledge in this area. It also provides a critical appraisal of the clinical use of antidepressant medication. The chapter on the clinical use of lithium by Baron Shopsin and Samuel Gershon is remarkably clear but perhaps too restricted to the treatment of psychopathological aspects. A discussion of the physiological aspects of lithium in relation to kidney function and thyroid metabolism would be welcome in a work of such high quality.

The last chapter, on lithium prophylaxis and experimental rubidium therapy, is interesting, but too much attention is given to rubidium, a salt of great theoretical interest but little clinical value in the treatment of affective disorders.

This volume brings together important and high quality contributions on basic research and clinical applications in the biological understanding and treatment of affective disorders. It can be recommended for clinicians and researchers in neurobiology and biological psychiatry.

J. MENDLEWICZ, M.D., PH.D.
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Psychotropic Drugs: A Guide for the Practitioner, *by H.M. van Praag. New York, N.Y., Brunner/Mazel, 1978, 442 pp., $17.50.*

Dr. van Praag is known for his work on the pharmacology of antidepressants and neuroleptics and for his many contributions to clinical psychiatry. He is a versatile and original scientist; his book is provocative, scholarly, and useful for the investigator, the practitioner, and the medical student. The volume also has the advantage of single authorship.

The book is divided into 5 parts and 27 chapters. The first 20 chapters deal with such diverse topics as definition and conceptualization of major mental disorders, the classification of psychotropic agents, and the pharmacology and clinical uses of neuroleptics, antidepressants, and ataractics. Dr. van Praag states that "the schizophrenia concept has no justification" and "sorely needs revision on the basis of empirical psychopathological studies" (p. 166). He proposes a new concept of "schizophrenic psychoses" based on symptomatology, etiology, and outcome.

The author divides depressions into "vital" and "personal." The former correspond to the endogenous and the latter to the exogenous depressions; the term "vital" implies that many psychosomatic functions are depressed. This fact is often overlooked by the clinician, but the question is whether this new terminology has any practical advantage over the traditional one.

Dr. van Praag's use of psychotropic agents is based on the proposition that these compounds do not "respect the boundaries of the conventional nosological entities" and that "the syndrome or symptoms determine the indication" (p. 101). (Lithium, which is specific for mania, is the exception.) However, he does not provide any experimental evidence to prove that his criteria for the prescription of psycho-

pharmacological agents are superior to those based on diagnostic considerations.

The author's attitude toward psychotherapy is traditional. When there is strong evidence that a biochemical error underlies a mental aberration, drugs are the treatment of choice. On the other hand, maladaptive behavior, whatever its cause, necessitates psychological methods of therapy. In certain forms of anxiety ataractics may be prescribed because not all neuroses respond to psychotherapy and because there are "simply not enough trained psychiatrists to meet the demands" (p. 331). A recommendation of this type seems odd, since throughout this book the author emphasizes that pharmacotherapy is not a trial-and-error proposition or a matter of expediency.

Chapter 21 deals with sedatives and hypnotics, a topic that is often neglected in standard textbooks of psychiatry. Most sedatives have become obsolete since the introduction of benzodiazepines in the practice of mental care, but two time-honored drugs, paraldehyde and chloral hydrate, can still be used effectively in some conditions.

Chapter 22 is devoted to the controversial subject of the psychodysleptics. On the basis of extensive clinical experience with LSD and related compounds, the author's position is that these agents have the potential of powerful therapeutic compounds but that more research is needed to dispel current misconceptions about their psychological effects.

Dr. van Praag also deals with the use of drugs in children, in the aged, and in emergency situations. His chapters are succinct and lucid and provide useful guidelines for psychiatrists and family physicians.

One may disagree with some of Dr. van Praag's concepts and clinical points of view. Nevertheless, he must be given credit for his success in breathing new life into a branch of medicine that is relatively young but is already threatened by stagnation and misuse.

GEORGE E. CRANE, M.D.
Solana Beach, Calif.

Books to Help Children Cope with Separation and Loss, *by Joanne E. Bernstein. New York, N.Y., R.R. Bowker Co., 1977, 232 pp., $13.95.*

The size and scope of contributions to thanatology are growing, and much of the work is becoming monotonously repetitive and even boring. This book focuses on the larger issues of separation and loss as experienced by children in many different ways through their development, such as going to a new school, being hospitalized, moving to a new area, losing friends, getting lost, losing parents through separation, desertion, divorce, death, war, or displacement, and coming to terms with fostering, adoption, and step-parents. There is also a section for those children who remain homeless, although the topic of institutions for children is not directly covered. Bernstein has read and graded 438 children's books dealing in some way with these topics, although a few strange ones, like sex education, manage to creep in. Each book is given a well-written synopsis, a short, critical comment, the ages for which it is appropriate, and the reading level that it requires.

This book is a boon for parents, but child and adolescent psychiatrists will also find it useful to look at these nonclinical presentations of familiar clinical problems and even recommend them to families undergoing a crisis of adjustment. For those who like to include bibliotherapy among their clinical skills, this book should prove a valuable addition to their library.

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Cocaine: 1977. NIDA Research Monograph 13, *edited by Robert C. Petersen, Ph.D., and Richard C. Stillman, M.D. Rockville, Md., National Institute on Drug Abuse, 1977, 210 pp., $3.00.*

This monograph presents the history of cocaine, its chemistry (including methods for the detection of cocaine in body fluids), its possible mechanism of action, reports of the experiences of users, and an examination of its abuse potential and its use in medicine. Although each chapter is written by a different author, the editing is well done so that the style is uniform and clear. The perspective maintained throughout is scientific and professional. This is a welcome relief from the bias so often found when one reads about drugs of current social concern. For the reader who wants to know the salient, established facts about cocaine this monograph has no peer.

EDWARD C. SENAY, M.D.
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Interpersonal Psychoanalysis: New Directions, *edited by Earl G. Witenberg, M.D. New York, N.Y., Gardner Press (Halsted Press, John Wiley & Sons, distributor), 1978, 150 pp., $16.95.*

I thoroughly enjoyed reading this fine, small book. It is a heartening illustration of the many avenues of discovery available to an "open" system of psychoanalysis. The papers, which derive from a continuing professional education course presented at the William Alanson White Institute in New York by seven of the senior faculty members, have no unifying theme and do not need one. Each presents the current or matured thinking of its author on a subject of special interest to him or her, and we are fortunate to be able to share their thoughts. The authors are Marianne Horney Eckardt, David E. Schecter, Gerard Chrzanowski, Silvano Arieti, Edgar Levenson, Edward Tauber, and Jerome Singer.

In his lucid introduction Witenberg states a common ground:

> All these authors subscribe to a certain set of assumptions. They would all agree that there are broadening and changing indications for the kind of person to whom psychoanalysis is applicable. What this implies is that treatment must be tailored to the individual. Today's analyst needs to have a flexible approach. As times have changed, so have the problems for which people seek relief. . . . Dreams, bodily sensations, cognitive styles, and values, as well as language, are used to discover the unknown.

For psychiatrists, who are respectful of but not quite intimidated by the complexities of human existence, this volume will be a welcome source book for expanded therapeu-

tic orientations and interventions as well as an example of the good work being done in our field.

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Washington, D.C.

Community Mental Health: A General Introduction, *by Bernard L. Bloom. Monterey, Calif., Brooks/Cole Publishing Co. (Wadsworth Publishing Co.), 1977, 316 pp., $10.95.*

There are many books about community mental health, but few are as well written as this one. Bloom has provided an introductory text that is based on a historical perspective and is presented in a highly informative and interesting format.

The book is divided into three major sections: The Development of the Community Mental Health Movement, Community Mental Health in Practice, and Community Mental Health in Perspective. There are also three lengthy appendixes, including President Kennedy's message to Congress and the various titles and regulations of the Community Mental Health Centers Act, Public Law 88-164.

The book appears to be primarily oriented to new students who are interested in continuing in the field or who want to understand how the present community mental health system in the United States came into being and how it developed. Although much of the historical information in the first section can be found elsewhere, the author's ability to write clearly about what can be boring material, particularly regarding the role of the federal government, makes for an exciting and interesting background for what is to follow.

In chapter three in the first section Bloom reviews the Community Mental Health Centers Act as an instrument of social policy. This is a different perspective than one usually finds in books about mental health. Once again, it provides a special opportunity for students and other readers to think about and discuss not only the professional implications but also the social and political context in which community mental health programs function. In this regard, Bloom provides the broader and unique view of the community psychologist. He follows this up with a specific example of the impact of the act on Boulder, Colo., where he teaches. Even those who have never worked in complex social systems will gain a feeling of reality from this approach.

In the second section of the book Bloom deals with such community mental health practices as preventive intervention, mental health consultation, crisis intervention, and the assessment of community structure and community needs. He chooses not to discuss the full panoply of services that community mental health centers provide, and this might be a weakness in the book. However, the areas he does discuss are handled extremely well. For example, preventive intervention is given a base in the public health approach to disease prevention, with an extremely lucid explanation of public health concepts. Real examples throughout these chapters also serve to concretize the reader's perception and memory. Bloom appears to be well grounded in both mental health and public health theory and service as well as in an understanding of community systems. Research findings are used throughout and serve to develop a scientific base for the field.

In the final section Bloom not only gives a number of critical views of the community mental health movement but also stimulates the reader's thinking in a look beyond community mental health. He raises an important research-based question about a future perspective in the field, i.e., whether to focus on individual emotional difficulties or on the broader issues of our environment and society.

This book packs a great deal into a small volume. With its excellent reference section, its many research-oriented statements, and its articulate approach, it has much to offer as an introductory text and as a compact compendium for the mental health practitioner who wants to further his or her understanding of where this major field has been and where it is going.

ARTHUR J. BINDMAN, PH.D., M.P.H.
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The Family, Sex and Marriage in England: 1500–1800, *by Lawrence Stone. New York, N.Y., Harper & Row, 1977, 800 pp., $30.00.*

Although not all of us are family therapists, the nature of the family, including the interaction among its members, is of practical concern to almost any psychiatrist whose practice is not made up exclusively of unmarried orphans. This book will help one have a better understanding of families because it provides an understanding of the attitudes, customs, and institutions of society, a prediction of future trends, and a knowledge of the past and of the way today's conditions developed.

Lawrence Stone, Dodge Professor of History at Princeton, traces the evolution of family structure, patterns of sexual behavior, mating arrangements, and child-rearing practices in various social classes over a period of three centuries. From the title one might be concerned that a study of behavior in England would not be applicable to the United States because our population includes not only people of English and Western European heritage but also those of Native American, Oriental, and African background. However, data from colonial America are included, there is no denying the influence of English cultural patterns on the development of the United States, and the author does not overgeneralize. Another concern might be that the study does not include the changes in the nineteenth century of "moral reform" and sexual repression in the Victorian period or the further changes in the current century. Actually, changes since 1800 are discussed in the concluding chapter.

Although some of us may look backward with longing to some aspect of life in the earlier part of this century, few if any would care to live in the society that existed in England in the early 1500s. Social relationships in general tended to be cool or even hostile. The individual had little freedom of choice in marriage, and there was apparently little affection between marriage partners. Partly because of high infant mortality, there was little emotional investment in children. Neither the rich nor the poor had any real privacy within the home, and it was virtually impossible to maintain personal cleanliness (even if one desired it). Things changed, but there was no continuous linear change, nor did changes result from growing technical knowledge, a higher level of civilization, or the democratic spirit alone.

Stone tells us about the changes with meticulous scholarship and ample documentation but in a thoroughly readable style. The psychiatrist reader will find some interesting minor points in addition to the main thesis because the author is well aware of the influence of family life, sexual mores, and

child-rearing practices on personality development and mental health. He notes the types of problems seen by a "psychological practitioner" in the early eighteenth century and tells us about the work of a "prototype of Masters and Johnson" in the late eighteenth century.

One is sure to get some new ideas from reading this account of olden times, and one is almost sure to accept the author's conclusion:

> There is no reason to assume that the end-product of affective individualism, namely the intensely self-centered, inwardly turned, emotionally bonded, sexually liberated, child oriented family type of the third quarter of the twentieth century, is any more permanent an institution than were the many family types which preceded it.

In fact, I think the family may have changed since the last workshop on the family I attended.

MERRILL T. EATON, M.D.
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economically viable and the choice is supported by the prevailing societal ideology. However, they opt for new careers, regardless of their basic characteristics, when career opportunities are limited by economic considerations or by changes in societal ideology. In other words, economic incentives and societal ideology are more compelling for most students than their personal characteristics or original career preferences. The study also found little evidence that either the role models provided by the medical school faculty or curricular changes affect choice of medical career. Finally, the author summarizes findings with regard to the career choices of women and students who choose careers in primary care.

I found it disappointing that the report had so little to say concerning choice of careers in medically deprived areas. However, on the whole this is a timely and valuable text. No physician can afford to be unaware of the issues defined and discussed in the closing chapters titled "Current Issues in Medicine," "The Role of Medical Schools," and "Conclusions."

H. WALDO BIRD, M.D.
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Medical Students, Medical Schools and Society During Five Eras: Factors Affecting the Career Choices of Physicians 1958–1976, *by Daniel Funkenstein, M.D. Cambridge, Mass., Ballinger Publishing Co., 1978, 221 pp., $16.50.*

This book, written by a premier student of medical students, fittingly caps the author's distinguished career in medical education. It reports the results of a principally prospective longitudinal study of the career choices of medical students and physicians from 1958 to 1976. The author examined data collected on Harvard medical students at matriculation and graduation and as alumni and alumnae in 1974 and 1975, on University of Michigan medical students in the class of 1973, and on a national sample of medical students at matriculation and at graduation in the year 1975. However, at no point is the study's total number of subjects reported; this is a mild disappointment. More to the point, the book is concise and lucid and its text illuminated by 12 figures, 28 tables, and 3 questionnaires. To my delight and surprise, it often provided exciting reading.

The primary objective of the study was to determine the relative importance of various factors, as they changed over five different eras, in the career choices of medical students and physicians. Secondary goals were to identify the characteristics of students and physicians who chose careers in primary care, the characteristics of those who chose to practice in medically deprived geographic areas, and the differences in career choices of women and the factors that influenced them as compared with men. Although he does not explicitly state it as a goal, the author successfully undertakes another noteworthy task. He constructs a working model for making manageable the enormous and unwieldy mass of information available to any investigator on the relationship between the personal characteristics of medical students and their choices of career. He accomplishes this task in chapter one.

The Funkenstein study teaches important lessons to all who would influence the career choice of medical school aspirants, medical students, and physicians. The study makes it clear that medical students are reality oriented. In stable eras, they choose careers congruent with their personal characteristics, but only after determining that the career is

Undergraduate Medical Education and the Elective System: Experience with the Duke Curriculum, 1966–1975, *edited by James F. Gifford, Jr.; William G. Anlyan, M.D., Ewald W. Busse, M.D., and Thomas D. Kinney, M.D., supervising editors. Durham, N.C., Duke University Press, 1978, 243 pp., $9.75.*

This book will not be read in full by many psychiatrists. However, it is of such interest to the small but influential group of psychiatrists who sit on curriculum committees that it deserves to be called to their attention.

In 1966 Duke University School of Medicine introduced a curriculum change that the authors describe as the first new model of medical education responsive to the pressures for change of the 1960s. The new format provided for basic sciences in the first year and clinical clerkships (medicine, surgery, obstetrics/gynecology, pediatrics, and psychiatry) in the second year. The third and fourth years of the medical curriculum were used for elective courses equally divided between clinical subjects and basic sciences.

The chapters of the book dealing with preliminary planning and with the basic sciences are the most dynamic in that they deal with the "turf" questions of whose time was cut. These cuts were not always cheerfully accepted.

As a whole, the style of the book is somewhat turgid and reminiscent of a grant application. The decision to include a chapter on every basic science and every clinical department provides an excess of detail on programs, some of which may not have been altogether innovative. However, it must be acknowledged that individual chapters will be read by chairmen of similar departments. They will be read with interest because it is not always easy to know what is happening in other people's departments unless the familiarity is gained through site visits or similar contact. The specialty chapters on behavioral science and psychiatry are not radically innovative by present-day standards, but they represent a substantial advance from the psychiatric teaching of the 1950s.

Chapters on a number of interdisciplinary programs are provocative. These programs include M.D. with J.D. degrees and M.D. with M.P.H. degrees, medical history, and

biomedical engineering. These programs confirm the fact that Duke allows itself the luxury and the risks of trail-blazing.

On balance, this is a worthwhile book; it emphasizes the fact that the medical curriculum is not graven in stone. Introducing sophomore students to clinical studies is entirely practical, gratifying to the teacher, and probably an excellent idea. Electives in the later undergraduate years lead to some diffuseness but are sensible as a recognition that structure ends with graduation and that holding fast to the student for a few additional months is futile. Basic sciences in the advanced years raises arguable questions about what mix of subjects best prepares the student for the clinical practice of medicine. This in turn raises questions about the respective places of clinical skills and research skills in medical education. The Duke study documents the value of pedagogical experimentation within sensible limits that is carefully monitored for outcome.

ROY CLOUSE, M.D.
Philadelphia, Pa.

The Paranoid Process, *by W.W. Meissner, S.J., M.D. New York, N.Y., Jason Aronson, 1978, 840 pp., $30.00.*

In this work Dr. Meissner investigates both the pathological aspects and the constructive contributions of the paranoid process in human development and the normal organization of social and cultural structures. The book has three major divisions: an opening discussion and review of the literature, a presentation of clinical cases, and an articulation of a clinical theory about the paranoid process.

Among the topics reviewed in the first section are dynamic formulations and phenomenology of paranoia, pathological paranoid syndromes and related psychic states (envy, jealousy, and prejudice), "normal" related states (group formation, belief and value systems), paranoid mechanisms (projection, introjection, denial, paranoid construction), and catalysts of paranoid reactions (loss, depression, narcissistic injury). This review covers the gambit of psychoanalytic thinking and includes selected contributions from such nonanalytic literature as family studies, game strategies, and animal experiments.

In his second section Dr. Meissner presents vivid but wordy descriptions of 10 clinical cases. The variation in pathology in these cases is striking, including patients with acute and chronic schizophrenia; schizoid, narcissistic, and borderline personalities; and "analyzable" neurotic characters with phobic, obsessional, and hysterical features. The common thread in these accounts involves the use of paranoid mechanisms for defense and coping. Dr. Meissner convincingly demonstrates that these mechanisms arise from the matrix of a fairly repetitive family constellation.

For each patient one parental figure was perceived as controlling, powerful, willful, domineering, angry, intrusive, critical, or sadistic. The other parent was seen as passive, helpless, depressed, weak, submissive, masochistic, and victimized. Each patient internalized both figures and reenacted the parental sado-masochistic interaction in subsequent life experience, usually by projecting the sadism onto the external world and identifying oneself with the helpless, innocent victim. Additionally, one parent was usually symbiotically attached to the patient while the other was unavailable and rejecting. This led to an extreme perception of object relationships as either engulfing or abandoning.

In his third section Dr. Meissner returns to clinical and theoretical discussions of such topics as introjection, projection, phobic states, nightmares, narcissism, and the paranoid process in human development. In his formulations he draws heavily on the works of Searles, Kohut, Winnicott, Mahler, Erikson, and Rochlin. The hallmark of this work is thoroughness. This exhaustive review preserves the complexities of the literature. Dr. Meissner summarizes articles rather than merely citing them, thus providing a useful condensation of the overwhelming amount of writing on this topic.

Dr. Meissner's clinical discussions and theoretical formulations clearly support his contention that the paranoid process forms a core of normal and pathological human experience. The only area of confusion, in my view, concerns his discussion of the role of introjection and projection in human development. At times he maintains that these mechanisms serve to differentiate self from object and are therefore fundamental to the establishment of ego boundaries. At other times he indicates that projection and introjection require the previous existence of a certain degree of self-object differentiation in order to operate, thus introducing a contradiction.

My view is that projection and introjection have little to do with the differentiation of inner from outer experience. These mechanisms create an apparent perceptual difference between the "good" introjected object and the "bad" projected object, but this still involves a fusion or de-differentiation of self and object representations, an absence of ego boundaries. However, since these mechanisms serve the pleasure principle, they may be an adjunct to self-object differentiation by reducing anxiety to a "good enough" extent that the normal maturation of the infant's neuronal apparatus can proceed without major disruption. Introjection and projection operate to minimize the intrusions of overwhelming affects on the expanding integrative capacity of the CNS. Thus, I differ with Dr. Meissner's assessment that libido fosters the integration of good and bad introjects but aggression opposes and destroys this union, which constitutes a value judgment for libido and against aggression and overlooks the fact that both are basic and necessary. Love, like hate, can blind one to reality, and any strong drive or affect can be destructive to the formation of psychic structure and object constancy.

The major drawback of this book has little to do with the author's grasp of the issues and the literature, which stands unequaled among that of recent workers in this area. It has to do with size and organization. The book could be far less redundant and half as long without loss of cogency. Dr. Meissner allows too much overlap in his effort to elaborate on all aspects of the paranoid process. The result is a work encyclopedic in quality but formidable in magnitude.

THOMAS H. MCGLASHAN, M.D.
Rockville, Md.

The Community Training Center: An Educational-Behavioral-Social Systems Model for Rehabilitating Psychiatric Patients, *by Michael D. Spiegler, Ph.D., and Haig Agigian, M.S.W., A.C.S.W. New York, N.Y., Brunner/Mazel, 1977, 363 pp., $15.00.*

The rehabilitation of mentally ill patients is an area of American psychiatry that has received little attention. Probably because we are a pragmatic, task-oriented society, we

focus our efforts on etiology and treatment, ignoring partial hospitalization, aftercare, and rehabilitation—measures vitally needed by mental patients. Too few workers in American psychiatry and the mental health professions acknowledge that many mental patients, particularly those who have been hospitalized for a long period of time, have social and other deficits that must be rectified if the marginal individual is to be able to return to a reasonably active life.

This volume describes explicitly a comprehensive program for the rehabilitation of psychiatric patients in a community training center located at the Palo Alto, Calif., Veterans Administration Hospital. The authors developed an educational-behavioral approach in 1969; its specific purpose was to use educational technologies to rehabilitate mental patients. In classrooms, patients learn interpersonal and cognitive skills, community survival skills, health-related skills, and socialization skills. The authors describe various didactic and practice models whereby an individual learns such tasks as how to begin a conversation, use the telephone, budget an income, and live independently.

One of the chapters presents an intensive evaluation of the community training center and the outcomes for the patients who participated in the program during its first few years. Spiegler and Agigian consider the community training center a successful program for "ameliorating some of the misery of psychiatric patients." They also believe that their model can be applied beneficially for socially marginal individuals other than psychiatric patients—the physically handicapped, for example. I recommend this volume as a practical, day-by-day guide for psychiatrists and mental health workers who wish to devote their time and energies to the critically important problem of rehabilitation of the mental patient.

JOHN J. SCHWAB, M.D.
Louisville, Ky.

Dying: A Psychoanalytic Study with Special Reference to Individual Creativity and Defensive Organizations, *by Tor-Bjorn Hagglund. New York, N.Y., International Universities Press, 1978, 253 pp., $15.00.*

Dr. Hagglund, a practicing psychoanalyst and training analyst in the Finnish Psychoanalytic Society, provides an excellent review of the literature on psychodynamics of individuals facing death. He also presents five case histories. Using material from these cases as well as his extensive analysis of the poetry of Edith Södergran, Hagglund formulates a description of the interplay among narcissism, phallic defenses, and the process of creativity during mourning.

Basically, when faced with death an individual increases all defenses, particularly denial. Mourning work begins with a decrease of denial, during which the person relinquishes objects, life, and body sequentially. The wish for immortality then leads to the formation of an "auxiliary ego" that maintains object relations after death, or the patient envisions life as continuing in children, work, or accomplishments. Finally, as death progresses, regression predominates and psychic life is regulated by splitting. Emotions and objects are divided into good and bad, with the world to be left as bad and the fantasy of death or of the person's oneness with the therapist (or other human) as good. This dynamic sequence leaves the impression that pathological mechanisms characterize the dying process; generalization from a small sample may have produced this distortion.

The author's prime theoretical contribution is his view of creativity as arising during the mourning process. Mourning is experienced as something unreasonable and possibly unbearable for the dying person's narcissism. The less creative person defends against mourning with phallic narcissistic defenses, which serve as a safeguard against frustrations in human relationships. The creative person, on the other hand, is able to identify with the subliminated form of woman's (mother's) inner space, which enables the individual to expand creatively the two-person relationship. For the nonpsychoanalyst reader the author's adherence to analytic jargon will make comprehension of his theory difficult.

NED H. CASSEM, M.D.
Boston, Mass.

Self-Starvation: From Individual to Family Therapy in the Treatment of Anorexia Nervosa, *by Mara Selvini Palazzoli; translated by Arnold Pomerans. New York, N.Y., Jason Aronson, 1978, 289 pp., $15.00.*

Mara Palazzoli is a classical psychoanalyst who has had years of experience in treating patients with anorexia nervosa through traditional analytic methods. In *Self-Starvation,* however, she advocates a family approach of brief intervention, using largely behavioral methods. The book is in part a historical treatise on the author's journey through the therapeutic maze that has led her to her present approach. As such it is an intriguing confession of an individual who has not compromised objective observation and experimentation by doctrine and who has not been bound by only one conceptual orientation to the phenomenon she observes. Dr. Palazzoli does not dismiss a psychoanalytic approach in attempting to gain a grasp on the developmental psychosexual dynamics of the disorder. Indeed, her work suggests that this approach gives a substantive basis from which to proceed with further experimental models of therapy. This book is not a polemic advocating a single model of approach or explanation of the therapy. Rather, it seems a worthy heir to Dollard and Miller's classic examination of the complementarity of psychoanalytic and behavioral approaches in understanding the human condition (1).

Most important, Dr. Palazzoli comes across in this book as a complete physician. She knows the subject she addresses, anorexia nervosa, and the patients who endure this prototypical psychosomatic disease. She realizes that there is flesh and life involved as well as psyche. She advises the psychiatrist to take cognizance of the body while examining the psyche. In this, she conveys her own competence as a physician as well as a psychologist. Above all, she knows patients and their families.

Dr. Palazzoli came to a family approach to the treatment of anorexia nervosa through the work of Dr. G. Benedetti of Basel in 1967. At that time she established the Milan Center of Family Studies. This work is a review of both her earlier approaches and her later ones. She apologizes for the absence of statistical analyses of her later family approaches. These have recently been published in her book *Paradox and Counter-Paradox* (2).

Self-Starvation, although first published in Italian in 1971, arrives on the American scene along with the work of Hilde Bruch (3) and Salvadore Minuchin and associates (4) as well as Levenkron's novel, *The Best Little Girl in the World* (5). Dr. Palazzoli acknowledges her debt to Dr. Bruch and Dr.

Minuchin and associates, but her work goes beyond both Dr. Bruch's psychological insights and phenomenological observations and Dr. Minuchin's family studies. Dr. Palazzoli's identification of specific communication processes, such as affirmation, negation, and deaffirmation, concepts drawn from Jay Haley's models (6), is especially clearly drawn and presented in recorded excerpts from family therapy sessions.

Although Dr. Palazzoli's presentation has a bit of the enthusiasm of a convert, this does not interfere with the precision of her observations of patients with anorexia nervosa and of their families. The work suffers a bit from the author's still uneven incorporation of existential terms into her more evenly used psychoanalytic vocabulary. This deficiency is less obvious in her latest work (2). What Dr. Palazzoli seems to be achieving in the present work is a synthesis based on the approaches of psychoanalysis, existential analysis, and behavioral theory, including communication theory. More important, the use of these conceptual models is based on the firm foundation of careful observation over many years. A great deal of wisdom will be obtained and synthesized by the reader working through this provocative book.

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3. Bruch H: The Gilded Cage: The Enigma of Anorexia Nervosa. Cambridge, Harvard University Press, 1978
4. Minuchin S, Rosman BL, Baker L: Psychosomatic Families: Anorexia Nervosa in Context. Cambridge, Harvard University Press, 1978
5. Levenkron S: The Best Little Girl in the World. Chicago, Contemporary Books, 1978
6. Haley J: Strategies of Psychotherapy. New York, Grune & Stratton, 1963

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This Month's Reviewers

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Books Received

This listing acknowledges the receipt of recent books. Books of particular interest to the readers of this journal will be reviewed as space permits, and copies of the reviews will be sent to the publishers. Books cannot be returned to the publishers.

A Handbook for Specific Learning Disabilities, *edited by William C. Adamson, M.D., and Katherine K. Adamson, M.D. New York, N.Y., John Wiley & Sons, 1979, 501 pp., $27.50.*

Music Therapy for the Autistic Child, *by Juliette Alvin. New York, N.Y., Oxford University Press, 1978, 120 pp., $8.95 (paper).*

QuaSAR: Quantitative Structure Activity Relationships of Analgesics, Narcotic Antagonists, and Hallucinogens. NIDA Research Monograph 22, *edited by Gene Barnett, Ph.D., Milan Trsic, Ph.D., and Robert E. Willette, Ph.D. Rockville, Md., National Institute on Drug Abuse, 1978, 487 pp., single copies free (paper).*

Behavioural Treatment of Obsessional States, *by H.R. Beech and M. Vaughan. New York, N.Y., John Wiley & Sons, 1978, 179 pp., $18.95.*

Dyslexia: An Appraisal of Current Knowledge, *edited by Arthur L. Benton and David Pearl. New York, N.Y., Oxford University Press, 1978, 525 pp., $19.50.*

Schizophrenia: Symptoms, Causes, Treatments, *by Kayla F. Bernheim and Richard R.J. Lewine. New York, N.Y., W.W. Norton & Co., 1979, 245 pp., $14.95; $5.95 (paper).*

The Schizophrenic Disorders: Long-Term Patient and Family Studies, *by Manfred Bleuler; translated by Siegfried M. Clemens. New Haven, Conn., Yale University Press, 1978, 519 pp., $45.00.*

How to Enjoy the Love of Your Life: Over 100 Ways to Enrich Your Love Life, *by Harold H. Bloomfield, M.D. Garden City, N.Y., Doubleday & Co., 1979, 215 pp., $8.95.*

.44, *by Jimmy Breslin and Dick Schaap. New York, N.Y., New American Library, 1978, 329 pp., $2.50 (paper).*

The Alcoholic Employee: A Handbook of Helpful Guidelines, *by Ashton Brisolara. New York, N.Y., Human Sciences Press, 1979, 164 pp., no price listed.*

The Health Care Dilemma: Problems of Technology in Health Care Delivery, *by J.H.U. Brown, Ph.D. New York, N.Y., Human Sciences Press, 1978, 176 pp., $14.95.*

Aging: Its Complex Management, *by Ronald Cape, M.D. Hagerstown, Md., Harper & Row Medical Department, 1978, 239 pp., $14.50.*

Annual Progress in Child Psychiatry and Child Development 1978, *edited by Stella Chess, M.D., and Alexander Thomas, M.D. New York, N.Y., Brunner/Mazel, 1978, 642 pp., no price listed.*

Psychiatry in Dissent: Controversial Issues in Thought and Practice, *by Anthony Clare. Philadelphia, Pa., Institute for the Study of Human Issues, 1979, 426 pp., $15.00.*

Sexual Consequences of Disability, *edited by Alex Comfort, M.B. Philadelphia, Pa., George F. Stickley Co., 1978, 281 pp., $24.50.*

Who's the Patient Here? Portraits of the Young Psychotherapist, *by Stuart Copans, M.D., and Robert Singer, M.D., New York, N.Y., Oxford University Press, 1978, 163 pp., $5.95 (paper).*

Dialysis Review, *edited by Alex M. Davison. Philadelphia, Pa., J.B. Lippincott Co., 1978, 277 pp., $19.00.*

Coronary-Prone Behavior, *edited by Theodore M. Dembroski, Stephen M. Weiss, Jim L. Shields, Suzanne G. Haynes, and Manning Feinleib. New York, N.Y., Springer-Verlag, 1978, 236 pp., $14.80.*

Serial Handbook of Modern Psychiatry, Vol. III: Textbook of Clinical Psychopharmacology, *by Herman C.B. Denber, M.D., Ph.D. New York, N.Y., Stratton Intercontinental Medical Book Corp., 1979, 335 pp., $29.50.*

Basic Groupwork, *by Tom Douglas. New York, N.Y., International Universities Press, 1978, 191 pp., $12.50.*

The American Way of Life Need Not Be Hazardous to Your Health, *by John W. Farquhar, M.D. New York, N.Y., W.W. Norton & Co., 1978, 191 pp., $9.95.*

Psychotherapy and the Human Predicament: A Psychosocial Approach, *by Jerome D. Frank; edited by Park Elliott Dietz. New York, N.Y., Schocken Books, 1978, 323 pp., $16.95.*

Annual Review of Behavior Therapy Theory and Practice, Vol. 6: 1978, *edited by Cyril M. Franks, Ph.D., and G. Terence Wilson, Ph.D. New York, N.Y., Brunner/Mazel, 1978, 734 pp., $27.50.*

Psychoanalysis and the Question of the Text, *edited by Geoffrey H. Hartman. Baltimore, Md., Johns Hopkins University Press, 1978, 182 pp., $7.50.*

Child Analysis and Therapy, *edited by Jules Glenn, M.D. New York, N.Y., Jason Aronson, 1978, 748 pp., $30.00.*

Neuroscience and Clinical Neurology Review, *by David Goldblatt, M.D. New York, N.Y., Arco Publishing Co., 1979, 205 pp., $10.00.*

When Your Child is Hyperactive, *by Kenneth Heiting. St. Meinrad, Ind., Abbey Press, 1978, 96 pp., $1.95 (paper).*

Classical Conditioning and Operant Conditioning: A Response Pattern Analysis, *by Wendon W. Henton and Iver H. Iversen. New York, N.Y., Springer-Verlag, 1978, 346 pp., $24.50.*

Treating Sexual Problems in Medical Practice, *by David K. Kentsmith, M.D., and Merrill T. Eaton, M.D. New York, N.Y., Arco Publishing Co., 1979, 168 pp., $12.00 (paper).*

A Beginning Manual for Psychotherapists, 2nd ed., *by Ernest Kramer, Ph.D. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1978, 166 pp., $10.75.*

Guide for the Beginning Therapist: Relationship Between Diagnosis and Treatment, *by Amy Lamson, Ph.D. New York, N.Y., Human Sciences Press, 1978, 116 pp., $4.95 (paper).*

The Wild Boy of Burundi: A Study of an Outcast Child, *by Harlan Lane and Richard Pillard. New York, N.Y., Random House, 1978, 180 pp., $10.00.*

International Journal of Psychoanalytic Psychotherapy, Vol. VII, 1978–1979, *edited by Robert Langs, M.D., and the Editorial Board. New York, N.Y., Jason Aronson, 1978, 608 pp., $27.50.*

Technique in Transition, *by Robert Langs, M.D. New York, N.Y., Jason Aronson, 1978, 712 pp., $30.00.*

Interpreting Transference, *by Nathan Leites. New York, N.Y., W. W. Norton & Co., 1979, 160 pp., $10.95.*

Activity, Consciousness, and Personality, *by A.N. Leont'ev; translated from Russian by Marie J. Hall. Englewood Cliffs, N.J., Prentice-Hall, 1978, 186 pp., $15.95.*

How's Your Family? A Guide to Identifying Your Family's Strengths and Weaknesses, *by Jerry M. Lewis, M.D. New York, N.Y., Brunner/Mazel, 1979, 185 pp., $10.00.*

The Classic Clitoris: Historic Contributions to Scientific Sexuality, *edited by Thomas Power Lowry, M.D. Chicago, Ill., Nelson-Hall, 1978, 113 pp., $14.95.*

New Vistas in Counseling Series, Vol. IX: Basic Behavior Modification, *by Albert Mehrabian, Ph.D. New York, N.Y., Human Sciences Press, 1978, 86 pp., $8.95.*

The Chemistry of Human Behavior, *by Herbert L. Meltzer, Ph.D. Chicago, Ill., Nelson-Hall, 1979, 254 pp., $17.95.*

Alcoholism: New Directions in Behavioral Research and Treatment, *edited by Peter R. Nathan, G. Alan Marlatt, and Tor Loberg. New York, N.Y., Plenum Press, 1978, 397 pp., $35.00.*

The Disordered Mind: What We Know Now About Schizophrenia, *by Patrick O'Brien, M.D. Englewood Cliffs, N.J., Prentice-Hall, 1978, 297 pp., no price listed.*

The Hippocampus as a Cognitive Map, *by John O'Keefe and Lynn Nadel. Oxford, England, Oxford University Press, 1978, 543 pp., $30.00.*

Having Children: Philosophical and Legal Reflections on Parenthood, *edited by Onora O'Neill and William Ruddick. New York, N.Y., Oxford University Press, 1979, 362 pp., $5.95 (paper).*

The Form and Technique of Psychotherapy, *by I.H. Paul. Chicago, Ill., University of Chicago Press, 1978, 283 pp., no price listed.*

Phencyclidine (PCP) Abuse: An Appraisal. NIDA Research Monograph 21, *edited by Robert C. Petersen, Ph.D., and Richard C. Stillman, M.D. Rockville, Md., National Institute on Drug Abuse, 1978, 313 pp., no price listed (paper).*

The Dissociation of a Personality: The Hunt for the Real Miss Beauchamp, *by Morton Prince. New York, N.Y., Oxford University Press, 1978, 570 pp., $7.95 (paper).*

Phone Calls from the Dead, *by D. Scott Rogo and Raymond Bayless. Englewood Cliffs, N.J., Prentice-Hall, 1979, 168 pp., $8.95.*

Career and Motherhood: Struggles for a New Identity, *by Alan Roland, Ph.D., and Barbara Harris, Ph.D. New York, N.Y., Human Sciences Press, 1979, 208 pp., $12.95.*

Total Glow: Dr. Rona's Unbeatable Health Program, *by Luanne Rona, M.D. Wilmington, Del., Enterprise Publishing Co., 1978, 259 pp., $9.95.*

Psychopharmacology and Psychotherapy: Synthesis or Antithesis? *edited by Norman Rosenzweig, M.D., and Hilda Griscom, Ph.D. New York, N.Y., Human Sciences Press, 1978, 245 pp., $14.95.*

Networking Families in Crisis, *by Uri Rueveni. New York, N.Y., Human Sciences Press, 1979, 158 pp., $14.95.*

Once Upon a Murder, *by Andrew Keogh Ruotolo, M.D. New York, N.Y., Grosset & Dunlap, 1978, 223 pp., $10.00 (paper).*

Mental Illness in Pregnancy and the Puerperium, *edited by Merton Sandler. New York, N.Y., Oxford University Press, 1978, 119 pp., $16.95.*

A Biodevelopmental Approach to Clinical Child Psychology: Cognitive Controls and Cognitive Therapy, *by Sebastiano Santostefano. New York, N.Y., John Wiley & Sons, 1978, 813 pp., $39.95.*

Social Order and Mental Health: The Florida Health Study, *by John J. Schwab, Roger A. Bell, George J. Warheit, and Ruby B. Schwab. New York, N.Y., Brunner/Mazel, 1979, 211 pp., $15.00.*

Psychoanalysis and Old Vienna: Freud, Reik, Schnitzler, Kraus, *edited by Murray H. Sherman. New York, N.Y., Human Sciences Press, 1978, 192 pp., $12.95.*

The Psychoanalytic Study of the Child, Vol. 33, *edited by Albert J. Solnit, M.D., Ruth S. Eissler, M.D., Anna Freud, LL.D., D.Sc., Marianne Kris, M.D., and Peter B. Neubauer, M.D. New Haven, Conn., Yale University Press, 1978, 652 pp., $22.50.*

Public Welfare, *by Arthur Spindler. New York, N.Y., Human Sciences Press, 1979, 505 pp., $29.95.*

Psychological Interventions in Medical Practice, *by James J. Strain, M.D. New York, N.Y., Appleton-Century-Crofts, 1978, 213 pp., no price listed.*

The Psychiatric Clinics of North America: Symposium on Obesity: Basic Mechanisms and Treatment, *edited by Albert J. Stunkard, M.D. Philadelphia, Pa., W.B. Saunders Co., 1978, 724 pp., no price listed.*

Wholistic Health: A Whole-Person Approach to Primary Health Care, *by Donald A. Tubesing, Ph.D. New York, N.Y., Human Sciences Press, 1979, 225 pp., $12.95.*

Unwanted Pregnancy: Accident or Illness? *by David Tunnadine and Roger Green. New York, N.Y., Oxford University Press, 1978, 183 pp., no price listed.*

Children's Rights: Contemporary Perspectives, *edited by Patricia A. Vardin and Ilene N. Brody. New York, N.Y., Teachers College Press, 1978, 182 pp., $6.95 (paper).*

Your Family Is Good for You, *by Harvey White, M.D. New York, N.Y., Random House, 1979, 204 pp., $8.95.*

Schizophrenia: Towards a New Synthesis, *edited by J.K. Wing. New York, N.Y., Grune & Stratton, 1978, 286 pp., $16.50.*

Human Learning and Memory: An Introduction, *by Arthur Wingfield. New York, N.Y., Harper & Row, 1979, 444 pp., $13.95.*

The Hidden Dimension: Psychodynamics in Compulsive Drug Use, *by Leon Wurmser, M.D. New York, N.Y., Jason Aronson, 1978, 624 pp., $30.00.*

Notice of Subscription Price Increase

The *American Journal of Psychiatry* has not increased its subscription price to nonmember subscribers since 1974, in spite of substantial increases in a number of areas: printing and mailing costs, cost of paper, size of the *Journal*, including the addition of the Clinical and Research Reports section, size of the staff, and new procedures for the critical review of manuscripts. We have now reluctantly come to the conclusion that a price increase to nonmember subscribers can no longer be postponed. Accordingly, effective March 1, new subscriptions to the *American Journal of Psychiatry* cost $27 a year for domestic subscribers and $35 a year for foreign subscribers, including those in Canada. A single issue costs $3.50 in the United States and $4.25 for orders from foreign countries, including Canada.

OFFICIAL ACTIONS

Fall Committee Meetings

Following is a summary of the proceedings of the fall committee meetings of the American Psychiatric Association, which were held in Washington, D.C., in September 1978.

The Council on Children, Adolescents, and Their Families

EDWARD H. FUTTERMAN, M.D., CHAIRPERSON

DURING the winter of 1978, the Council received 24 volumes in tentative draft form of the Juvenile Justice Standards Project prepared by the Institute of Judicial Administration and the American Bar Association. Major decisions of the project were "to reject the medical or rehabilitative model of the Juvenile Court" and to establish the principle of family autonomy as paramount over the child's best interests. APA urged the American Bar Association to delay approval pending further review. The Council appointed an *Ad Hoc Task Force on the Juvenile Justice Standards Project,* chaired by Frank Rafferty, M.D., which worked rapidly and effectively to prepare a response to the overall thrust of the recommendations and a detailed critique of those volumes about which clinical commentary would be most relevant. The 115-page APA response was approved for transmittal by the Board of Trustees, and a summary of the APA response is being prepared by Andre Derdyn, M.D. Both of these documents are available through the Central Office. The American Bar Association is scheduled to vote on these standards at its February 1979 meetings.

The *Task Force on the Rights of Children, Adolescents, and Their Parents,* Perry Bach, M.D., chairperson, drafted a critique of the Mental Health Law Project *Legislative Guide* section on "Mental Health Treatment For Minors." Viola Bernard, M.D., completed the critique for the Council, which was joined by the American Academy of Child Psychiatry, the American Association of Psychiatric Services for Children, and the American Society of Adolescent Psychiatry in submitting the product to the *Mental Health Disabilities Law Reporter.* In view of the delay by the Supreme Court in handing down a decision on the Parham case and the accelerated activities in many states to introduce a variety of procedures affecting the treatment and hospitalization of minors, the Commission on Government Relations agreed to distribute the Council's critique to the membership for use in dealing with individual state legislatures. In addition, the Council decided that an alternative model statute for treatment of minors, prepared by psychiatrists, was needed to compete with the one developed by the Mental Health Law Project. Therefore, a Task Force on Commitment of Minors was formed, chaired by Michael Kalogerakis, M.D., and charged with drafting a model commitment law for minors. It has begun its work by reviewing the legal and clinical literature pertaining to the rights and needs of children, adolescents, and parents regarding psychiatric hospitalization and reviewing existing state statutes. The task force is at the stage of preparing a first draft of a model statute with commentary.

The *Task Force on Children and Adolescents in State Mental Hospitals,* Paul N. Graffagnino, M.D., chairperson, has completed its survey of all 50 states. The data are being tabulated, examined, and analyzed. A final report on this work is anticipated by the summer of 1979.

The *Task Force on Alternative Patterns of Organization of State Governments to Provide Mental Health Services to Children and Adolescents,* Frank Rafferty, M.D., chairperson, has completed its work. On the basis of surveys and informal information obtained from all 50 states, the task force identified five patterns of organization of child and adolescent services. They could not identify any dynamic flow from one pattern to another or a clear rationale for the choice of a particular pattern by a particular state. A historical review revealed a lack of influence by the child guidance movement and by the mental retardation movement on state departments of mental health. State actions seemed to be more dependent on federal categorical initiatives than on any other variable. Another finding from the report was a steady decline in participation by psychiatrists and other skilled child mental health professionals in state systems. The implications of this report will be studied by the Council for appropriate action.

The Council's statement on Public Law 94-142 (Education for All Handicapped Children Act) has been circulated to all components of APA. The newly formed *Task Force on Treatment and Education,* Irving Berlin, M.D., chairperson, is following up on this issue and collecting information from local groups who are working with schools to identify the problems and possibilities. Since each state must develop its

own plan and there are 20,000 autonomous school districts in the nation, the task force is interested in obtaining information from district branches. They are working not only on the need to amend the law and its regulations but also on its implications for mental health manpower and for training. The task force has also developed a paradigm for distinguishing activities in any setting that would be considered educational as opposed to therapeutic. The financial implications of these distinctions are also being considered.

The *Task Force on Services to Children and Adolescents in Community Mental Health Centers,* David Leaverton, M.D., chairperson, is preparing its final report. This product will include the results of a survey of services to children and adolescents in CMHCs, a narrative description of some successes and failures, a review of the role of child psychiatrists in CMHCs, a review of the standards of care for children, adolescents, and families in CMHCs, and recommendations about federal guideline changes and future federal support.

The *Task Force on Child Psychiatry and Family Practice,* Ruth Lavietes, M.D., chairperson, is nearing completion of its work. The plan to develop a curriculum in child psychiatry for family practice training programs has been abandoned since most programs are not amenable to input from specialties. Therefore, the task force has turned its emphasis to the process of integrating child psychiatric concepts into the training of family practitioners. Recommendations are being formulated regarding education and liaison.

The *Task Force on Teenage Parents and Their Children,* JoAnne Fineman, M.D., chairperson, has begun its work by amassing a comprehensive bibliography and reviewing causes and interventions. Most current programs are embedded in pediatric, medical, church, educational, and casework settings, with very little psychiatric input. No cohesive national policy has emerged. The task force is developing preliminary position papers and educational activities as well as monitoring legislation on teenage pregnancy. The task force is also maintaining liaison with the Coalition for Optional Parenthood Education (Taghi Modarressi, M.D., APA representative).

The Council reviewed work on television and children being done by the American Medical Association, the American Academy of Child Psychiatry, the Group for the Advancement of Psychiatry, Conference on Television and Socialization of Minority Children, PTAs, and NIMH. The Council recommends that APA continue to support the American Medical Association's position on television and violence. Furthermore, the Council asked that the delegates to the Section Council on Psychiatry of the American Medical Association introduce a resolution at the AMA House of Delegates in support of an AMA Conference on Family Violence. In transmitting this recommendation, the Council stressed the need for inclusion of the roles of television and other media in influencing violence in the family.

Due to the lack of integration of family therapy into general psychiatric practice and controversy about the relationship between family therapy and other therapeutic modalities, the Council decided to establish a Task Force on Psychiatry and Family Therapy. The preliminary charge of the task force is to study the clinical indications and contraindications for family therapy, to review the integration of family therapy with other psychiatric therapeutic modalities, and to review issues in training and accreditation in family therapy.

The Council expressed concern about the impact on child development of the variety of family life patterns now emerging, such as single-parent families, father-only families, nonrelated extended families, homosexual parents, etc. The Council proposed a new Task Force on Alternative Family Styles. The preliminary charge was to review the impact of the variety of emerging family life patterns on child development; to identify and review issues of psychiatric concern such as needed services and supports and implications for child custody; to make specific recommendations regarding prevention of child psychopathology, promotion of optimal child development, models of service, and forensic positions relating to custody; and to prepare a position statement on fitness for parenthood. Since the Council on National Affairs also proposed a Task Force on Changing Roles and Family Practices in American Life, it was agreed to merge these two efforts.

The Council also proposed establishment of a Task Force on Clinical Assessment in Child Custody. The preliminary charge was to develop guidelines for practice in custody evaluations, including attention to voluntary and involuntary requests for evaluations, relationship to the courts and other professional agencies, critieria for making recommendations and preparation of reports. At the request of the Reference Committee, the Council agreed to review the draft of the report on child custody by the Group for Advancement of Psychiatry prior to establishing this task force.

In other actions, the Council worked with the Commission on Government Relations on guidelines for national health insurance, the Child Health Assessment Act, and other legislative matters. The Council also participated in the final drafting of the APA position statement on abortion. In addition, liaison has been maintained with the National Consortium on Child Mental Health, the National Institute of Mental Health, and the American Academy of Child Psychiatry. Regarding the latter, the Council worked with Robert Reichler, M.D., in preparing responses to drafts of Department of Health, Education, and Welfare regulations regarding research on children.

The Council wishes to express its appreciation for the support and cooperation of all components of APA in its efforts to integrate child and adolescent psychiatry into the fabric of the organization.

The Council on Emerging Issues

PERRY OTTENBERG, M.D., CHAIRPERSON

THE COUNCIL, which meets three times each year, is presently deeply concerned with a number of major emerging issues:

1. A Task Force on Impaired Physicians will be formed in 1979 and charged with studying the problems, resistances, and delays in obtaining help for impaired physicians, including psychiatrists; paying particular attention to physicians who function in isolated office settings outside of hospital staffs and without frequent contact with colleagues; determining what are the optimal intervention models and confrontational techniques that are useful in helping to get impaired physicians into therapy; and considering whether there is overreliance on the Alcoholics Anonymous model for the treatment of alcoholism in the absence of psychiatric care for impaired physicians. The Council views these as the core tasks; after they have been dealt with, the Council believes that the same or another task force should undertake an assessment of the life experiences that underlie successful mastery of stresses inherent in the physician's role.

2. The Council also proposes to establish a Task Force on Changing Family Patterns in American Life. Interest in the subject is largely an outgrowth of special sessions at the last two annual meetings on the changing roles of males and is also influenced by such factors as the growing numbers of single-parent families, recombinant families (newly divorced, with children, and remarried), dual career families, and increasing numbers of individuals who prefer to live alone.

3. The Council's *Committee on Abuse and Misuse of Psychiatry in the United States,* Eugene B. Feigelson, M.D., chairperson, has formulated a charge for its work:

> The Committee shall receive and evaluate any charges of psychiatry being used for political purposes in the United States; where appropriate, such charges shall be referred to the World Psychiatric Association. The Committee shall also develop and receive instances of individual or institutional misuse and/or abuse of psychiatry in the United States that do not fall under the Ethics Committee (an extension of the intent of a process begun in 1972 by APA).

4. The *Task Force on Psychosocial Impacts of Nuclear Advances* has met several times under chairperson Rita Rogers, M.D. It is currently developing a questionnaire intended to elicit attitudes toward three target groups: the Clamshell Alliance, the "I-don't-knows," and the Committee on the Present Danger. Other major concerns of the task force include educational aspects of nuclear advances, the pervasive secrecy that surrounds the social issue, how terrorism is involved, dehumanization, collective attitudes toward massive death, and personnel problems and nuclear accidents.

5. The *Task Force on Sex Education and Sex Therapy,* Edward Auer, M.D., chairperson, is concentrating on the role of psychiatrists in sex education for medical and graduate students and in continuing education. A survey of directors of residency training has been carried out most successfully. The task force will meet soon in Chicago to consult with leading authorities on sex therapy.

Other concerns of the Council involve ego aspects of death and dying, programs in thanatology, the problems of the chronic mental patient, antipsychiatry groups, the problem of "boundaries" in psychiatry, the impact of *DSM-III* on clinicians and private practitioners, and problems in polarization of the membership.

The Council on Internal Organization

ROBERT O. PASNAU, M.D., CHAIRPERSON

THE COUNCIL met for two and a half days during the September 1978 committee meetings, heard reports from many of its 29 components, and discussed a wide variety of topics. The Council is charged with "all those matters involved in the organizational structure of the Association not covered by Consitutional Committees." It oversees an enormous range of activities including the annual meeting and its programs; advertising in publications; membership benefits, including all insurance plans; prizes and awards; the House Committee, which oversees all elements of the Central Office buildings; the Hospital & Community Psychiatry Service, journal, and Institute; the Committee to Review *Psychiatric News*; and the Committee of Residents. The Council also serves as an overseeing body for the APA staff and the other councils, making certain that they function efficiently and have the support necessary to accomplish their tasks. The highlights of the actions that took place during the September Council meeting will be described in this report.

Morris A. Lipton, Ph.D., M.D., chairperson of the *Task Force to Study the Impact of Potential Loss of Pharmaceutical Support,* announced that his task force had come to some conclusions. It was noted that the net income to the APA from the pharmaceutical industry is about one-half million dollars annually. Most of this amount (approximately $20 per member per year) is derived from advertising in the publications and exhibits at the meetings. Only about $15,000

comes in the form of direct grants and support, mostly for the named lectureships at the annual meeting. The task force recommended "vigilance rather than divestiture." While noting that there is great division in the membership as to the ethical issues involved in the relationship between medical associations and the pharmaceutical industry, the task force recommended that APA should not discontinue pharmaceutical advertising in the publications nor exhibiting at the scientific meetings. However, APA should begin a dialogue at once between the leaders of the pharmaceutical industry and the officers of APA to work toward the elimination of named lectures and funds identified with individual firms; instead, there should be a listing of supporting sponsors in the programs at the meetings. The task force emphasized that this should not be done immediately but only after careful consultation and advice from industry. The Advertising Committee was praised for its pioneering work in monitoring APA advertising, and it was suggested that the committee's vigilance continue in the future. Finally, the task force recommended that district branches and areas follow the guidelines set down by the Board of Trustees. The Council voted to receive the task force report and to present it to the Board of Trustees for action.

Chester Trent, M.D., chairperson, reported on the work accomplished by the *Committee on Professional Liability Insurance*. There has been an improvement during the past year with respect to losses among psychiatrists compared with other specialty groups, and the overall premium rate will increase by not more than 1% this year, compared with 10% or more in recent years. The committee also had considered the problem of claims against psychiatrists involving sexual activity, in accordance with previous instructions from the Board of Trustees, and has developed an exclusion for such coverage. This matter was sent to the Board of Trustees for full discussion of the APA policy and position on this matter.

Christopher T. Bever, M.D., chairperson of the *House Committee*, is also chairperson of the newly established *Building Committee*, which consists of members of the House Committee and staff. The Building Committee was established by the Board of Trustees in May 1978 and is charged with overseeing the purchase of property and construction of APA's new Central Office building. Dr. Bever reported that the committee has been meeting regularly with the architect and others concerned to discuss progress and problems. His report included selection of the architect, purchase of the real property, zoning problems, design of the building, planning of space allocation within the building, parking facilities, and various other aspects of the committee's work.

Charles B. Wilkinson, M.D., chairperson of the *Committee on Grants and Awards*, informed the Council that the Manfred S. Guttmacher Award continues to be in financial difficulty, in spite of the additional funds transferred to its account. The Committee on Grants and Awards recommended that the Guttmacher Award be given only every second year, alternating with the Isaac Ray Award. The Committee on Grants and Awards also recommended that efforts continue to be made to find qualified applicants for the Marie H. Eldredge Award.

Lawrence C. Kolb, M.D., chairperson, presented the report of the *Committee to Review Psychiatric News*. The committee recommended that a membership poll be authorized to make available data on which to base recommendations to improve *Psychiatric News* and make it more attractive to its readership. The committee also asked that a legal opinion be sought as to whether limiting the amount of advertising in *Psychiatric News*, which had been suggested previously, would be found to be restraint of trade. The committee reported that it would recommend no changes with respect to *Psychiatric News* until the membership poll is completed.

The Council heard a report from staff on the advertising survey recently conducted for APA, which led to a renegotiation of APA's contract with Steven K. Herlitz, Inc., its principal advertising representative, and to termination of APA's contract with its other advertising representative, Fred Michalove. The new contract with Herlitz provides for a reduction in the commission rate paid to Herlitz and a new termination clause that is much more favorable to APA than the previous one. The Council agreed that experience with the Herlitz company during the next year would determine whether APA would continue to use the company or hire its own in-house advertising representative.

The *Committee on the Annual Meeting*, William S. Davis, M.D., chairperson, reported that it had received a request to solicit information from members as to their preferences for annual meeting sites. The Council recommended that *Psychiatric News* publish a list of cities where an APA annual meeting would be feasible, indicating advantages and disadvantages of each, and request input from members. This committee recommended to the Council that day-care facilities at annual meetings be discontinued, since they were underutilized and were not cost effective. However, the Committee on Women told the Council that they considered this program important and requested the Council to consider their proposals for a day-care program. As a result, the Council authorized the establishment of a task force to develop a day-care program, which would be expected to become self-supporting once in operation.

Allan A. Beigel, M.D., chairperson of the *Committee on Program*, reported that his committee had received many comments concerning the scheduling of the opening session, convocation, and business session at the annual meeting. Comments were that the opening session was too long, that the convocation was poorly attended, and that the business session tended to interrupt the flow of the scientific program and used space that could be available for scientific sessions. The Council voted to take this question to the Board of Trustees and to recommend that the Board establish a task force or ad hoc committee to explore the alternatives and make recommendations. The Council also recommended approval of the committee's request to schedule scientific program activities on Wednesday evening, the night of the banquet, during the 1979 annual meeting.

The Council again discussed election issues and voted to recommend that establishing election procedures and guidelines and dealing with violations thereof should be made a function of the Board of Trustees itself rather than of a separate committee. The Council recommended approval of a staff request to raise the charge to district branches participating in centralized dues billing from 50 cents per member to $1.00 per member and that a minimum charge of $50 per district branch be established. It also agreed to recommend to the Assembly that fiscal years of district branches be changed to conform to APA's fiscal year in order to facilitate their participation in centralized dues billing.

Richard G. Johnson, M.D., chairperson of the *Committee on Member Life, Accident, and Health Insurance*, reported that the limitation on inpatient psychiatric coverage that had been authorized for one year is working satisfactorily. The Council recommended approval of the committee's request

for a task force, consisting of members of the full committee, to study problems concerned with the definition of "psychiatric illness." Dr. Johnson also reported on a proposal for a national field trial of an insurance coding project, which would use a six-category code instead of *DSM-II*. APA members will be requested to participate in the field trial if their patients agree. The Council recommended establishment of a task force to handle the national field trial. Dr. Johnson also presented a draft of the final report of the *Task Force on Consideration of Problems with Psychiatric Underwriting*, of which he is chairperson. The report will be modified and augmented before being given consideration for publication.

Jack Wolford, M.D., reported to the Council on the activities of the *Committee to Coordinate the Functions of the Hospital & Community Psychiatry Service, Journal, and Institute*, which included recommendations for changes in content and style of the journal and making films and videotapes available to all members of APA, with a lower charge to those who are members of the H&CP Service than to non-Service members.

Dr. Pasnau would like to express his appreciation to Dr. Jack White, staff liaison, Mrs. Patricia Scheidemandel, administrative assistant, Malkah Notman, M.D., Lester Rudy, M.D., Richard Steinhilber, M.D., and Stanley Yolles, M.D., for their enthusiastic efforts.

The Council on International Affairs

ALFRED M. FREEDMAN, M.D., CHAIRPERSON

THE COUNCIL has had an active year, one in which communication among psychiatrists on an international scale has flourished. Joint meetings with our French colleagues were held in connection with the APA annual meeting in Atlanta. Forty-five French psychiatrists and spouses attended the meeting and several joint sessions were held. Then 350 American psychiatrists attended the meeting in Paris, France, planned by the APA task force in cooperation with the Société Medico-Psychologique. Both joint meetings were highly successful.

Our continuing concern with abuses and misuse of psychiatry and psychiatrists has led to the appointment of Drs. Joe Yamamoto, Harold Carmel, and Melvin Lipsett to a subcommittee of the Council to write a proposal about receiving and monitoring complaints of human rights abuses, working with the World Psychiatric Association's Committee on Abuse, and maintaining close and continuing follow-up.

A letter has been sent to the President of Argentina, General Videla, asking for information about psychiatrists and others who have been allegedly arrested in Argentina and held without charges. On November 22, 1978, a telegram was sent to the Supreme Court Chairman in Russia appealing for a reduction in the sentence of Alexander Podrabinek. In addition, APA is an active member of the Human Rights Workshop organized and supported by the American Association for the Advancement of Science's Committee on Scientific Freedom and Responsibility.

The *Task Force to the International Organizing Committee of the 2nd Pacific Congress of Psychiatry*, chaired by Norman Rosenzweig, M.D., is proceeding with plans to hold the 2nd Pacific Congress of Psychiatry in Manila, Philippines, May 11-16, 1980, immediately after the APA annual meeting in San Francisco. Lindbergh Sata, M.D., has agreed to head the program committee for that meeting.

The *Task Force on Transcultural Aspects of Ethnocentricity Among Psychiatrists*, Ramon Parres, M.D., chairperson, will present some of its preliminary findings from its research at the 1979 annual meeting.

The *Committee on Psychiatry and Foreign Affairs*, chaired by William Davidson, M.D., was established in December 1977 to continue the work done by previous task forces concerning foreign affairs in specific areas. The committee hopes to have a meeting of Israeli and Egyptian psychiatrists in Washington, D.C., sometime in 1979. Outside funding is being sought for this project.

APA's representative to the *Inter-American Council of Psychiatric Associations*, Reginald Lourie, M.D., is also its president this year. The Inter-American Council and Dr. Lourie are investigating ways in which satellite communication can be used for program presentations at meetings in Central and South America. Also, the translation of some of the audio tapes from APA's annual meeting into Spanish is being undertaken.

Last year the Council offered complimentary subscriptions to the *American Journal of Psychiatry* and *Psychiatric News* to member associations of the World Psychiatric Association to promote communication. To date we have had replies from 43 countries, and they are now receiving our publications regularly.

During the summer of 1978, we wrote to the chairpersons of the other APA Councils about carrying out joint projects. Many joint efforts are possible: an international meeting on the regulation of research with a special emphasis on human experimentation; an international meeting on psychiatric training standards and certification; a joint project with the Council on Children, Adolescents, and Their Families, since 1980 is the Year of the Child at the United Nations; and input from our Council to the new project on ethnicity under the auspices of the Council on National Affairs. Cooperative projects are needed, and the subjects are unlimited.

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The Council on Medical Education and Career Development

PAUL J. FINK, M.D., CHAIRPERSON

THE COUNCIL met in May and September 1978. In addition to receiving reports from the components, the Council addressed a variety of programs and issues pertaining to the medical education and career development of psychiatrists.

APA will participate in a consortium, formed under the leadership of the American College of Psychiatrists (ACP), to develop and administer a psychiatric residency in-service examination. Other organizations represented in the consortium are Ohio State University (which has developed an examination program and computerized techniques for analyzing and reporting scores), the American Association of Directors of Psychiatric Residency Training (AADPRT), and the Association for Academic Psychiatry (AAP). The American Board of Psychiatry and Neurology (ABPN) will be represented by a nonparticipant observer.

The in-service examination is a pilot project to include 50 resident training programs selected on the basis of the willingness of the total resident population to participate. The program will utilize the current content areas of the ABPN written examination. The Council underscored the differences in the objectives of the in-service training examination and the ABPN certifying examination. The former provides assessment of resident performance and assistance in remediation and serves as part of preparation for certification, and the latter provides certification of competence.

APA has authorized the Office of Education to develop a consultation system in program planning, development, implementation, and evaluation for psychiatric residency training directors. While this service will not guarantee future accreditation and/or NIMH grant funding, it will provide assistance to program directors in various aspects of educational needs. The program will be self-supporting through fees paid by the training program directors, working collaboratively with the consortium of psychiatric education organizations. The office has solicited names of psychiatrists to serve as consultants to the program from a consortium of psychiatric education organizations and the NIMH psychiatric education branch. The Council is currently developing guidelines and procedures concerning confidentiality, consultant selection process, reimbursement procedures, performance evaluation, and progress reports. It is anticipated that the program will be operational in late 1979.

The APA Board of Trustees at its May 1977 meeting charged the Council to develop guidelines and procedures for renomination of current directors for reelection to the American Board of Psychiatry and Neurology (ABPN). The Council reviewed the present process, the recommendations of Ad Hoc Committee on Implementation of the 1976 Referenda on the ABPN, and the reports from the liaisons to the ABPN.

The Council recommended that renomination for reelection should not be automatic and that the director's performance should be evaluated informally with consideration given to participation in committees and activities of the ABPN and APA, most notably the Task Force to Facilitate Communication Between the APA and the ABPN. Incumbents may also be asked to respond to questions prepared by the APA Board of Trustees. Further, the directors eligible for reelection to the ABPN should be renominated according to their performance. The Council reaffirmed its previous recommendation to suggest that the ABPN study the issue of the present system of tenure and consider a possible six-year term for directors.

In 1976, the APA Board of Trustees referred the planning and development of the next decade conference on psychiatric education to the Council. The most recent conference was held in 1975; preparatory components met in 1974. The Council, after reviewing the history of previous conferences, recommended that a steering committee, including members of the Council and other appropriate APA components and the Assembly, officers, and representatives of major psychiatric education organizations (including Canadian groups), oversee the development and implementation of the conference, with the Office of Education coordinating staff support.

The Council reviewed correspondence concerning education in drug and alcohol abuse, geriatric psychiatry, and care of chronically mentally ill patients and referred these content areas to the undergraduate, graduate, and continuing education components.

The Council noted that concerns regarding these areas extend beyond education to issues involving service delivery and research, which are the responsibility of other councils, and suggested that coordination of all deliberations and recommendations take place at the level of the Reference Committee.

The Council discussed the need for a component to address issues of manpower distribution, utilization, and deployment. There is need to study manpower issues extensively, but there is no separate component that addresses these issues exclusively. APA staff have attempted to coordinate documents, articles, information, and other data, as well as responses to regulations concerned with manpower.

The Council noted that it has the responsibility to coordinate data collection and dissemination concerning manpower and recommended the founding of a separate task force to develop guidelines for data collection on manpower issues, to review and synthesize data contained in the report of the President's Commission on Mental Health, the Graduate Medical Education National Advisory Council (GMENAC), the Council of Medical Specialty Societies (CMSS), the Mendenhall study of physician activities, and other such studies, and to advise APA on approaches to future manpower studies. Such a task force could also serve as an advisory group to professions involved with data collection on psychiatric manpower for APA.

Nancy Roeske, M.D., reported on reorganization of the Section Council of the American Medical Association (AMA) and noted that the activities of the specialty section councils have been transferred to the aegis of the medical specialty societies. The AMA has responded to a number of resolutions from the section councils and plans to sponsor a national conference on the chronic mental patient within the year.

The Council has been asked to develop a plan for liaison activities between APA and the section council. Since Dr. George Pollock, who chairs the section council, is a member of the Committee on Graduate Education and Dr. Roeske is

a member of the section council as well as this Council, both can provide direct liaison.

Nancy Roeske, M.D., APA representative to the AMA Inter-specialty Advisory Board (IAB), reported that the AMA wishes to increase its cooperation with specialty societies in areas of continuing education. As part of these expanded activities, the IAB has requested that 1) each specialty society nominate a person to be a representative to the AMA Council on Continuing Physician Education, and 2) specialty societies alert their members to the fact that the AMA desires a wide variety of specialists to participate in its scientific meetings.

The AMA has also been concerned with the complex issues involving mandatory continuing education, certification, and recertification. There are particular problems for the large number of physicians who do not have initial certification. Many states are considering the possibility of demanding participation in specialty recertification programs or successful completion of a general "recertification examination" or "relicensure examination" developed by the states as a requirement for reregistration of licensure. This could create a two-class system of certification/recertification, as well as duplicate efforts or requirements.

The Council reviewed Public Law 92-603. The regulations to implement Section 227 of the Social Security Amendments of 1972 may restrict teaching physicians, who function as supervisors of residents, from billing Medicare on a fee-for-service basis.

The Council discussed the personnel section of the President's Commission on Mental Health report and the report of the DHEW task force concerned with implementation of the recommendations contained in the President's Commission report. Staff coordinated suggestions in preparing a response. In addition, the Council and staff will continue informal communication with the Alcohol, Drug Abuse, and Mental Health Administration (ADAMHA) to follow up on these recommendations and their proposed implementation.

The guidelines for EEG of the American Electroencephalographic Society (AEEGS) were referred by the Medical Director to APA components for study and comment. Although the development of standards for diagnostic testing extends beyond the Council's charge, the Council noted several concerns including quality control and educational objectives.

The Council supported collaborative efforts with the Council on International Affairs in substantive educational areas and noted the degree of interest abroad in the entire field of psychiatric education, including evaluation of training, certification, and recertification. The Council suggested additional areas for joint efforts including international education programs, standards for educational content and program quality, evaluation of foreign medical graduate applicants prior to training, and study of education experiences of medical students in other countries who wish to enter psychiatry.

The Council is working with the Council on Mental Health Services and its Task Force on Emergency Psychiatric Care Issues to address the educational aspects of emergency psychiatric care. Dr. Gail Barton, member of the task force, has developed a set of protocols, as has the Canadian Psychiatric Association, which will be reviewed by the Council and appropriate components. The Council suggested that components include the following topics on their future agendas: 1) the availability of psychiatrists in emergency room settings to teach as well as provide service; 2) curricula in psychiatry for training emergency room physicians; 3) learning experiences for psychiatrists who work in emergency rooms; 4) standards for psychiatric services in emergency rooms of general hospitals; and 5) the role of behavioral scientists other than psychiatrists in providing emergency care.

An APA Falk Fellow represented the Council during the spring meeting of the Residency Review Committee for Psychiatry and Neurology (RRC). The Council views this as important and suggested continued representation by the senior Falk Fellow on the Council while recognizing that the degree of participation is the prerogative of the RRC.

The report of the *Commission on Certification in Administrative Psychiatry* was given by Thomas T. Tourlentes, M.D., in the absence of Dean K. Brooks, M.D., chairperson. The Commission proposed an educational retreat to continue the discussions initiated during the July 1978 meeting. The retreat will provide an opportunity to clarify the commission's purposes, objectives, role, and activities and to develop strategies for more effectively implementing the Commission's charge. The Council concurred with these plans. The Board of Trustees reversed its earlier action to terminate the Commission in May 1980. The Council recommended change in status to a permanent committee with established membership.

The Council reviewed the administrative psychiatry examination procedures. The oral examination has been conducted in hotel guest rooms, a practice that the Council believes is inappropriate. The Council recommended that the oral certifying examination be conducted in public meeting rooms or in other appropriate facilities. Since this will increase the cost, the APA staff will work with the Commission to develop cost accounting for the resulting candidate fee increases. The Commission will develop a pilot written examination to be given to candidates in May 1979. Since it is experimental and will require further evaluation, this examination will not enter into the grading of candidates at this time.

APA will cosponsor an Invitational Conference on Administrative Psychiatry in November 1978 under the auspices of the Albert Einstein College of Medicine.

Gary Tucker, M.D., chairperson of the *Committee on Graduate Education*, reported that the Residency Review Committee for Psychiatry and Neurology has prepared a draft revision of the "Guide" and "Essentials" of residency programs in psychiatry. The committee, joined by Dr. William Webb, chairperson of the RRC, discussed these documents in detail (comments will be forwarded directly to Dr. Donald Langsley, a member of the RRC coordinating the revision).

The Council discussed the PGY-1 (first postgraduate year in psychiatry). The Liaison Committee on Graduate Medical Education (LCGME) has the final authority for accrediting and developing policy concerning PGY-1 experiences in medicine as a whole. The Council felt that APA, through the Committee on Graduate Medical Education, should review the first graduate year educational experiences and make specific recommendations for psychiatry. These could be transmitted to the LCGME through the RRC and the Council on Medical Specialty Societies (CMSS) for use in LCGME deliberations.

The Council also asked the committee to discuss at its next meeting the content and process of graduate education concerning the care of the chronic mental patient, geriatric psychiatry, and education in alcohol and drug abuse.

Leroy T. Levitt, M.D., vice-chairperson of the *Subcommittee on Foreign Medical Graduates*, recommended to the Council that the various APA components on foreign

medical graduates be consolidated into one group to address the variety of issues related to foreign medical graduates, including education, manpower, career activities, deployment, and service delivery. This recommendation was supported by the Council, the Task Force on Foreign Medical Graduates, and members of the Council on National Affairs during a joint meeting. This newly proposed committee could serve as a resource body to consider matters of current and future legislation affecting foreign medical graduates, problems of acculturation, credentialing, licensure, education and training, continuing education and career development, practice setting, etc. Therefore, the Council also recommended that the Subcommittee on Foreign Medical Graduates be discontinued.

Larry Silver, M.D., chairperson of the *Committee on Medical Education*, presented a paper, "The Role of the Admissions Committee in Assessing the Mental Health of Medical School Applicants," written by the committee for proposed submission (contingent on APA approval) to the *Journal of Medical Education*. The Council requested that the committee prepare a revision of the paper for further review and comment.

The committee also discussed possible future agenda items including 1) an expansion of the study of admissions committees with a specific case example format; 2) a study of mental health issues of medical students, focusing on the mental health problems of medical students, effects on student functioning, and the preventive, supportive, and treatment resources needed by medical schools (the committee and Council considered the possibility of a task force to explore these issues in more detail and to make recommendations on the types of services needed); 3) a study of the undergraduate medical student curriculum, focusing on content areas (chronic mental patient, alcohol and drug abuse, geriatrics, and primary care); 4) a study of factors related to specialty selection by medical students, particularly in terms of understanding what might be done to increase the numbers of students entering psychiatry; and 5) a study of the benefit of the one-year fellowship in psychiatry for third- or fourth-year residents in other specialty areas.

Robert L. Leon, M.D., chairperson of the *Committee on Continuing Education*, recommended a special meeting to review continuing education requirements for APA members. The need for a more in-depth response to these members was discussed in light of the first three years' experience and their relationship to the requirements of other medical specialty societies.

Additionally, the committee noted the need for an extensive discussion of issues of credentialing and accreditation in continuing education and a review of the issues raised by the Assembly Area V Action Paper, "Mandate for the Study of Appropriate Continuing Medical Education for Psychiatrists."

The *Task Force on Self-Study Materials and Audio Tapes* and the *Task Force on Continuing Medical Education Exemptions and Exceptions* were formed during the past year. Since there is considerable overlap in the work of the two task forces and that of the Committee on Continuing Education, the Council approved a recommendation that the task forces report directly to the Committee on Continuing Education to provide integration and consolidation of efforts and present policy recommendations.

The Council endorsed the recommendation of the committee and task forces that APA should develop appropriate self-study materials for APA members for Category I. They noted that such materials will be particularly useful for disabled members, members who live in rural or isolated areas or in foreign countries, and others for whom continuing education programs are less accessible or who prefer to enrich their on-site learning with participation in self-study learning programs. The Committee on Continuing Education will be responsible for planning and developing guidelines for design, implementation, funding, and production of such materials.

The Council also endorsed the recommendation of the committee that APA not endorse the self-study learning packages or series (such as a journal club, subscription series or psychiatric update, etc.) currently being developed by various representatives of industry, noting that these materials are often superficial and must be purchased by subscription rather than by topic only. While the Council endorsed APA work with industry or outside organizations to prepare and publish in-depth self-study materials that address specific topics, APA must maintain complete control of the contents and these materials must be available to members on an individual package basis.

The Committee on Continuing Education reviewed two self-study pilot projects submitted by Audio Digest and AV/MD into Category I self-study packages. Although the committee noted that these did meet the requirements for Category I, the committee felt these proposals did not provide a sufficiently intensive study of a topic area and recommended rejection of these proposals. However, the Council instructed the committee to provide written feedback to both companies, describing the specific differences in the content areas and making suggestions for an improved format. The Office of Education staff will work with the committee and with these companies to see if appropriate learning packages can be developed on a pilot basis.

The *Task Force on Self-Study Materials and Audio Tapes*, Layton McCurdy, M.D., chairperson, reported on the development of guidelines and procedures for self-study materials, including audio tapes. To improve the quality of audio tapes, the task force proposed increased collaboration with representatives of commercial firms that tape sessions at the APA annual meeting.

Two members of the task force (including the chairperson) will meet with representatives of the APA Program Committee and the appropriate commercial audio tape firms (currently Audio Digest and AV/MD) to review the final draft of the APA Scientific Program and to select sessions to be taped at the APA annual meeting. The Office of Education staff will attend such meetings at no cost to the Council. Prior to the meeting, the task force will develop guidelines for taping sessions. An editorial review panel will be selected to assist the task force in the review of audio tapes. This panel will review tapes and materials and use a review process similar to that of the *American Journal of Psychiatry*. Consistent with the APA policy of not paying for members' participation at the annual meeting, funding will not be provided for travel expenses. Since neither honoraria nor consultation fees can be provided, the Council requested that their work be given public recognition in some way.

The *Task Force to Consider CME Exemptions and Exceptions*, Eugene Feigelson, M.D., chairperson, held its first meeting in September. To facilitate members' compliance with the APA continuing medical education requirements, the Council concurred with the task force proposals that quality self-study materials be available for APA members who do not have reasonable access to continuing medical education experiences (e.g., rural or foreign members or members who are ill, disabled, or otherwise housebound or

who prefer to use self-study materials as an adjunct to their continuing education activities).

The Council noted that the function of the continuing education requirement is to encourage members to become and remain up to date in their practice activities and concurred with the task force that Board certification provides a measure of professional competence in psychiatry and should be recognized in lieu of one portion of the continuing education requirement. Thus, the Council will recommend that 50 hours of Category I credit should be awarded for becoming certified by the American Board of Psychiatry and Neurology or the Royal College of Physicians and Surgeons—25 credits for Part I and 25 credits for Part II. In addition, psychiatrists who became APA members after July 1, 1976, have a full three years from date of membership to complete the CME requirement.

The task force studied requests for Category I credit for sabbatical activities and concluded that sabbaticals should not qualify for any special educational credits, since members on sabbaticals will have many opportunities to obtain the required Category I credit during the three-year reporting period. In addition, the task force felt that researchers and administrators can find appropriate continuing education activities in the existing categories and do not necessarily require special exemptions. The task force requested that staff carefully monitor the CME reports of the APA members residing outside of the United States and considered the possibility of recommending the use of self-study educational materials for Category I as a more suitable alternative.

The task force suggested that there should be a mechanism for reviewing appeals stemming from rejection of applications for exemptions or exceptions from the CME requirements and recommended that such appeals be processed by the Committee on Continuing Education. Staff were requested to monitor and report data on reported Category VI activities (meritorious learning experiences) to assist the task force in developing standards of acceptability and quality control.

The *Committee on Psychiatry and Primary Care Education,* F. Patrick McKegney, M.D., chairperson, discussed the recent emphasis on increased primary care education for medical students in, among other documents, the Report of the Institute of Medicine (IOM). The committee felt that this emphasis provided a major opportunity for American psychiatry but expressed concern that there was no psychiatrist on the IOM committee authoring that report.

The committee recommended that a policy statement stressing that medical student curriculum include consultation-liaison psychiatry be transmitted to the appropriate national medical-psychiatric education organizations. Similarly, the committee discussed the recent emphasis on an increased need for psychiatrists who relate to primary care medicine as described in the ADAMHA Manpower Task Force Report. The Council concurred with the committee that a policy statement stressing that all psychiatric residency programs include significant, supervised experience in which the resident functions as a consultation-liaison psychiatrist in an ambulatory care setting should be transmitted to the appropriate national organizations.

The committee discussed APA's role in fostering the career development of psychiatric residents in working with primary care physicians and recommended to the Council that a fellowship program similar to the development of psychiatrists having a major commitment to teaching patient care in primary care settings be developed. These fellowships would be given to residents in psychiatry and primary care fields for a two-year period. The educational program would include at least one meeting yearly of at least two days' duration and a funded faculty of primary care physicians and consultation-liaison psychiatrists.

The Council endorsed the concept of such a program and requested that the Committee on Psychiatry and Primary Care Education explore methods of development and implementation for the next Council meeting.

The committee has a variety of continuing liaison activities with APA components, including the Committee on Syllabus for PKSAP-IV, the Committee on Graduate Education, the Task Force on Psychiatric Emergency Care, and the Committee on the Chronic Mental Patient, as well as with such other organizations as the American Academy of Family Physicians. The committee also heard from the chairperson of the Committee on Liaison with the American College of Physicians, who reported that liaison with the American College of Physicians (ACP) has not been optimal. The Council and the committee encouraged CMSS delegates to work informally with the ACP and encouraged the Medical Director's office to continue active contact with the Executive Director of the ACP. Staff also was instructed to continue follow-up work on the liaison activities with the American Academy of Family Physicians.

The Council charged the committee to include in future deliberations discussion about education concerning chronic mental patients, geriatric patients, and drug and alcohol abuse.

The *Task Force on Recertification,* Herbert Pardes, M.D., chairperson, reported on preliminary plans for an intricate system that deals with the process of recertification and utilizes multiple approaches and methodologies. An extensive discussion ensued concerning the relationship of this task force to the American Board of Psychiatry and Neurology (ABPN) and its Subcommittee on Recertification in Psychiatry, the American Board of Medical Specialties (ABMS), the APA Assembly of District Branches, and other groups.

The Council postponed action on the preliminary plans and requested the task force chairperson to discuss these directly with the ABPN subcommittee and focus on the relationship to the guidelines and standards for recertification of the ABMS.

The task force plans to maintain dialogue with appropriate components of the Association through a variety of activities at the 1979 annual meeting, including the development of an issue workshop, preparation of materials on recertification to be incorporated with the Office of Education exhibit, and participation in the meetings of continuing education coordinators, district branch presidents-elect, and district branch newsletter editors, as well as preparation of articles for *Psychiatric News* and the district branch newsletters.

The *Task Force to Facilitate Communication Between APA and the ABPN,* Chester Pierce, M.D., chairperson, reported action items for the American Psychiatric Association as well as for the American Board of Psychiatry and Neurology. The Council noted that the total report reflected the substantial recent move forward in the relationship between APA and the ABPN. Among the topics included for review by the ABPN are matters related to the quality of examiners, the concept of regional examinations, feedback from examiners and candidates concerning their experiences in, and feelings about, the examination process, and a possible additional yearly meeting of ABPN, APA, and other relevant national organizations to encourage dialogue about the activities of the ABPN.

Lindbergh Sata, M.D., submitted a request on behalf of

the Committee on Asian-American Psychiatrists to develop a study of the performance of foreign medical graduate psychiatrists compared with American medical graduate psychiatrists on the ABPN examination. The study would also consider location of residency training (for example, university-based compared with other training facilities) and other factors influencing the quality of graduate education. The Council endorsed the task force recommendation that Dr. Sata meet with Dr. Donald Langsley (chairperson of the ABPN Research and Development Committee) to explore the possibility and methodology of the study.

The task force recommended that APA designate a facilitator to expedite APA members' complaints or problems regarding the ABPN. The Council was sympathetic to the concept and suggested that APA encourage members to communicate with APA but noted that designation of an official facilitator could appear to interfere with the autonomy of the ABPN, particularly if the facilitator were given responsibility for specific follow-up actions. Both the task force and the Council complimented Dr. Lester Rudy, the Executive Director of the ABPN, on the high quality and quantity of communication with candidates and encouraged APA staff to continue to respond to correspondence directly, as well as to communicate with the task force on concerns raised by APA members.

The task force asked the Council to determine what APA might do to encourage more APA members to become Board certified. The Council requested that the chairperson write to the Speaker of the Assembly to inquire how the Council can assist the Assembly on this issue.

The Council congratulated Dr. Pierce on the progress of the task force and thanked him for his excellent efforts. The Council noted with regret Dr. Pierce's retirement from the ABPN when his second term is completed (December 31, 1978) and wished him well in future activities. Dr. Marc Hollender will assume position as chairperson of the task force on January 1, 1979.

Robert Seidenberg, M.D., chairperson of the *Task Force on the Curriculum of Psychology of Women and Men,* reported on its progress and presented an outline of the proposed curriculum, indicating that the current plan was more extensive than orginally intended and that the project may result in a detailed monograph. The Council reviewed the outlines of the curriculum and the monograph, as well as the timetable for completing the project, and expressed some concern over the proposal, noting that this might be an overwhelming task which could not be completed within a reasonable time. The Council requested that the task force prepare several sample chapters of the monograph for its review. Drs. Roeske and Oken agreed to serve as Council liaisons to the task force and to review the draft materials in advance of the spring Council meeting.

The *Ad Hoc Committee for the Implementation of the 1976 Referenda on Psychiatric Certification,* Leigh Roberts, M.D., chairperson, presented the preliminary data gathered from the Membership Survey on Board Certification.

The data analysis on approximately 1,500 charts of tabulations and cross-tabulations is not complete. The Council voiced concern about the storage and utilization of this material by APA components and external groups, noting that issues of confidentiality, legal and financial implications, and staff requirements to maintain these data should be considered in determining policy for its utilization. The Council requested that the Office of Education prepare guidelines for internal and external utilization of these materials for review.

The Council noted that the preliminary data may be very useful to the American Board of Psychiatry and Neurology (ABPN) during its November policy meeting and requested that the Executive Committee during its September 9-10, 1978, meeting approve release of the preliminary data (consisting of frequency counts and percentages) to the ABPN for internal use only.

The ad hoc committee presented an outline for a proposed special session during the APA annual meeting in Chicago in May 1979. The Council endorsed the program and suggested that the committee consider the inclusion of a discussant representing the public to address concerns about the certification process, the adequacy of uncertified physicians, as well as provide an additional perspective to the session. The ad hoc committee expects to complete its work by May 1979.

The Council on Mental Health Services

J. M. STUBBLEBINE, M.D., CHAIRPERSON

THE COUNCIL has been noted for being busy, and the year 1978 set a high mark for activity and productiveness. As a result there have been some changes in the structure of the Council, with several new committees and task forces and charges. Because of a palpably diminished interest in or pressure around national health insurance, the Council was able to devote a good bit of attention to things other than simply the financing of mental health services.

The most interesting development is a Task Force on Aging that has been appointed by the Council on the advice and with the consent of two other councils. There appears to be considerable sentiment within APA for the evolution of a separate council on aging. It was felt that the quickest way to begin was to appoint a task force that would make a statement regarding the appropriateness of a committee or council structure within APA. The task force, chaired by Russell Barton, M.D., has prepared proposals for presentation to the Reference Committee in February 1979.

After searching consideration and analysis the Reference Committee approved the creation of a Committee on Rehabilitation. This has been approved by the Board, and Bertram Pepper, M.D., is chairperson. It promises to be a very active committee and will put APA in contact with many important organizations and federal agencies. It is expected

that the committee will also be making presentations to the APA membership that will be of clinical importance. One possible topic is "The Disability Process," in which psychological antecedents to accidents can be seen, identified, and sometimes dealt with before an accident or disability occurs. There is also a belief that a series of psychic states accompanies any physical disability, and knowledge of such states by the psychiatrist will allow him or her to be of greater help to physically disabled patients.

Another area of much interest and importance is the field of community mental health programs. The Council appointed a Task Force on Community Mental Health Programs to work with a similar component in the Assembly. This task force met, reviewed its charge, and is embarking first on liaison with the Assembly task force and after that with important governmental agencies and personnel in Washington. The focus of these meetings is to begin to collect data on the process of quality determination and evaluation of function of the community mental health centers. It will focus especially on the role and function of the psychiatrist in community settings.

With the termination of the Task Force on Substance Abuse in the Council on Research and Development and with stimulation from many sources within APA, a new Task Force on Substance Abuse was formed, a charge from the Council was drafted, and a report is expected to be made by the task force one month before the Council's fall meetings in 1979. The charge is to address itself not just to alcoholism but other substances of abuse and to determine APA's and individual psychiatrists' proper roles with regard to these vexing major social problems.

Yet another area of progress within the Council has been the emergence of a *Task Force on Psychiatric Emergency Care Issues,* Gail Barton, M.D., chairperson. This task force has been very active for well over a year in maintaining APA liaison with the AMA and emergency room physicians. The task force has spent most of its energy and attention assisting in the development of a curriculum and accreditation mechanism for emergency paramedic training programs. In December 1978, the Board of Trustees agreed to be one of seven sponsoring organizations in the ensuing accreditation programs.

The *Committee on Financing Mental Health Care,* Jay S. Reibel, M.D., chairperson, is studying the implications of mental health benefit packages of various levels that may be offered in proposed bills on national health insurance. The members wish to see an up-to-date utilization and cost data base developed from which there can be good projections of the cost of any proposal. The committee is working in particular with APA's consultant health economist, Mr. John Krizay, and the Joint Commission on Government Relations.

The *Committee on Peer Review,* Henry Altman, M.D., chairperson, continued its difficult and demanding work on a revision of the *Manual on Psychiatric Peer Review,* which will include, in addition to revisions of current material, the report of the Task Force on ECT, the new Federal Employees Program (FEP) guidelines, the psychopharmacology criteria sets, perhaps something from the CHAMPUS guidelines, material from the (non-APA) Committee on Adolescents and the Long-Term Hospital Patient Committee. In addition, there may be revisions submitted by the American Psychoanalytic Association and the American Academy of Child Psychiatry. Even though the current criteria sets are recognized by many as too general, the committee has decided not to change the criteria sets at this time, expecting that a more complete revision will be necessary when *DSM-III* is finalized.

A task force of the APA Commission on Standards of Practice and Third-Party Payment assisted the Blue Cross/Blue Shield Federal Employees Program to revise their claims review guidelines. Mr. Richard Leuhrs, BC/BS FEP Program Director, reported in September to the Committee on Peer Review that the revised guidelines had been distributed to about 70 participating Blue Cross plans in early August. Mr. Luehrs expressed appreciation for APA's assistance and emphasized the importance of liaison between district branch peer review committees and local Blue Cross/Blue Shield plans.

Dr. O. B. Towery, ADAMHA, attended the committee's meeting and reported that DHEW has given a contract to the American Association of PSROs for the purpose of revising criteria sets developed by the various specialty societies that were contracted by AMA to do such work two years ago. AAPSRO will be in touch with about six or eight specialty societies, including psychiatry, to revise their criteria sets. Dr. Towery reported that last year there was some question about the viability of the PSRO system, but at this point its future appears healthy. The APA has suggested several members to serve as consultants to this project.

The *Committee on Federal Government Health Services,* Col. John R. Rogers, M.D., chairperson, is concerned about the quality of service and retention of psychiatrists in the Veterans Administration and other federal agencies. The Council expects to receive a detailed report, with suggestions for appropriate action, sometime during 1979.

As requested by the CPHA and authorized by the Board, the *Task Force to Consult with the Commission on Professional and Hospital Activities,* Henry Pinsker, M.D., chairperson, has provided psychiatric consultation to the Commission and assisted in ensuring inclusion of some psychiatric diagnoses and minimal criteria in the present program. The task force hopes to be involved in the development of the next Quality Assurance Monitor Program.

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The Council on National Affairs

FRANK M. OCHBERG, M.D., CHAIRPERSON

HAROLD VISOTSKY, M.D., retired as chairperson after three outstanding years. The principal activities and accomplishments of this Council continue to occur within the several committees and task forces. Three new task forces were formed: the Task Force on Psychiatric Aspects of Terrorism; the Task Force on Gay, Lesbian, and Bisexual Issues; the Task Force on Multi-Ethnicity and Cultural Psychiatry. Plans to transfer the Committee on Confidentiality to a new Council on Law and Psychiatry were approved. The Committee on Comprehensive Health Planning has designed a task force, funded for action in 1979, to educate district branches. Thus there have been several significant changes in the content of Council activity, but there has also been continuity of effort addressing the needs and concerns of minority groups and women in psychiatry.

Several new procedures have been instituted. All voting members are assigned to assist two or three components and to review and approve budget requests. Falk Fellows and APA/NIMH Fellows are assigned to and assist Council members in the supervision of components. Fellows may also work intensively with one of the Council components. A caucus of minority and women committee chairpersons will meet during the fall committee meetings and again during the annual meeting. The observer-consultant will focus attention on this caucus and the cross-cutting issues that affect the minority committees.

The caucus of minority committees consists of the chairpersons of the minority committees, the Committee on Women, chairperson and vice-chairperson of the Council, the APA Deputy Medical Director for Minority Affairs, and the observer-consultant to the Council. It is the intent of this caucus to identify issues of concern to the minority components and to advance these issues through collaborative action. The following issues were highlighted:

The *Committee on Spanish-Speaking Psychiatrists* expressed its concern about the Bach-y-Rita case, which involved a charge of discrimination in relation to a faculty appointment. The Council asked the Executive Committee to request that the Commission on Judicial Action review the facts and consider the possibility of entering the case as amicus curiae. Members of this caucus agreed as individuals to be available to form a support group. Action was to be deferred pending communication and approval of Dr. Bach-y-Rita and his lawyer.

Believing that there may be serious slippage in the momentum of affirmative action programs, particularly in medical schools as a result of the Bakke case, the caucus asked the Deputy Medical Director for Minority Affairs to collaborate with her counterparts at the Association of American Medical Colleges and collect and distribute statistics noting the trend in admissions of minority students and women to American medical schools. The caucus will review the statistics and suggest future actions.

Members of the caucus are concerned about the possible demise of the APA/NIMH Fellowship Program, which they view as an erosion of affirmative action in the Association. The caucus recommended APA support for this program, regardless of the availability of federal funds. They also recommend a letter-writing campaign by chairpersons of departments of psychiatry and relevant APA components to the Administrator of the Alcohol, Drug Abuse, and Mental Health Administration and the Director of NIMH, promoting the continuation of this fellowship program.

The caucus encourages the appointment of highly qualified women and minority members to APA components and will identify such individuals to the President-Elect.

The *Committee on Indian Affairs,* Johanna Clevenger, M.D., chairperson, assisted in publication of the *White Cloud Journal,* which will include an abridgement of an exhaustive survey of Indian health services. The committee also planned a field trip to the Chicago Urban Indian Center to coincide with the annual meeting and located a full component of American Indian members and focused on concerns of Indian psychiatrists rather than issues in psychiatry affecting Indians. The Committee on Indian Affairs will now be called the Committee of American Indian and Alaskan Native Psychiatrists.

The *Committee on Asian-American Psychiatrists,* Lindbergh S. Sata, M.D., chairperson, initiated an in-depth survey of Asian women psychiatrists. The project is currently awaiting approval from the Council on Research and Development. The coordinator of the project is Dr. Elizabeth Chan Small from Boston. The committee also participated in the planning of a project to develop a culturally relevant minority psychiatric curriculum. This project, headed by Dr. Albert Gaw from Boston, has been approved for funding by NIMH. An analysis of Asian psychiatric performance on specialty certification with the American Board of Psychiatry and Neurology is being coordinated by Dr. Sata. The committee arranged to house the Asian-American Psychiatric Newsletter in the Menninger Foundation. It will be coedited by Dr. Normund Wong (Topeka, Kans.) and Dr. Luke Kim (Davis, Calif.), with publication scheduled for 1979.

The *Committee of Black Psychiatrists,* Mae McMillan, M.D., chairperson, continued to address itself to the issues of mental health care for the black populations. Major educational efforts for 1978 included the development of the Solomon Carter Fuller Lecture and a special session, "Mental Health Care of Black Americans," which were held at the annual meeting. Dr. Jacqueline J. Jackson, a medical sociologist, the recipient of the Fuller Award, delivered the lecture, "Epidemiological Aspects of Mental Illness Among Aged Black Women and Men." The committee generated a position statement on affirmative action and one on the Joint Conference Committee. The latter is in substantial agreement with the statement of the Massachusetts District Branch. Other significant issues addressed included 1) minority recruitment for medicine and the specialty of psychiatry, and 2) the development of a Division of Minority Mental Health Programs in NIMH. The committee has established active liaison with other APA components and the Black Psychiatrists of America. These efforts yielded significant contributions to the President's Commission on Mental Health, the development of a proposal for the Academy on Issues in Psychiatry for Black Populations, and activities related to support for the ratification of the Equal Rights Amendment. The goal of previous efforts of the committee was recognized in the appointment of a committee to survey

psychiatry services for black South Africans and the actual survey conducted by the appointed group. This activity stemmed from concerns that the Committee of Black Psychiatrists forwarded to APA's Board of Trustees, concerns that have come to be known as the "Bradshaw Memorandum."

Plans for the 1979 annual meeting call for the delivery of the Solomon Carter Fuller Lecture by Eleanor Holmes Norton, Director of the Equal Employment Opportunity Comton, Director of the Equal Employment Opportunity Commission, and an evening panel on politicization of psychiatry.

The *Committee on Spanish-Speaking Psychiatrists*, Angel Gregorio Gomez, M.D., chairperson, obtained a new census of 2,500 Hispanic psychiatrists, encouraged Hispanic psychiatrists to participate actively within their district branches in order to promote organized voting power, joined with other minority committees in the Council to obtain fair representation in the Assembly, moved for a commitment from the Council and APA in the Bach-y-Rita case, anticipating similar cases to be faced in the near future, participated in the planning of a workshop to develop a model curriculum on Cultural Issues in Psychiatric Training for U.S. psychiatric residency training programs, and joined efforts with ASPIRA, COSSMHO (Coalition of Spanish-Speaking Mental Health Organizations), and MALDEF (Mexican American Legal Defense and Educational Fund, Inc.) in order to help the Hispanic Americans to meet their most pressing needs.

The committee's plans for the 1979 APA annual meeting in Chicago include a special session titled "Psychiatry: Relevant for Hispanics in the 80's?" an issue workshop titled "The Spanish-Speaking Women Psychiatrists: A Dual Minority," and the 1979 Simon Bolivar Lecture to be given by Dr. Karl Menninger.

The *Committee on Women*, Elaine Hilberman, M.D., chairperson, continued efforts to mobilize active APA support for ERA ratification and to recommend educational strategies that sensitize the membership to the psychological consequences of sexual inequality. Then, it met to organize a broad educational program about women for the Chicago meeting and initiated efforts that yielded a revision of APA's position statement on abortion and drafted new positions on child sexual abuse and patient package inserts. The committee recommended that APA participate in ERA Mail Day, child-care at annual meetings be continued with a new coordinating structure, the new component on substance abuse include women participants and a focus on substance abuse by women, the report on the chronic mental patient include a family inpact statement, and all proposals concerning insurance coverage for mental disorders be evaluated for possible sex bias against women as providers and consumers of mental health services. Concerns about the representation of papers by and about women and the absence of a blind reviewing procedure in the *American Journal of Psychiatry* continue as unresolved issues. Pending issues include women and substance abuse, home births, elderly women, and a nonsexist language policy for APA journals and scientific meetings.

The *Committee on Comprehensive Health Planning*, Howard Gurevitz, M.D., chairperson, in cooperation with the APA Division on Government Relations and Office of Comprehensive Health Planning, prepared testimony for hearings on the renewal of Public Law 93-641 (the Health Planning and Resources Development Act), prepared formal comments on numerous DHEW regulatory proposals, and coordinated legislative/regulatory activities with other major professional associations. The committee sponsored/conducted a session at the 1978 annual meeting in Atlanta—"Psychiatric Participation in Mental Health Planning." A chronic mental patient policy statement, "A Call to Action for the Chronic Mental Patient," was sent to the committee's district branch liaison for use in review of the long-term care component of Health Systems Agencies plans, and a health planning workshop was conducted for Area II in conjunction with the Area II Tenth Biennial Meeting in New York City. The committee also developed and submitted a proposal for a workshop on current issues in mental health planning for the APA 1979 annual meeting in Chicago and maintained a liaison network with APA district branches concerning health planning matters.

The *Committee on Confidentiality*, Jerome S. Beigler, M.D., chairperson, prepared a Model Law on Confidentiality that has been accepted by APA (see the January 1979 issue of the *Journal*). Other activities of the committee included giving testimony before congressional subcommittees on various confidentiality-related issues, consulting on *DSM-III*, which resulted in Axes IV and V being used only for educational and research purposes, consulting on the CHAMPUS Peer Review project and registering strong objections about certain confidentiality aspects, preparing a statement on the Tarasoff decision, working in cooperation with Jay Cutler and the Joint Commission on Government Relations regarding various confidentiality-related issues in Congress and government agencies, and coordinating activities and legislative interventions with the National Commission on the Confidentiality of Health Records.

The *Task Force on Manpower and Foreign Medical Graduates*, Leroy P. Levitt, M.D., chairperson, studied the impact of Public Law 84-484 on foreign medical graduates in psychiatry and, in conjunction with the Subcommittee on Foreign Medical Graduates within the Council on Medical Education, developed a plan for a Committee on Foreign Medical Graduates. This plan has been approved by the Council and forwarded to the Reference Committee.

The *Task Force on Ecopsychiatric Data Base*, Jay Shurley, M.D., chairperson, after five years concluded an exhaustive literature review, produced an annotated bibliography, and submitted a final report. The final products have been approved by the Reference Committee for publication and sale to the membership.

The *Task Force on Psychiatric Aspects of Terrorism*, Elissa Benedek, M.D., chairperson, identified ethical concerns for psychiatrists who collaborate with law enforcement agencies that respond to terrorist events, identified researchable issues relating to victims of hostage incidents, sought and received a request for collaboration from the Research Committee of the Cabinet Committee for Combating Terrorism, and requested and received approval for outside funding from this group.

The *Task Force on Gay, Lesbian, and Bisexual Issues*, James A. Paulsen, M.D., chairperson, designed a questionnaire based on a similar instrument prepared by the American Psychological Association that, after approval by the Council on Research and Development, can be used to assess the attitudes of APA membership to training, research, and clinical work affecting the purview of this task force.

The *Task Force on Multiethnicity and Cultural Psychiatry*, John P. Spiegel, M.D., chairperson, planned its first meeting to explore tentative goals, which include assembling curricula and other educational materials from existing multiethnic training programs in order to prepare a model training program.

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The Council on Professions and Associations

WILLIAM R. SORUM, CHAIRPERSON

DISCUSSION AND ACTIONS took place at three Council meetings in 1978—at the annual meeting in May, at a special meeting on June 24–25, and at a meeting on September 7–8. The Reference Committee meetings in June and December also involved the Council.

Many activities included in or implied by the charge to this Council have been assumed or duplicated in recent years by other components, liaison persons, APA staff, or combinations thereof. In the fall of 1977, therefore, the Council decided to make an in-depth review of its functions and to consider options for redirecting its efforts and/or eliminating redundant activities. During the 1978 discussions, two views developed: 1) that the Council was anachronistic, no longer cost-effective, and should be abolished, with its functions being distributed to other staff and membership groups in APA; and 2) that the Council should continue, with a revised charge that would emphasize active monitoring of and liaison with other organizations important to psychiatry and mental health. In support of the latter view, the Council made an inventory of such organizations and drafted a new charge:

> It is the responsibility of the Council on Professions and Associations to monitor and report on the activities of the many associations, organizations and agencies engaged in the common effort to advance the mental health of citizens or whose sphere of activities may have significance for psychiatric patients and psychiatrists. In addition, where appropriate, the Council may maintain liaison with organizations or may monitor the liaison activities of other components of the APA. This includes, but is not limited to, the areas of psychology, social work, nursing, religion, rehabilitation, regulatory agencies and certain social agencies.
>
> The Council's function is to develop recommendations for the APA's position in relation to these organizations and issues that pertain to them.

In its June and December meetings, the Reference Committee took the position that a decision on the future of the Council should be made following a review of all APA components—to begin in February 1979—which will be made by the Board of Trustees' Ad Hoc Committee to Study Councils and Components.[1] Specifically, the Reference Committee delayed approval of the revised charge for the Council until a full discussion of liaison activities is held in February 1979, set aside a suggestion by the Council that there be a task force to study the quality and effectiveness of liaison activities, and recommended against assigning the Ad Hoc Committee on Interprofessional Services to the Council, pending a determination of the Council's future.

The *Committee on Liaison with the American College of Physicians* will present several proposals to the Executive Director of the American College of Physicians. Depending upon the outcome, future liaison may take place at a component or staff level.

The *Committee on Liaison with the American Hospital Association* recommended support from APA for an amendment, prepared by the American Hospital Association, to the Community Mental Health Centers Act.

The Council recommended development of liaison with the American Anthropological Association and also recommended that staff explore the possibility of participation in the National Coalition for Disease Prevention and Environmental Health. APA has received a number of commendations for reestablishing a Committee on Psychiatry and Religion that will focus on liaison activities.

The *Task Force to Formulate Guidelines for Psychiatrists Working with Paraprofessionals* presented a draft report to the Council and the Assembly, and comments have been received from a number of district branches and APA components; the proposed guidelines are being revised and will be resubmitted to the Council.

A draft statement on criteria for involuntary hospitalization was prepared by the *Committee on Psychiatry and the Law*. An ad hoc committee, comprised of representatives of the Commission on Judicial Action, the Committee on Psychiatry and the Law, and the Joint Commission on Government Relations is presently making additions and revisions to the statement. It is hoped that this much-needed document will be ready for approval by the Assembly and Board of Trustees in 1979.

The key consideration now is the outcome of discussions of the Ad Hoc Committee to Study Councils and Components and the Reference Committee; both committees will review the scope and functions of the Council on Professions and Associations along with those of other APA components.

[1] At its February 2, 1979, meeting the Reference Committee recommended that the Council on Professions and Associations be dissolved. The Board of Trustees, at its February 23, 1979, meeting terminated the Council with thanks.

The Council on Research and Development

LESTER GRINSPOON, M.D., CHAIRPERSON

THE COUNCIL met three times during 1978—in April, during the annual meeting, and during the September committee meetings. During the year various Falk Fellows and NIMH Fellows attended meetings of the Council.

Following the recommendation of the Council and the Reference Committee, the Board of Trustees accepted the report on confidentiality by Drs. Jacob J. Lindenthal and Claudewell S. Thomas. It was suggested that Drs. Lindenthal and Thomas publish the full report, which is quite lengthy, and that they submit a 20-page abstract of the report for consideration for publication in the *American Journal of Psychiatry*. Because the Council continues to believe that there is a need for ethical guidelines in regard to role conflict, it voted to recommend that the Task Force on Abuse and Misuse of Psychiatry in the United States further consider this issue. The Reference Committee at its fall meeting approved this recommendation.

After reviewing its work for another year, the Council decided that the Committee on Substance Abuse was not appropriately placed in the Council on Research and Development. It was therefore recommended that this committee be dismissed with thanks and that the Reference Committee consider whether it might be wise to organize some kind of component on drug abuse and place it in a different council. During the spring meeting, the Reference Committee created the Task Force on Substance Abuse and placed it within the Council on Mental Health Services.

The *Task Force on Biofeedback,* Martin T. Orne, M.D., chairperson, has nearly completed its review of research results on biofeedback. This report, which will pay particular attention to clinical applications of biofeedback, will be completed in time for the spring meeting of the Council.

The *Task Force on Electroconvulsive Therapy,* Fred H. Frankel, M.D., chairperson, completed the revision of its report after it was considered by the Council, the Assembly, and a group of ECT experts from this country and abroad. The Board of Trustees, after considering the final report at its meeting in Atlanta in May, accepted it for publication. It is now available from the APA Publications Office as Task Force Report 14.

The *Task Force on Research on Aging,* Carl Eisdorfer, Ph.D., M.D., and Lissy F. Jarvik, Ph.D., M.D., chairpersons, has revised the preliminary draft of its report and a final draft should be available to the Council by the time of its spring meeting.

The *Task Force on Late Neurological Effects of Antipsychotic Drugs (Tardive Dyskinesia),* Ross J. Baldessarini, M.D., chairperson, is completing a draft of its report, which will deal with the current state of knowledge regarding etiology, incidence, prevalence, diagnosis, treatment, and prevention of these disorders. The report will be ready for the Council's consideration by the time of the spring meeting in Chicago.

The *Task Force on Nomenclature and Statistics,* Robert L. Spitzer, M.D., chairperson, continued its work toward the completion of *DSM-III.* In addition to making revisions of the *DSM-III* draft, the task force conducted a field study to attempt to determine the degree of acceptance of and the kinds of problems generated by *DSM-III* among a sample of those using the new nomenclature in the field. While the Council was aware of the existing bias in the sample, it was encouraged by the results of this survey.

Because of a crescendo of criticism of *DSM-III,* particularly for its paucity of psychodynamic material, it was decided to hold a one-day meeting during which members of the task force, the Council, and the Ad Hoc Committee of the American Psychoanalytic Association on *DSM-III* would exchange views. The whole day was devoted to this on September 8, 1978, and a better understanding of the issues and the differences was arrived at. At the conclusion of this meeting, the Council instructed the task force to emphasize the tentative nature of the diagnostic criteria and their potential for abuse in their discussion of the Diagnostic Criteria Section of *DSM-III.* In addition, it was recommended that the wording of part of Chapter II of the January draft be changed so that a diagnosis may be made in the absence of criteria; thus, the ultimate decision regarding any diagnosis is left to the discretion of the treating physician. Furthermore, the Council moved to endorse the compromise with the Task Force on Confidentiality regarding Axes IV (Psychosocial Stressors) and V (Highest Level of Adaptive Functioning); these axes will be considered supplemental, i.e., to be used for purposes of research and education. The Reference Committee during its November meeting further voted to direct the task force to include a 20-page (approximate) appendix that would be aimed at helping psychoanalysts and psychodynamically oriented psychotherapists use *DSM-III.* The timetable for *DSM-III* is such that approval by the Council, the Assembly, and the Board of Trustees should be completed by June 1979.

The *Task Force on Guidelines for the Protection of Human Subjects in Psychiatric Research.* Donald F. Klein, M.D., chairperson, in addition to developing the guidelines, has devoted a great deal of time and energy to monitoring new government regulations of psychiatric research. It has formulated responses to a number of recent DHEW research guidelines because the Council believes that this growing emphasis on regulation will have a chilling effect on psychiatric research. The Council agreed to recommend to the Reference Committee that some kind of standing component be created to monitor this activity and to provide input to governmental bodies developing legislation and regulations concerning psychiatric research.

The Council, believing that it was time for the American Psychiatric Association to join other professional organizations in supporting a policy of decriminalization of marijuana, developed a decriminalization statement to take the place of the 1969 Position Statement on Marijuana. The new position statement was accepted by the Reference Committee and by the Board of Trustees and, with some modifications, by the Assembly.

In response to the request from Dr. Michael Gluck of the Food and Drug Administration that APA undertake the development of safety and performance standards for ECT devices, the Council voted to create a task force for the development of these standards, the *Task Force for the Development of a Safety and Performance Standard for ECT Devices.* Richard D. Weiner, M.D., Ph.D., has accepted the

task force chair, and the task force has begun to develop these standards.

Over the course of the year, the Council reviewed six questionnaires that were to be sent out to the membership, approving some and disapproving others.

The Council was requested to reestablish the Task Force on Lithium Therapy in view of recent findings indicating nephrotoxicity of lithium. However, the Council felt that while studies in this area should be encouraged, there are not enough data at the present time to justify the establishment of a task force. The Council agreed that it would reconsider the issue at its fall meeting in 1979.

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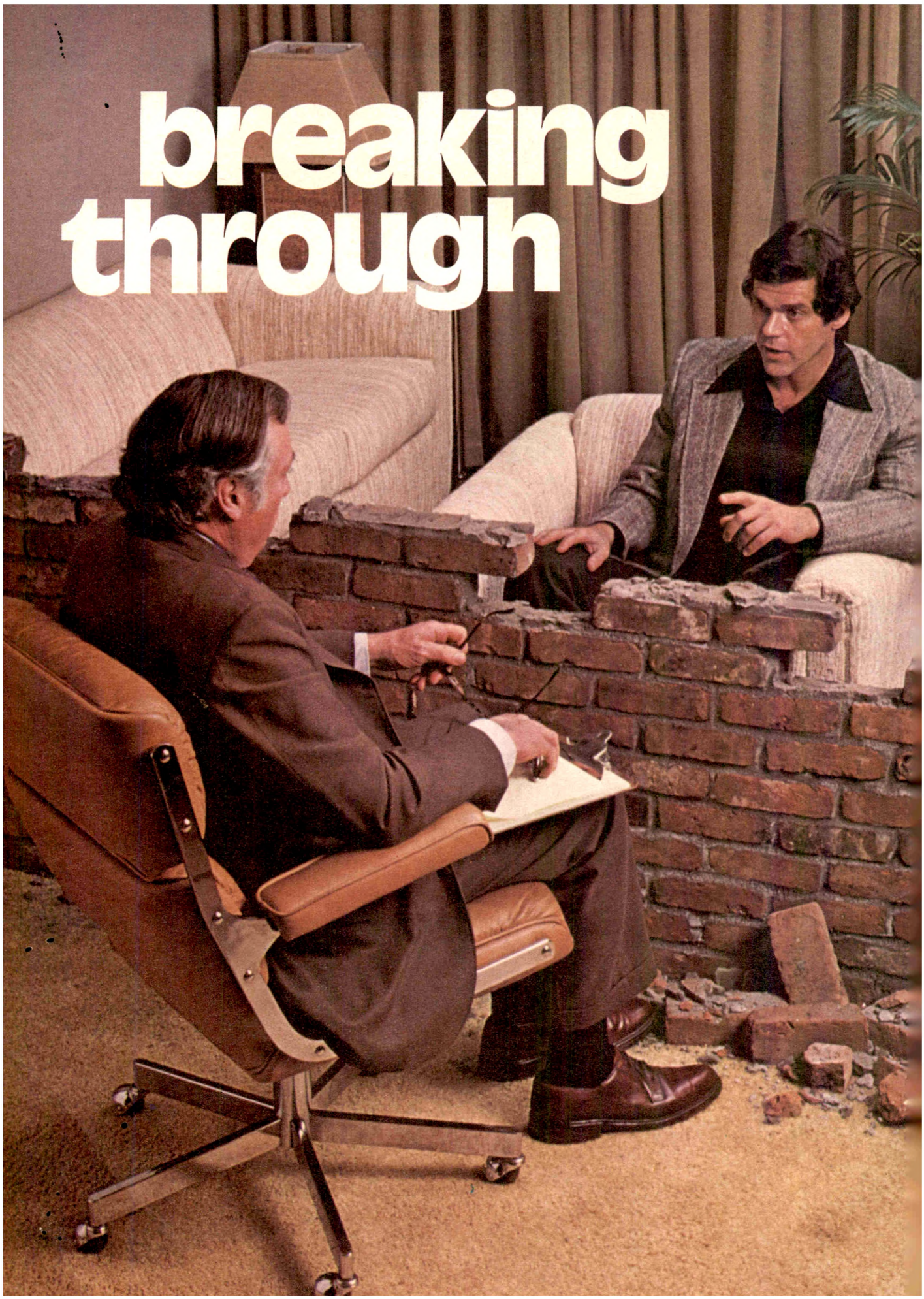
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PRECAUTIONS: Suicide is a possibility in depressed patients and may remain until significant remission occurs. Such patients should not have access to large quantities of this drug.

Perphenazine: Should not be used indiscriminately. Use with caution in patients who have previously exhibited severe adverse reactions to other phenothiazines. Likelihood of some untoward actions is greater with high doses. Closely supervise with any dosage. The antiemetic effect of perphenazine may obscure signs of toxicity due to overdosage of other drugs or make more difficult the diagnosis of disorders such as brain tumor or intestinal obstruction. A significant, not otherwise explained, rise in body temperature may suggest individual intolerance to perphenazine, in which case discontinue.

If hypotension develops, epinephrine should not be employed, as its action is blocked and partially reversed by perphenazine. Phenothiazines may potentiate the action of central nervous system depressants (opiates, analgesics, antihistamines, barbiturates, alcohol) and atropine. In concurrent therapy with any of these, TRIAVIL should be given in reduced dosage. May also potentiate the action of heat and phosphorous insecticides. There is sufficient experimental evidence to conclude that chronic administration of antipsychotic drugs which increase prolactin secretion has the potential to induce mammary neoplasms in rodents under the appropriate conditions. There are recognized differences in the physiological role of prolactin between rodents and humans. Since there are, at present, no adequate epidemiological studies, the relevance to human mammary cancer risk from prolonged exposure to perphenazine and other antipsychotic drugs is not known.

Amitriptyline: In manic-depressive psychosis, depressed patients may experience a shift toward the manic phase if they are treated with an antidepressant. Patients with paranoid symptomatology may have an exaggeration of such symptoms. The tranquilizing effect of TRIAVIL seems to reduce the likelihood of this effect. When amitriptyline HCl is given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required. Paralytic ileus may occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs.

Caution is advised if patients receive large doses of ethchlorvynol concurrently. Transient delirium has been reported in patients who were treated with 1 g of ethchlorvynol and 75-150 mg of amitriptyline HCl.

Amitriptyline HCl may enhance the response to alcohol and the effects of barbiturates and other CNS depressants.

Concurrent administration of amitriptyline HCl and electroshock therapy may increase the hazards associated with such therapy. Such treatment should be limited to patients for whom it is essential. Discontinue several days before elective surgery if possible. Elevation and lowering of blood sugar levels have both been reported. Use with caution in patients with impaired liver function.

ADVERSE REACTIONS: Similar to those reported with either constituent alone.
Perphenazine: Extrapyramidal symptoms (opisthotonus, oculogyric crisis, hyperreflexia, dystonia, akathisia, acute dyskinesia, ataxia, parkinsonism) have been reported and can usually be controlled by the concomitant use of effective antiparkinsonian drugs and/or by reduction in dosage, but sometimes persist after discontinuation of the phenothiazine.

Tardive dyskinesia may appear in some patients on long-term therapy or may occur after drug therapy with phenothiazines and related agents has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. Symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw. Involuntary movements of the extremities sometimes occur. There is no known treatment for tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms. It is advised that all antipsychotic agents be discontinued if the above symptoms appear. If treatment is reinstituted, or dosage of the particular drug increased, or another drug substituted, the syndrome may be masked. Fine vermicular movements of the tongue may be an early sign of the syndrome. The full-blown syndrome may not develop if medication is stopped when lingual vermiculation appears.

Other side effects are skin disorders (photosensitivity, itching, erythema, urticaria, eczema, up to exfoliative dermatitis); other allergic reactions (asthma, laryngeal edema, angioneurotic edema, anaphylactoid reactions); peripheral edema; reversed epinephrine effect; hyperglycemia; endocrine disturbances (lactation, galactorrhea, gynecomastia, disturbances of menstrual cycle); altered cerebrospinal fluid proteins; paradoxical excitement; hypertension, hypotension, tachycardia, and ECG abnormalities (quinidine-like effect); reactivation of psychotic processes; catatonic-like states; autonomic reactions, such as dry mouth or salivation, headache, anorexia, nausea, vomiting, constipation, obstipation, urinary frequency or incontinence, blurred vision, nasal congestion, and a change in pulse rate; other adverse reactions reported with various phenothiazine compounds, but not with perphenazine, include grand mal convulsions, cerebral edema, polyphagia, pigmentary retinopathy, photophobia, skin pigmentation, and failure of ejaculation.

The phenothiazine compounds have produced blood dyscrasias (pancytopenia, thrombocytopenic purpura, leukopenia, agranulocytosis, eosinophilia); and liver damage (jaundice, biliary stasis).

Pigmentation of the cornea and lens has been reported to occur after long-term administration of some phenothiazines. Although it has not been reported in patients receiving TRIAVIL, the possibility that it might occur should be considered.

Hypnotic effects, lassitude, muscle weakness, and mild insomnia have also been reported.

Amitriptyline: Note: Listing includes a few reactions not reported for this drug, but which have occurred with other pharmacologically similar tricyclic antidepressant drugs and must be considered when amitriptyline is administered. *Cardiovascular:* Hypotension; hypertension; tachycardia; palpitation; myocardial infarction; arrhythmias; heart block; stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth; blurred vision; disturbance of accommodation; increased intraocular pressure; constipation; paralytic ileus; urinary retention; dilatation of urinary tract. *Allergic:* Skin rash; urticaria; photosensitization; edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis; leukopenia; eosinophilia; purpura; thrombocytopenia. *Gastrointestinal:* Nausea; epigastric distress; vomiting; anorexia; stomatitis; peculiar taste; diarrhea; parotid swelling; black tongue. Rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido; elevated or lowered blood sugar levels. *Other:* Dizziness, weakness; fatigue; headache; weight gain or loss; increased perspiration; urinary frequency; mydriasis; drowsiness; alopecia. *Withdrawal Symptoms:* Abrupt cessation after prolonged administration may produce nausea, headache, and malaise. These are not indicative of addiction.

OVERDOSAGE: All patients suspected of having taken an overdosage should be admitted to a hospital as soon as possible. Treatment is symptomatic and supportive. However, the intravenous administration of 1–3 mg of physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage of physostigmine should be repeated as required particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine. On this basis, in severe overdosage with perphenazine-amitriptyline combinations, symptomatic treatment of central anticholinergic effects with physostigmine salicylate should be considered.

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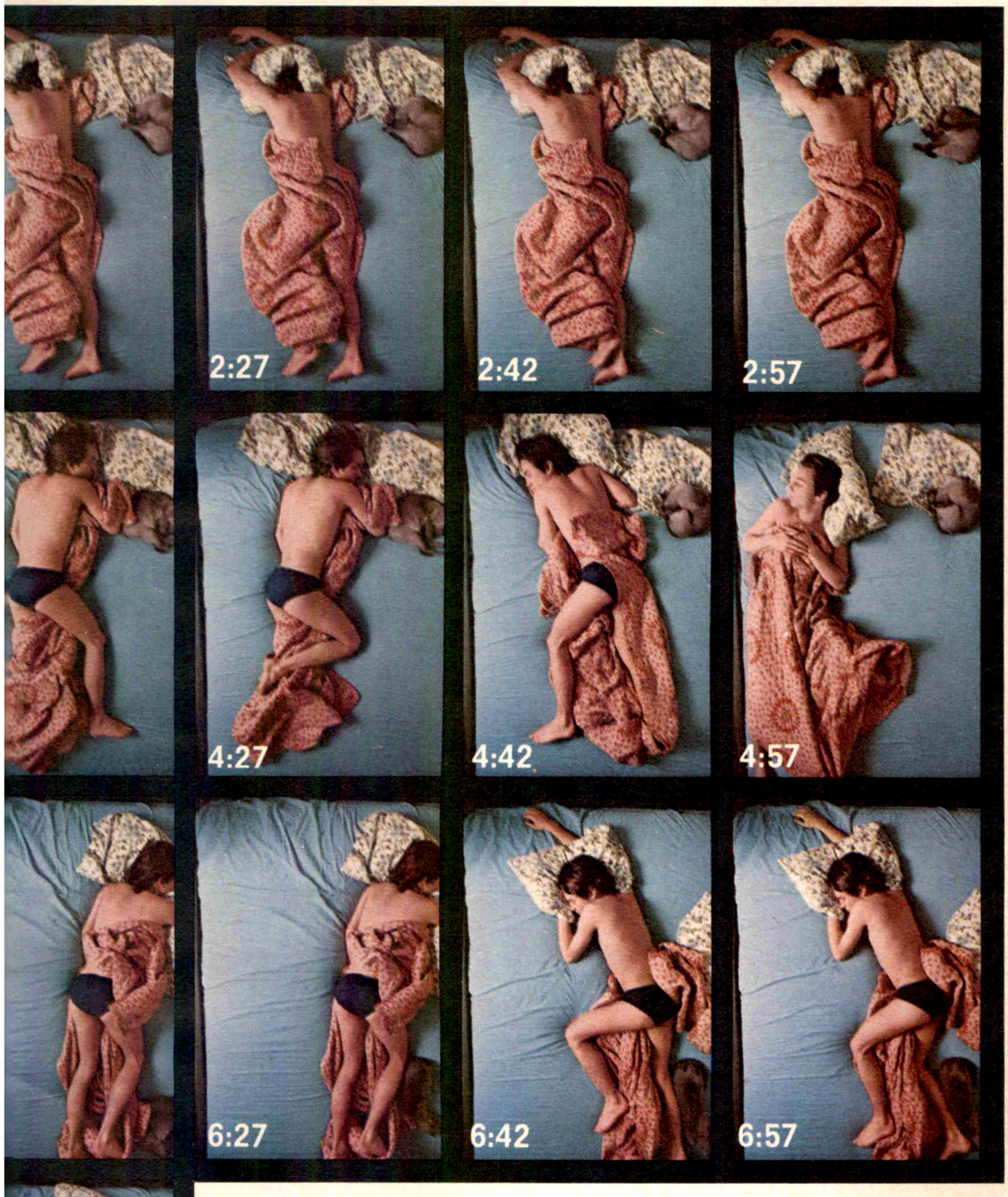
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*Hobson A, Spagna T: Time-lapse photography and the ethology of sleep. *Sleep Res* 5:210, 1976.

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...as proved by nightlong electrophysiologic evidence of increased total sleep time in insomniac subjects in eight sleep laboratory studies.[1]



ASLEEP FASTER: ON AVERAGE, WITHIN 17 MINUTES

...as proved in the same eight studies.[1] Of the hypnotics studied, only Dalmane (flurazepam HCl) improved all three parameters: sleep induction, number of nighttime awakenings and total sleep time.



LONGER EFFICACY: 28 CONSECUTIVE NIGHTS

...of relief from chronic insomnia, as proved in one study using an original 47-night protocol[2] and confirmed in another.[3] (Prolonged administration, though seldom necessary, should be monitored by periodic blood counts and liver and kidney function tests.)

Before prescribing Dalmane (flurazepam HCl), please consult complete product information, a summary of which follows:
Indications: Effective in all types of insomnia characterized by difficulty in falling asleep, frequent nocturnal awakenings and/or early morning awakening; in patients with recurring insomnia or poor sleeping habits; in acute or chronic medical situations requiring restful sleep. Since insomnia is often transient and intermittent, prolonged administration is generally not necessary or recommended.
Contraindications: Known hypersensitivity to flurazepam HCl.
Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).
Usage in Pregnancy: **Several studies of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) suggest increased risk of congenital malformations during the first trimester of pregnancy. Dalmane, a benzodiazepine, has not been studied adequately to determine whether it may be associated with such an increased risk. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**
Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage.
Precautions: In elderly and debilitated, limit initial dosage to 15 mg to preclude oversedation, dizziness and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in patients who are severely depressed, or with latent depression or suicidal tendencies. Periodic blood counts and liver and kidney function tests are advised during repeated therapy. Observe usual precautions in presence of impaired renal or hepatic function.
Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity, and elevated SGOT, SGPT, total and direct bilirubins and alkaline phosphatase.
Dosage: Individualize for maximum beneficial effect.
Adults: 30 mg usual dosage; 15 mg may suffice in some patients. *Elderly or debilitated patients:* 15 mg initially until response is determined.
Supplied: Capsules containing 15 mg or 30 mg flurazepam HCl.

REFERENCES: 1. Data on file, Medical Department, Hoffmann-La Roche Inc., Nutley, NJ. 2. Kales A, *et al: Clin Pharmacol Ther* 18:356-363, Sep 1975. 3. Dement WC, Guilleminault C, Zarcone V: Progress in clinical sleep research. Scientific exhibit at the American Medical Association, Atlantic City, Jun 14-18, 1975. 4. Kales A, *et al: Arch Gen Psychiatry* 23:226-232, Sep 1970. 5. Kales A, Kales J: Recent advances in the diagnosis and treatment of sleep disorders, in *Sleep Research and Clinical Practice,* edited by Usdin G. New York, Brunner/Mazel Inc., 1973, p. 61. 6. Kales A, Kales JD, Martin ED: *Drug Therapy* 5:31-39, Jun 1975. 7. Frost JD Jr: Data on file, Medical Department, Hoffmann-La Roche Inc., Nutley NJ



MORE
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CUMENTATION
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oved more effective than
ral hydrate,[4,5] glute-
ide,[4,5] pentobarbital[2]
secobarbital[5,6] — over 14
ts of administration in the
o laboratory.



AWAKENINGS
SHORTER AND
LESS FREQUENT

...after sleep onset, without repeating dosage during the night.[1] The usual adult dosage is 30 mg *h.s.*, 15 mg may suffice in some patients.



NO DOSAGE
INCREASE FROM
NIGHT TO NIGHT

...for continued effectiveness in chronic insomnia.[2,3] Sleep induction and maintenance are provided by one *h.s.* dosage for 28 nights if necessary. Generally well tolerated, Dalmane (flurazepam HCl) seldom causes morning "hang-over."



EFFECTIVENESS
OBJECTIVELY
PROVED IN
ELDERLY
INSOMNIACS

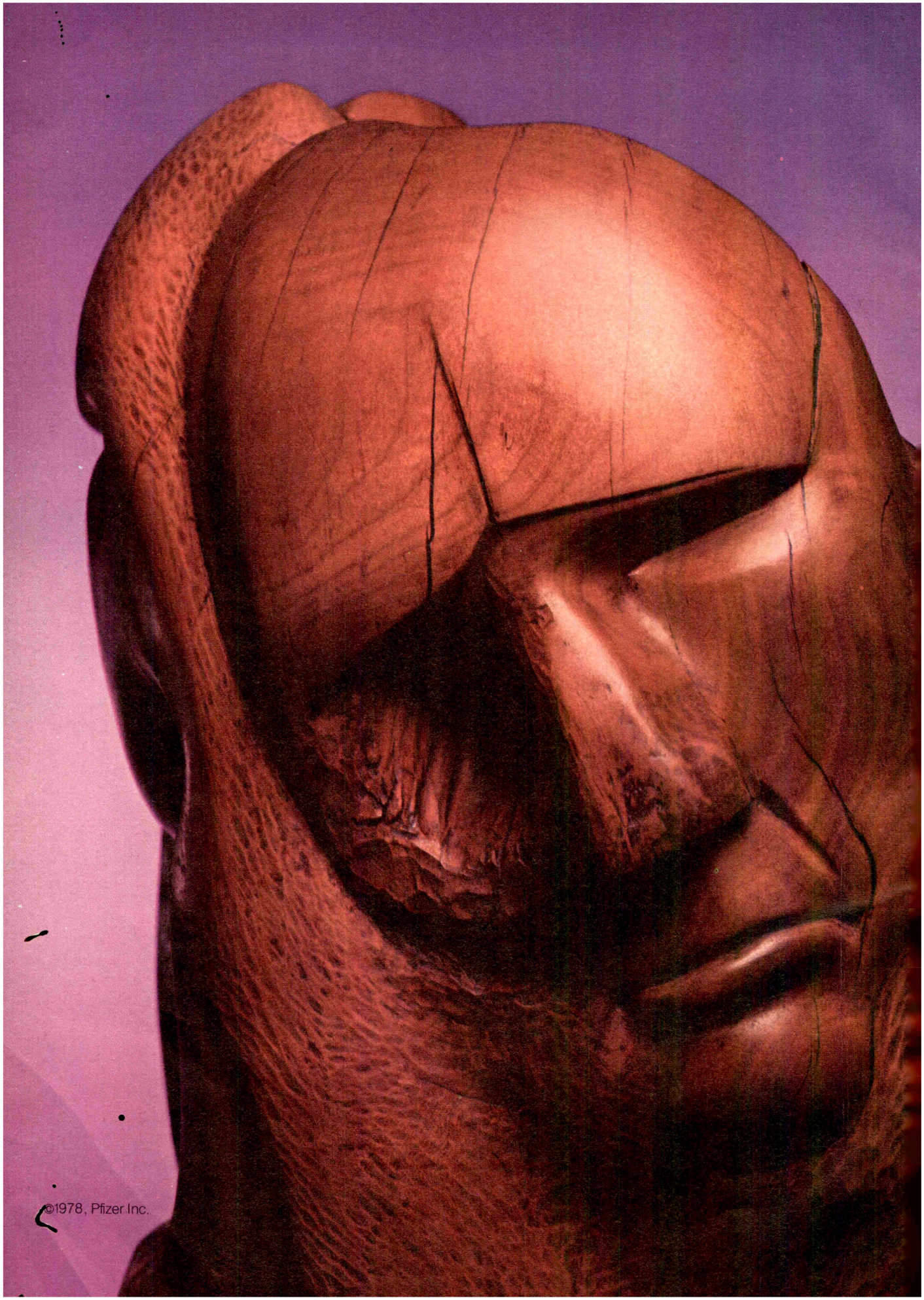
...at 15 mg: this dosage (recommended to *initiate* therapy in the elderly or debilitated to help preclude oversedation, dizziness or ataxia) provided elderly insomniacs with 83 additional minutes of sleep, on average, in the sleep laboratory.[7]

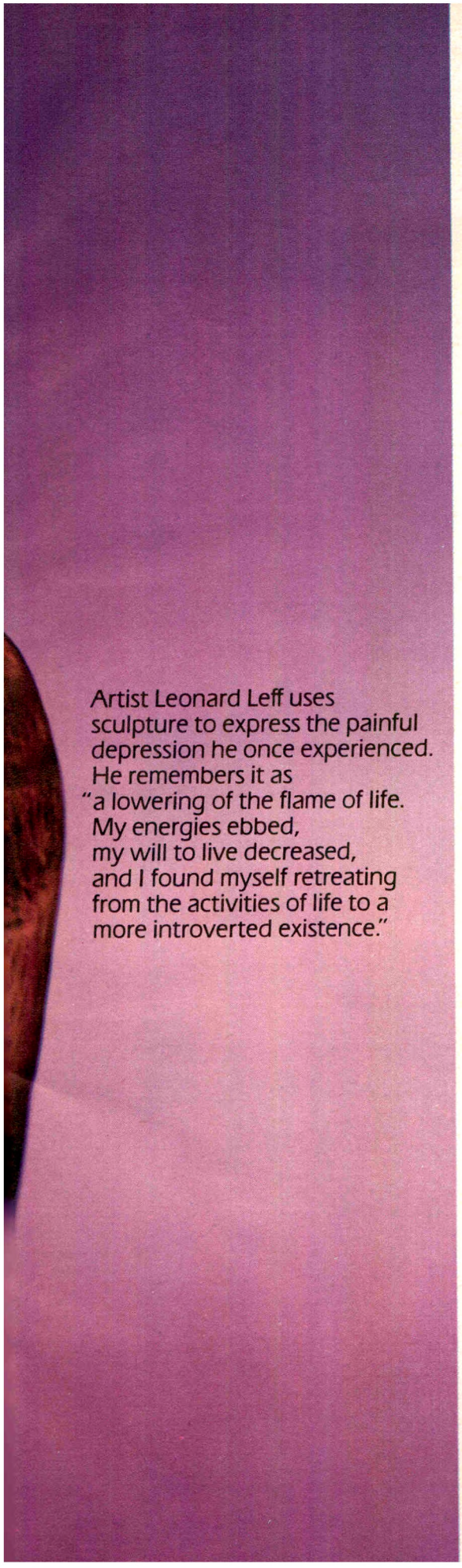
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your first choice
sleep medication

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Artist Leonard Leff uses sculpture to express the painful depression he once experienced. He remembers it as "a lowering of the flame of life. My energies ebbed, my will to live decreased, and I found myself retreating from the activities of life to a more introverted existence."

WHEN DEPRESSION EXPRESSES ITSELF

SINEQUAN®
(DOXEPIN HCl)

ANTIDEPRESSANT EFFECTIVENESS with convenient once-a-day *h.s.* dosage*

150-MG CAPSULE†

Also available in:
100-mg, 75-mg, 50-mg, 25-mg, 10-mg CAPSULES and ORAL CONCENTRATE, 10 mg/ml, in 120-ml (4-oz) bottles

*The total daily dosage of Sinequan, up to 150 mg, may be administered on a once-a-day schedule without loss of effectiveness.

† The 150-mg capsule strength is intended for maintenance therapy only and is not recommended for initiation of treatment.

See Brief Summary on next page for information on contraindications, warnings, precautions and adverse reactions.

ANTIDEPRESSANT EFFECTIVENESS

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(DOXEPIN HCl)

150-mg CAPSULE*

CONVENIENT ONCE-A-DAY *h.s.* DOSAGE

which may improve patient compliance. The total daily dosage, up to 150 mg per day, may be given on a once-a-day schedule without loss of effectiveness. Sinequan may also be given on a divided dosage schedule, up to 300 mg per day.

PROMINENT SEDATIVE EFFECT

which may help to relieve the difficulty in falling and staying asleep, and the early-morning awakening often associated with depression.

ESTABLISHED ANTIANXIETY ACTIVITY

to help alleviate the anxiety which often accompanies clinical depression.

USUALLY WELL TOLERATED

At doses up to 150 mg per day, Sinequan does not generally affect the antihypertensive activity of guanethidine and related compounds. Tachycardia and hypotension have been reported occasionally. Drowsiness is the most commonly observed side effect. Dry mouth, blurred vision, constipation and urinary retention have been reported.

EXTENDED RANGE OF DOSAGE STRENGTHS

for flexibility in individualizing therapy.

*The 150-mg capsule strength is intended for maintenance therapy only and is not recommended for initiation of treatment.

BRIEF SUMMARY
SINEQUAN® (doxepin HCl) Capsules/Oral Concentrate

Contraindications. Contraindicated in individuals who have shown hypersensitivity to the drug, and in patients with glaucoma or a tendency to urinary retention. These disorders should be ruled out, particularly in older patients. Possibility of cross sensitivity with other dibenzoxepines should be kept in mind.

Warnings. The once-a-day dosage regimen of SINEQUAN (doxepin HCl) in patients with intercurrent illness or patients taking other medications should be carefully adjusted. This is especially important in patients receiving other medications with anticholinergic effects.

Usage in Geriatrics: The use of SINEQUAN on a once-a-day dosage regimen in geriatric patients should be adjusted carefully based on the patient's condition.

Usage in Pregnancy: Reproduction studies performed in animals have shown no evidence of harm to the animal fetus. Since there is no experience in pregnant women receiving this drug, safety in pregnancy has not been established. There are no data with respect to the secretion of the drug in human milk and its effect on the nursing infant.

Usage in Children: Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

MAO Inhibitors: Serious side effects and even death have been reported following the concomitant use of certain drugs with MAO inhibitors. Therefore, MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with this drug. The exact length of time may vary and is dependent upon the particular MAO inhibitor being used, the length of time it has been administered and the dosage involved.

Usage with Alcohol: It should be borne in mind that alcohol ingestion may increase the danger inherent in any intentional or unintentional SINEQUAN overdosage. This is especially important in patients who may use alcohol excessively.

Precautions. Since drowsiness may occur with the use of this drug, patients should be warned of that possibility and cautioned against driving a car or operating dangerous machinery while taking this drug.

Patients should also be cautioned that their response to alcohol may be potentiated.

Since suicide is an inherent risk in any depressed patient, and may remain so until significant improvement has occurred, patients should be closely supervised during the early course of therapy. Prescriptions should be written for the smallest feasible amount.

Should increased symptoms of psychosis or shift to manic symptomatology occur, it may be necessary to reduce dosage or add a major tranquilizer to the dosage regimen.

Adverse Reactions. NOTE: Some of the adverse reactions noted below have not been specifically reported with SINEQUAN use. However, due to the close pharmacological similarities among the tricyclics, the reactions should be considered when prescribing SINEQUAN.

Anticholinergic Effects: Dry mouth, blurred vision, constipation, and urinary retention have been reported. If they do not subside with continued therapy, or become severe, it may be necessary to reduce the dosage.

Central Nervous System Effects: Drowsiness is the most commonly noticed side effect. This tends to disappear as therapy is continued. Other infrequently reported CNS side effects are confusion, disorientation, hallucinations, numbness, paresthesias, ataxia, and extrapyramidal symptoms and seizures.

Cardiovascular: Cardiovascular effects including hypotension and tachycardia have been reported occasionally.

Allergic: Skin rash, edema, photosensitization, and pruritus have occasionally occurred.

Hematologic: Eosinophilia has been reported in a few patients. There have been occasional reports of bone marrow depression manifesting as agranulocytosis, leukopenia, thrombocytopenia, and purpura.

Gastrointestinal: Nausea, vomiting, indigestion, taste disturbances, diarrhea, anorexia, and aphthous stomatitis have been reported. (See anticholinergic effects.)

Endocrine: Raised or lowered libido, testicular swelling, gynecomastia in males, enlargement of breasts and galactorrhea in the female, raising or lowering of blood sugar levels have been reported with tricyclic administration.

Other: Dizziness, tinnitus, weight gain, sweating, chills, fatigue, weakness, flushing, jaundice, alopecia, and headache have been occasionally observed as adverse effects.

Dosage and Administration. For most patients with illness of mild to moderate severity, a starting daily dose of 75 mg is recommended. Dosage may subsequently be increased or decreased at appropriate intervals and according to individual response. The usual optimum dose range is 75 mg/day to 150 mg/day.

In more severely ill patients higher doses may be required with subsequent gradual increase to 300 mg/day if necessary. Additional therapeutic effect is rarely to be obtained by exceeding a dose of 300 mg/day.

In patients with very mild symptomatology or emotional symptoms accompanying organic disease, lower doses may suffice. Some of these patients have been controlled on doses as low as 25-50 mg/day.

The total daily dosage of SINEQUAN (doxepin HCl) may be given on a divided or once-a-day dosage schedule. If the once-a-day schedule is employed the maximum recommended dose is 150 mg/day. This dose may be given at bedtime. **The 150 mg capsule strength is intended for maintenance therapy only and is not recommended for initiation of treatment.**

Antianxiety effect is apparent before the antidepressant effect. Optimal antidepressant effect may not be evident for two to three weeks.

Overdosage.

A. Signs and Symptoms

1. Mild: Drowsiness, stupor, blurred vision, excessive dryness of mouth.
2. Severe: Respiratory depression, hypotension, coma, convulsions, cardiac arrhythmias and tachycardias.

Also: urinary retention (bladder atony), decreased gastrointestinal motility (paralytic ileus), hyperthermia (or hypothermia), hypertension, dilated pupils, hyperactive reflexes.

B. Management and Treatment

1. Mild: Observation and supportive therapy is all that is usually necessary.
2. Severe: Medical management of severe SINEQUAN overdosage consists of aggressive supportive therapy. If the patient is conscious, gastric lavage, with appropriate precautions to prevent pulmonary aspiration, should be performed even though SINEQUAN is rapidly absorbed. The use of activated charcoal has been recommended, as has been continuous gastric lavage with saline for 24 hours or more. An adequate airway should be established in comatose patients and assisted ventilation used if necessary. EKG monitoring may be required for several days, since relapse after apparent recovery has been reported. Arrhythmias should be treated with the appropriate antiarrhythmic agent. It has been reported that many of the cardiovascular and CNS symptoms of tricyclic antidepressant poisoning in adults may be reversed by the slow intravenous administration of 1 mg to 3 mg of physostigmine salicylate. Because physostigmine is rapidly metabolized, the dosage should be repeated as required. Convulsions may respond to standard anticonvulsant therapy; however, barbiturates may potentiate any respiratory depression. Dialysis and forced diuresis generally are not of value in the management of overdosage due to high tissue and protein binding of SINEQUAN.

Supply. SINEQUAN is available as capsules containing doxepin HCl equivalent to: 10 mg, 75 mg, and 100 mg doxepin: bottles of 100, 1000, and unit-dose packages of 100 (10 x 10's). 25 mg and 50 mg doxepin: bottles of 100, 1000, 5000, and unit-dose packages of 100 (10 x 10's). 150 mg doxepin: bottles of 50, 500, and unit-dose packages of 100 (10 x 10's). SINEQUAN Oral Concentrate (10 mg/ml) is available in 120 ml bottles with an accompanying dropper calibrated at 5 mg, 10 mg, 15 mg, 20 mg, and 25 mg. Each ml contains doxepin HCl equivalent to 10 mg doxepin. Just prior to administration, SINEQUAN Oral Concentrate should be diluted with approximately 120 ml of water, whole or skimmed milk, or orange, grapefruit, tomato, prune or pineapple juice. SINEQUAN Oral Concentrate is not physically compatible with a number of carbonated beverages. For those patients requiring antidepressant therapy who are on methadone maintenance, SINEQUAN Oral Concentrate and methadone syrup can be mixed together with Gatorade®, lemonade, orange juice, sugar water, Tang®, or water; but not with grape juice. Preparation and storage of bulk dilutions is not recommended.

More detailed professional information available on request.

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Announcing a Special Issue of **The American Journal of Psychiatry** *on*

DEPRESSION and ALCOHOLISM

This mid-April special issue, included in regular subscriptions to the *American Journal of Psychiatry,* focuses on two topics of intense concern to the psychiatric profession: depression and alcoholism.

Containing 30 original articles, it covers the spectrum of diagnostic considerations, treatment, management and complications, etiology and phenomenology. The emphasis throughout is clinical. While several articles explore new work on alcoholism and on the relationship between depression and alcoholism, current studies on depression dominate this valuable collection.

Partial List of Contributors

David Avery, M.D., Karen Beckman, Robert H. Belmaker, M.D., Walter A. Brown, M.D., Jesse O. Cavenar, Jr., M.D., Alberto DiMascio, Ph.D.,* David L. Dunner, M.D., Larry Ereshefsky, Pharm.D., Ronald R. Fieve, M.D., Eric W. Fine, M.D., Evelyn L. Goldberg, Sc.D., John E. Hamm, M.D., John E. Helzer, M.D., Shirley Y. Hill, Ph.D., Martin H. Keeler, M.D., Gerald L. Klerman, M.D., James H. Kocsis, M.D., Eleftherios Lykouras, M.D., Anthony J. Marsella, Ph.D., Roy J. Mathew, M.D., D.P.M., Demmie Mayfield, M.D., Jerome A. Motto, M.D., Michael R. O'Leary, Ph.D., Irving Philips, M.D., Brigitte A. Prusoff, M.P.H., Timothy M. Rivinus, M.D., Marc A. Schuckit, M.D., Fred A. Steinberg, Ph.D., George J. Warheit, Ph.D., Myrna M. Weissman, Ph.D., Steven J. Wolin, M.D.

*deceased

Partial List of Topics

Diagnosis

Depression simulating organic brain disease — diagnosis and lithium treatment of manic-depressive disorder in the retarded — delineation of an MMPI symptom pattern unique to lithium responders

Treatment, Management

Depression treated with imipramine and ECT — possible cardiovascular effect of lithium — lithium maintenance: factors affecting outcome — efficacy of drugs and psychotherapy in the treatment of acute depressive episodes

Etiology, Phenomenology

Life events at the onset of bipolar affective illness — childhood depression: interpersonal interactions and depressive phenomena — drug abuse among alcoholic women — psychiatric problems in women during alcoholic detoxification — life events, coping, stress, and depressive symptomatology

A special Guest Editorial by Morris A. Lipton, Ph.D., M.D., provides a unifying perspective to this special issue.

Extra copies of the April 1979 Special Issue (the 2nd April issue) will be available at $3.50 (domestic), $4.25 (foreign). *Payment must be included with order.* Order from:

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The Prolixin® (Fluphenazine) system of schizophrenia management—

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There is scarcely a patient-type presenting with schizophrenia that cannot be successfully managed with one of the numerous forms of Prolixin (Fluphenazine).

For the newly admitted, agitated patient, a first choice would be short-acting Prolixin Injection (Fluphenazine Hydrochloride Injection USP).

For long-term management of more "dependable" patients, you have the titration flexibility of four potencies of Prolixin Tablets (Fluphenazine Hydrochloride Tablets USP).

For patients who "cheek" or otherwise resist tablet medication, you can utilize pleasant tasting, orange-flavored Prolixin Elixir (Fluphenazine Hydrochloride Elixir USP).

For long-term maintenance of less compliant patients, you can employ unique, long-acting Prolixin Decanoate (Fluphenazine Decanoate Injection) and achieve effective neuroleptization for up to four weeks and longer with a single injection.

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multiple dose vial
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Fluphenazine
Hydrochloride
Elixir USP

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Hydrochloride
Tablets USP

0.5 mg per ml

10 mg 5 mg 2.5 mg 1 mg

See next page for brief summaries

PROLIXIN® (Fluphenazine Hydrochloride)
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Prolixin Tablets (Fluphenazine Hydrochloride Tablets USP) provide 1, 2.5, 5, or 10 mg fluphenazine hydrochloride per tablet. Prolixin Elixir (Fluphenazine Hydrochloride Elixir USP) provides 0.5 mg fluphenazine hydrochloride per ml (2.5 mg per 5 ml teaspoonful) with 14% alcohol by volume. Prolixin Injection (Fluphenazine Hydrochloride Injection USP) provides 2.5 mg fluphenazine hydrochloride per ml; it contains 0.1% methylparaben and 0.01% propylparaben as preservatives.

CONTRAINDICATIONS: In presence of suspected or established subcortical brain damage. In patients who have a blood dyscrasia or liver damage, or who are receiving large doses of hypnotics, or who are comatose or severely depressed. In patients who have shown hypersensitivity to fluphenazine; cross-sensitivity to phenothiazine derivatives may occur.

WARNINGS: Mental and physical abilities required for driving a car or operating heavy machinery may be impaired by use of this drug. Potentiation of effects of alcohol may occur. Safety and efficacy in children have not been established because of inadequate experience in use in children.

Usage in Pregnancy: Safety for use during pregnancy has not been established; weigh possible hazards against potential benefits if administering this drug to pregnant patients.

PRECAUTIONS: Caution must be exercised if another phenothiazine compound caused cholestatic jaundice, dermatoses or other allergic reactions because of the possibility of cross-sensitivity. When psychotic patients on large doses of a phenothiazine drug are to undergo surgery, hypotensive phenomena should be watched for; less anesthetics or central nervous system depressants may be required. Because of added anticholinergic effects, fluphenazine may potentiate the effects of atropine.

Use fluphenazine cautiously in patients exposed to extreme heat or phosphorus insecticides; in patients with a history of convulsive disorders since grand mal convulsions have occurred; and in patients with special medical disorders such as mitral insufficiency or other cardiovascular diseases, and pheochromocytoma. Bear in mind that with prolonged therapy there is the possibility of liver damage, pigmentary retinopathy, lenticular and corneal deposits, and development of irreversible dyskinesia.

There is sufficient experimental evidence to conclude that chronic administration of antipsychotic drugs which increase prolactin secretion has the potential to induce mammary neoplasms in rodents under the appropriate conditions. There are recognized differences in the physiological role of prolactin between rodents and humans. Since there are, at present, no adequate epidemiological studies, the relevance to human mammary cancer risk from prolonged exposure to fluphenazine hydrochloride and other antipsychotic drugs is not known.

Periodic checking of hepatic and renal functions and blood picture should be done. Monitor renal function of patients on long-term therapy; if BUN becomes abnormal, discontinue fluphenazine. "Silent pneumonias" are possible.

Abrupt Withdrawal: In general, phenothiazines do not produce psychic dependence. However, gastritis, nausea and vomiting, dizziness, and tremulousness have been reported following abrupt cessation of high dose therapy; reports suggest that these symptoms can be reduced if concomitant antiparkinsonian agents are continued for several weeks after the phenothiazine is withdrawn.

ADVERSE REACTIONS: *Central Nervous System*—Extrapyramidal symptoms are most frequently reported. Most often these symptoms are reversible, but they may be persistent. They include pseudoparkinsonism, dystonia, dyskinesia, akathisia, oculogyric crises, opisthotonos, hyperreflexia. The incidence and severity of such reactions will depend more on individual patient sensitivity, but dosage level and patient age are also determinants. As these reactions may be alarming, the patient should be forewarned and reassured. These reactions can usually be controlled by administration of an anti-parkinsonian drug such as benztropine mesylate and by subsequent reduction in dosage.

Persistent Tardive Dyskinesia: As with all antipsychotic agents, persistent and sometimes irreversible tardive dyskinesia may appear in some patients on long-term therapy or may occur after discontinuation of drug. The risk seems greater in elderly patients, especially females, on high dosages. The syndrome is characterized by rhythmical involuntary movements of tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and may be accompanied by involuntary movements of extremities. There is no known effective therapy for tardive dyskinesia; usually the symptoms are not alleviated by antiparkinsonism agents. If the symptoms appear, discontinuation of all antipsychotic agents is suggested. The syndrome may be masked if treatment is reinstituted, or drug dosage increased, or a different antipsychotic agent used. Reports are that fine vermicular movements of the tongue may be an early sign of the syndrome which may not develop if medication is stopped at that time.

Phenothiazine derivatives have been known to cause restlessness, excitement, or bizarre dreams; reactivation or aggravation of psychotic processes may be encountered. If drowsiness or lethargy occur, the dosage may need to be reduced. Dosages, far in excess of the recommended amounts, may induce a catatonic-like state.

Autonomic Nervous System—Hypertension and fluctuations in blood pressure have been reported. Although hypotension is rarely a problem, patients with pheochromocytoma, cerebral vascular or renal insufficiency or severe cardiac reserve deficiency such as mitral insufficiency appear to be particularly prone to this reaction and should be observed carefully. Supportive measures including intravenous vasopressor drugs should be instituted immediately should severe hypotension occur; Levarterenol Bitartrate Injection is the most suitable drug; *epinephrine should not be used* since phenothiazine derivatives have been found to reverse its action. Nausea, loss of appetite, salivation, polyuria, perspiration, dry mouth, headache and constipation may occur. Reducing or temporarily discontinuing the dosage will usually control these effects. Blurred vision, glaucoma, bladder paralysis, fecal impaction, paralytic ileus, tachycardia, or nasal congestion have occurred in some patients on phenothiazine derivatives.

Metabolic and Endocrine—Weight change, peripheral edema, abnormal lactation, gynecomastia, menstrual irregularities, false results on pregnancy tests, impotency in men and increased libido in women have occurred in some patients on phenothiazine therapy.

Allergic Reactions—Itching, erythema, urticaria, seborrhea, photosensitivity, eczema and exfoliative dermatitis have been reported with phenothiazines. The possibility of anaphylactoid reactions should be borne in mind.

Hematologic—Blood dyscrasias including leukopenia, agranulocytosis, thrombocytopenic or nonthrombocytopenic purpura, eosinophilia, and pancytopenia have been observed with phenothiazines. If soreness of the mouth, gums or throat or any symptoms of upper respiratory infection occur and confirmatory leukocyte count indicates cellular depression, therapy should be discontinued and other appropriate measures instituted immediately.

Hepatic—Liver damage manifested by cholestatic jaundice, particularly during the first months of therapy, may occur; treatment should be discontinued. A cephalin flocculation increase, sometimes accompanied by alterations in other liver function tests, has been reported in patients who have had no clinical evidence of liver damage.

Others—Sudden deaths have been reported in hospitalized patients on phenothiazines. Previous brain damage or seizures may be predisposing factors. High doses should be avoided in known seizure patients. Shortly before death, several patients showed flare-ups of psychotic behavior patterns. Autopsy findings have usually revealed acute fulminating pneumonia or pneumonitis, aspiration of gastric contents, or intramyocardial lesions. Although not a general feature of fluphenazine, potentiation of central nervous system depressants such as opiates, analgesics, antihistamines, barbiturates, and alcohol may occur.

Systemic lupus erythematosus-like syndrome, hypotension severe enough to cause fatal cardiac arrest, altered electrocardiographic and electroencephalographic tracings, altered cerebrospinal fluid proteins, cerebral edema, asthma, laryngeal edema, and angioneurotic edema, with long-term use, skin pigmentation and lenticular and corneal opacities have occurred with phenothiazines.

For full prescribing information, consult package inserts.

HOW SUPPLIED: *Tablets*—1 mg in bottles of 50 and 500; 2.5 mg and 5 mg in bottles of 50 and 500 and in Unimatic® single-dose cartons of 100; 10 mg in bottles of 50 and 500. *Elixir*—in bottles of 473 ml (1 pint) and in 60 ml dropper-assembly bottles with dropper calibrated at 0.5 ml (0.25 mg), 1 ml (0.5 mg), 1.5 ml (0.75 mg), and 2 ml (1 mg). *Injection*—in multiple-dose vials of 10 ml.

PROLIXIN DECANOATE®
Fluphenazine Decanoate Injection

Prolixin Decanoate (Fluphenazine Decanoate Injection) provides 25 mg fluphenazine decanoate per ml in a sesame oil vehicle with 1.2% (w/v) benzyl alcohol as a preservative.

CONTRAINDICATIONS: In presence of suspected or established subcortical brain damage. In patients who have a blood dyscrasia or liver damage, or who are receiving large doses of hypnotics, or who are comatose or severely depressed. In patients who have shown hypersensitivity to fluphenazine; cross-sensitivity to phenothiazine derivatives may occur.

Not intended for use in children under 12.

WARNINGS: Mental and physical abilities required for driving a car or operating heavy machinery may be impaired by use of this drug. Physicians should be alert to the possibility that severe adverse reactions may occur which require immediate medical attention. Potentiation of effects of alcohol may occur. Safety and efficacy in children have not been established because of inadequate experience in use in children.

Usage in Pregnancy: Safety for use during pregnancy has not been established; weigh possible hazards against potential benefits if administering this drug to pregnant patients.

PRECAUTIONS: Caution must be exercised if another phenothiazine compound caused cholestatic jaundice, dermatoses or other allergic reactions because of the possibility of cross-sensitivity. When psychotic patients on large doses of a phenothiazine drug are to undergo surgery, hypotensive phenomena should be watched for; less anesthetics or central nervous system depressants may be required. Because of added anticholinergic effects, fluphenazine may potentiate the effects of atropine.

Use fluphenazine decanoate cautiously in patients exposed to extreme heat or phosphorus insecticides; in patients with a history of convulsive disorders since grand mal convulsions have occurred; and in patients with special medical disorders such as mitral insufficiency or other cardiovascular diseases, and pheochromocytoma. Bear in mind that with prolonged therapy there is the possibility of liver damage, pigmentary retinopathy, lenticular and corneal deposits and development of irreversible dyskinesia.

Fluphenazine decanoate should be administered under the direction of a physician experienced in the clinical use of psychotropic drugs. Periodic checking of hepatic and renal functions and blood picture should be done. Renal function of patients on long-term therapy should be monitored; if BUN becomes abnormal, treatment should be discontinued. "Silent pneumonias" are possible.

There is sufficient experimental evidence to conclude that chronic administration of antipsychotic drugs which increase prolactin secretion has the potential to induce mammary neoplasms in rodents under the appropriate conditions. There are recognized differences in the physiological role of prolactin between rodents and humans. Since there are, at present, no adequate epidemiological studies, the relevance to human mammary cancer risk from prolonged exposure to fluphenazine decanoate and other antipsychotic drugs is not known.

ADVERSE REACTIONS: *Central Nervous System*—Extrapyramidal symptoms are most frequently reported. Most often these symptoms are reversible, but they may be persistent. They include pseudoparkinsonism, dystonia, dyskinesia, akathisia, oculogyric crises, opisthotonos, hyperreflexia. Muscle rigidity sometimes accompanied by hyperthermia has been reported following use of fluphenazine decanoate. One can expect a higher incidence of such reactions with fluphenazine decanoate than with less potent piperazine derivatives or straight-chain phenothiazines. The incidence and severity will depend more on individual patient sensitivity, but dosage level and patient age are also determinants. As these reactions may be alarming, the patient should be forewarned and reassured. These reactions can usually be controlled by administration of an antiparkinsonian drug such as benztropine mesylate and by subsequent reduction in dosage.

Persistent Tardive Dyskinesia: As with all antipsychotic agents, persistent and sometimes irreversible tardive dyskinesia may appear in some patients on long-term therapy or may occur after discontinuation of drug. The risk seems greater in elderly patients, especially females, on high dosages. The syndrome is characterized by rhythmical involuntary movements of tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and may be accompanied by involuntary movements of extremities. There is no known effective therapy for tardive dyskinesia; usually the symptoms are not alleviated by antiparkinsonism agents. If the symptoms appear, discontinuation of all antipsychotic agents is suggested. The syndrome may be masked if treatment is reinstituted, or drug dosage increased, or a different antipsychotic agent used. Reports are that fine vermicular movements of the tongue may be an early sign of the syndrome which may not develop if medication is stopped at that time.

Phenothiazine derivatives have been known to cause restlessness, excitement, or bizarre dreams; reactivation or aggravation of psychotic processes may be encountered. If drowsiness or lethargy occur, the dosage may need to be reduced. Dosages, far in excess of the recommended amounts, may induce a catatonic-like state.

Autonomic Nervous System—Hypertension and fluctuations in blood pressure have been reported. Although hypotension is rarely a problem, patients with pheochromocytoma, cerebral vascular or renal insufficiency or severe cardiac reserve deficiency such as mitral insufficiency appear to be particularly prone to this reaction and should be observed carefully. Supportive measures including intravenous vasopressor drugs should be instituted immediately should severe hypotension occur; Levarterenol Bitartrate Injection is the most suitable drug; *epinephrine should not be used* since phenothiazine derivatives have been found to reverse its action. Nausea, loss of appetite, salivation, polyuria, perspiration, dry mouth, headache and constipation may occur. Reducing or temporarily discontinuing the dosage will usually control these effects. Blurred vision, glaucoma, bladder paralysis, fecal impaction, paralytic ileus, tachycardia, or nasal congestion have occurred in some patients on phenothiazine derivatives.

Metabolic and Endocrine—Weight change, peripheral edema, abnormal lactation, gynecomastia, menstrual irregularities, false results on pregnancy tests, impotency in men and increased libido in women have occurred in some patients on phenothiazine therapy.

Allergic Reactions—Itching, erythema, urticaria, seborrhea, photosensitivity, eczema and exfoliative dermatitis have been reported with phenothiazines. The possibility of anaphylactoid reactions should be borne in mind.

Hematologic—Blood dyscrasias including leukopenia, agranulocytosis, thrombocytopenic or nonthrombocytopenic purpura, eosinophilia, and pancytopenia have been observed with phenothiazines. If soreness of the mouth, gums or throat or any symptoms of upper respiratory infection occur and confirmatory leukocyte count indicates cellular depression, therapy should be discontinued and other appropriate measures instituted immediately.

Hepatic—Liver damage manifested by cholestatic jaundice, particularly during the first months of therapy, may occur; treatment should be discontinued. A cephalin flocculation increase, sometimes accompanied by alterations in other liver function tests, has been reported in patients who have had no clinical evidence of liver damage.

Others—Sudden deaths have been reported in hospitalized patients on phenothiazines. Previous brain damage or seizures may be predisposing factors. High doses should be avoided in known seizure patients. Shortly before death, several patients showed flare-ups of psychotic behavior patterns. Autopsy findings have usually revealed acute fulminating pneumonia or pneumonitis, aspiration of gastric contents, or intramyocardial lesions. Although not a general feature of fluphenazine, potentiation of central nervous system depressants such as opiates, analgesics, antihistamines, barbiturates, and alcohol may occur.

Systemic lupus erythematosus-like syndrome, hypotension severe enough to cause fatal cardiac arrest, altered electrocardiographic and electroencephalographic tracings, altered cerebrospinal fluid proteins, cerebral edema, asthma, laryngeal edema, and angioneurotic edema; with long-term use, skin pigmentation and lenticular and corneal opacities have occurred with phenothiazines. Local tissue reactions occur only rarely with injections of fluphenazine decanoate.

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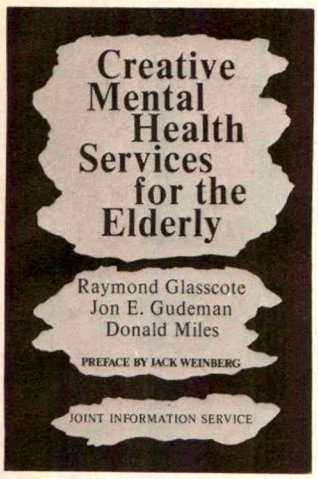
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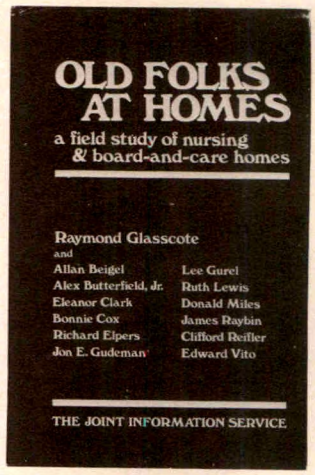
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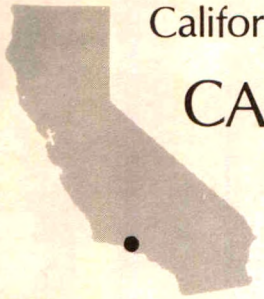
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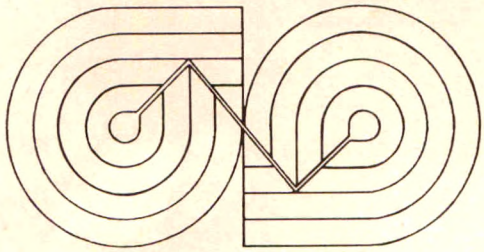
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Patients on TRANXENE for prolonged periods should have blood counts and liver function tests periodically. The usual precautions in treating patients with impaired renal or hepatic function should also be observed.

In elderly or debilitated patients, the initial dose should be small, and increments should be made gradually, in accordance with the response of the patient, to preclude ataxia or excessive sedation.

ADVERSE REACTIONS — The side effect most frequently reported was drowsiness. Less commonly reported (in descending order of occurrence) were: dizziness, various gastrointestinal complaints, nervousness, blurred vision, dry mouth, headache, and mental confusion. Other side effects included insomnia, transient skin rashes, fatigue, ataxia, genitourinary complaints, irritability, diplopia, depression and slurred speech.

There have been reports of abnormal liver and kidney function tests and of decrease in hematocrit.

Decrease in systolic blood pressure has been observed.

DOSAGE AND ADMINISTRATION

For the symptomatic relief of anxiety:

TRANXENE is administered orally. The capsules may be given in divided doses. The usual daily dose is 30 mg. The dose should be adjusted gradually within the range of 15 to 60 mg. daily in accordance with the response of the patient. In elderly or debilitated patients it is advisable to initiate treatment at a daily dose of 7.5 to 15 mg.

TRANXENE capsules may also be administered as a single dose daily at bedtime; the recommended initial dose is 15 mg. After the initial dose, the response of the patient may require adjustment of subsequent dosage. Lower doses may be indicated in the elderly patient. Drowsiness may occur at the initiation of treatment and with dosage increment.

TRANXENE-SD tablets (22.5 mg.) may be administered as a single dose every 24 hours. This tablet is intended as an alternate dosage form for the convenience of patients stabilized on a dose of 7.5 mg. capsules three times a day. TRANXENE-SD tablets should not be used to initiate therapy.

TRANXENE-SD HALF STRENGTH tablets (11.25 mg.) may be administered as a single dose every 24 hours.

For the symptomatic relief of acute alcohol withdrawal:

Recommended schedule: 1st 24 hours, 30 mg. TRANXENE initially, followed by 30 to 60 mg. in divided doses; 2nd 24 hours, 45 to 90 mg. in divided doses; 3rd 24 hours, 22.5 to 45 mg. in divided doses; 4th day, 15 to 30 mg. in divided doses. Thereafter gradually reduce to 7.5 to 15 mg. daily, and discontinue as soon as condition is stable. Maximum daily dose is 90 mg. Avoid excessive reductions in total drug on successive days.

DRUG INTERACTIONS — If TRANXENE is to be combined with other drugs acting on the central nervous system, careful consideration should be given to the pharmacology of the agents to be employed. Animal experience indicates that TRANXENE prolongs the sleeping time after hexobarbital or after ethyl alcohol, increases the inhibitory effects of chlorpromazine, but does not exhibit monoamine oxidase inhibition. Clinical studies have shown increased sedation with concurrent hypnotic medications. The actions of the benzodiazepines may be potentiated by barbiturates, narcotics, phenothiazines, monoamine oxidase inhibitors or other antidepressants.

If TRANXENE is used to treat anxiety associated with somatic disease states, careful attention must be paid to possible drug interaction with concomitant medication.

MANAGEMENT OF OVERDOSAGE — Overdosage is usually manifested by varying degrees of CNS depression ranging from slight sedation to coma. As in the management of overdosage with any drug, it should be borne in mind that multiple agents may have been taken.

There are no specific antidotes for the benzodiazepines. The treatment of overdosage should consist of the general measures employed in the management of overdosage of any CNS depressant. Gastric evacuation either by the induction of emesis, lavage, or both, should be performed immediately. General supportive care, including frequent monitoring of the vital signs and close observation of the patient, is indicated. Hypotension, though rarely reported, may occur with large overdoses. In such cases the use of agents such as Levophed® (levarterenol) or Aramine® (metaraminol) should be considered.

While reports indicate that individuals have survived overdoses of TRANXENE (clorazepate dipotassium) as high as 450 to 675 mg., these doses are not necessarily an accurate indication of the amount of drug absorbed since the time interval between ingestion and the institution of treatment was not always known. Sedation in varying degrees was the most common physiological manifestation of TRANXENE overdosage. Deep coma when it occurred was usually associated with the ingestion of other drugs in addition to TRANXENE.

CLINICAL PHARMACOLOGY — Studies in healthy men have shown that TRANXENE has depressant effects on the central nervous system. Prolonged administration of single daily doses as high as 120 mg. was without toxic effects. Abrupt cessation of high doses was followed in some patients by nervousness, insomnia, irritability, diarrhea, muscle aches, or memory impairment.

Absorption — Excretion:

After oral administration of TRANXENE, there is essentially no circulating parent drug. Nordiazepam, its primary metabolite, quickly appears in the blood stream. In 2 volunteers given 15 mg. (50 μC) of ^{14}C-TRANXENE, about 80% was recovered in the urine and feces within 10 days. Excretion was primarily in the urine with about 1% excreted per day on day 10.

HOW SUPPLIED — TRANXENE (clorazepate dipotassium) is supplied as capsules in three dosage strengths:

3.75 mg. capsules (gray with white cap) in bottles of 100 (NDC 0074-3417-13) and 500 (NDC 0074-3417-53). Also available in ABBO-PAC® unit dose packages of 100 capsules (NDC 0074-3417-11).

7.5 mg. capsules (gray with maroon cap) in bottles of 30 (NDC 0074-3418-30), 100 (NDC 0074-3418-13) and 500 (NDC 0074-3418-53). Also available in ABBO-PAC unit dose packages of 100 capsules (NDC 0074-3418-11).

15 mg. capsules (all gray) in bottles of 100 (NDC 0074-3419-13) and 500 (NDC 0074-3419-53). Also available in ABBO-PAC unit dose packages of 100 capsules (NDC 0074-3419-11).

TRANXENE-SD single dose tablets are supplied in two dosage strengths:

TRANXENE-SD 22.5 mg. tablets (tan-colored) in bottles of 100 (NDC 0074-2997-13).

TRANXENE-SD HALF STRENGTH 11.25 mg. tablets (blue-colored) in bottles of 30 (NDC 0074-2699-30) and 100 (NDC 0074-2699-13).

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Troublesome drowsiness uncommon. Tranxene lets you manage anxiety in most patients without problem sedation. Any initial drowsiness is usually transient. By avoiding excessive sedation, Tranxene helps you calm patients without compromising their capacities.*

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*Of course patients should be cautioned against hazardous tasks requiring mental alertness. See overleaf for prescribing information.



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Vol. 136, No. 4B April 1979

THE AMERICAN JOURNAL OF PSYCHIATRY

Special issue

Depression and Alcoholism

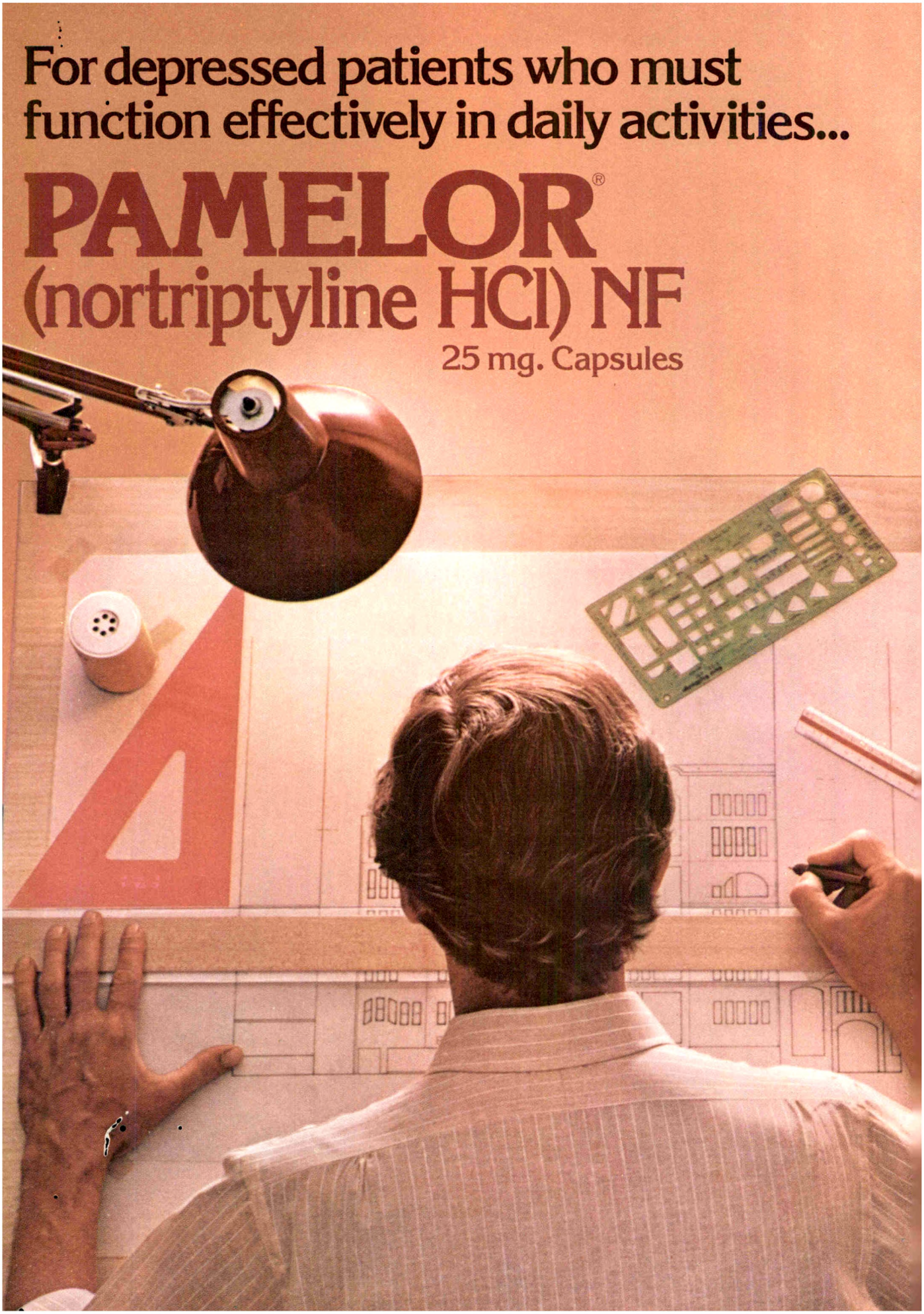
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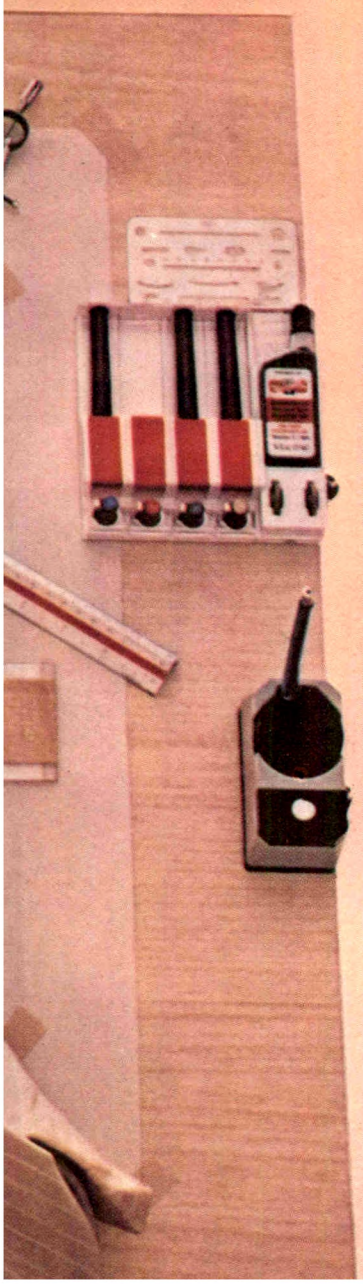
Most depressed patients must function effectively in their daily activities, on the job or at home. For these patients, PAMELOR capsules may be an appropriate therapeutic choice. PAMELOR relieves depression, yet rarely causes daytime drowsiness. As with all antidepressants, however, patients should be cautioned against driving or operating hazardous machinery.

Insomnia of depression begins to improve within a week.

PAMELOR capsules are effective for relieving insomnia, a cardinal symptom of depressive illness. Patients begin to sleep better within the first week of therapy. The full therapeutic effect of PAMELOR is usually observed by the second week.

PAMELOR therapy is well tolerated.

In 90 studies, a total of 818 patients were treated with PAMELOR capsules. Of the patients who improved completely or markedly, over half (54%) had no side effects. Those who experienced side effects most commonly complained of dry mouth.



Indications: For relief of depressive symptoms. Endogenous depressions are more likely to be alleviated than others.

Contraindications: Hypersensitivity. Should not be given concomitantly with MAO inhibitors or within 2 weeks of the use of this drug since hyperpyretic crises, severe convulsions, and fatalities have occurred when similar tricyclic antidepressants were used in such combinations. Cross-sensitivity with other dibenzazepines is a possibility. Contraindicated during acute recovery period after myocardial infarction.

Warnings: Use with caution in patients with cardiovascular disease because of tendency to produce sinus tachycardia and prolong conduction time. Myocardial infarction, arrhythmia, and strokes have occurred. May block antihypertensive action of guanethidine and similar agents. Because of anticholinergic activity, use cautiously in patients with glaucoma or a history of urinary retention. Patients with a history of seizures should be followed closely because the drug is known to lower the convulsive threshold. Great care is required for hyperthyroid patients and those taking thyroid medication because of possible development of cardiac arrhythmia. Caution patients about possibility of impaired mental and/or physical ability to operate a motor vehicle or dangerous machinery. Response to alcoholic beverages may be exaggerated and may lead to suicidal attempts. Safe use during pregnancy, lactation, and women of childbearing potential has not been established and the drug should not be given unless clinical situation warrants potential risk. Not recommended for use in children.

Precautions: Psychotic symptoms may be exacerbated in schizophrenic patients. Increased anxiety and agitation may occur in overactive or agitated patients. Manic-depressive patients may experience shift to manic phase. Hostility may be aroused. Concomitant administration of reserpine may produce a "stimulating" effect. Watch for possible epileptiform seizures during treatment. Use cautiously with anticholinergic or sympathomimetic drugs. Concurrent electroconvulsive therapy may increase hazards associated with nortriptyline HCl. When possible, discontinue drug several days prior to surgery. Potentially suicidal patients require supervision and protective measures during therapy. Prescriptions should be limited to the least possible quantity. Both elevation and lowering of blood sugar levels have been reported.

Adverse Reactions: Note: The pharmacologic similarities among the tricyclic antidepressant drugs require that each of the following reactions be considered when nortriptyline is administered.

Cardiovascular: Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke.

Psychiatric: Confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia, panic, nightmares; hypomania; exacerbation of psychosis.

Neurologic: Numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures, alteration in EEG patterns; tinnitus.

Anticholinergic: Dry mouth and rarely, associated sublingual adenitis; blurred vision, disturbance of accommodation, mydriasis; constipation, paralytic ileus; urinary retention, delayed micturition, dilation of the urinary tract.

Allergic: Skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight); edema (general or of face and tongue), drug fever, cross-sensitivity with other tricyclic drugs.

Hematologic: Bone-marrow depression, including agranulocytosis; eosinophilia; purpura; thrombocytopenia.

Gastrointestinal: Nausea and vomiting, anorexia, epigastric distress, diarrhea, peculiar taste, stomatitis, abdominal cramps, black-tongue.

Endocrine: Gynecomastia in the male, breast enlargement and galactorrhea in the female; increased or decreased libido, impotence, testicular swelling; elevation or depression of blood sugar levels.

Other: Jaundice (simulating obstructive); altered liver function; weight gain or loss; perspiration; flushing; urinary frequency, nocturia; drowsiness, dizziness, weakness, fatigue; headache; parotid swelling; alopecia.

Withdrawal Symptoms: Though these are not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache, and malaise.

Dosage and Administration: *Usual adult dose*—25 mg. three or four times daily; dosage should begin at a low level and increase as required. As an alternate regimen, the total daily dosage may be given once-a-day. *Elderly and Adolescent*—30 to 50 mg. per day, in divided doses, or the total dosage may be given once-a-day. Doses above 100 mg. per day and use in children are not recommended. If a patient develops minor side effects, the dosage should be reduced. The drug should be discontinued promptly if adverse effects of a serious nature or allergic manifestations occur.

How Supplied: Capsules 10 mg. and 25 mg.; solution 10 mg./5 cc.

For more detailed information see full prescribing information.

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ANNOUNCING PUBLICATION OF

THE CHRONIC MENTAL PATIENT

Report of a Conference held in January 1978
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John A. Talbott, M.D.

with contributions by James T. Barter, M.D., Paul R. Friedman, J.D., Raymond Glasscote, M.A., Trevor D. Glenn, M.D., Norman V. Lourie, M.S.W., W. Walter Menninger, M.D., Arthur T. Meyerson, M.D., Kenneth Minkoff, M.D., Samuel Muszynski, M.S.W., Lucy D. Ozarin, M.D., Gordon L. Paul, Ph.D., Ronald Peterson, Steven S. Sharfstein, M.D., Judith Clark Turner, Jane Bloom Yohalem, J.D.

This 277-page Report of the Conference, sponsored by APA and President Carter's Commission on Mental Health, identifies the chronic mental patient population, spells out where they are and what their needs and rights are. It specifies what programs work and what programs do not work in meeting the needs of these patients. It elaborates on the obstacles to implementing effective programs and the economic issues involved. It delineates the pros and cons of case management and specifies responsibility for coordinating, implementing, and monitoring services to chronic mental patients.

Finally, it proposes a *Call to Action* which opens with this statement: "There is no more urgent concern than the needs of the chronic mentally ill who suffer from severe, persistent, or recurrent mental illnesses with residual social and vocational disabilities. As a result of the deinstitutionalization programs of the past decade and the continuing growth of high risk populations that generate chronically ill, the problems associated with the care of these patients constitute a national crisis."

Since the Conference, the APA Assembly and the Board of Trustees have both approved the "Call to Action" which calls upon the APA to take the lead in undertaking programs to elevate the prestige and value of work with chronic mentally ill patients.

It follows that all APA members should be thoroughly versed in the current problems of this chronic patient population. This is best accomplished by reading this Conference Report now available from APA Publications Services at $11.00 a copy.

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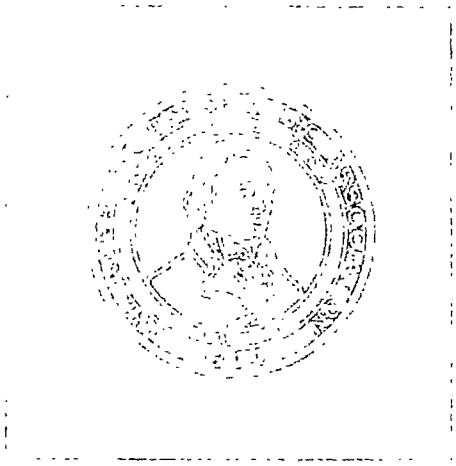
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Manuscripts should be submitted in triplicate to John C. Nemiah, M.D., Editor, *American Journal of Psychiatry*, 1700 Eighteenth St., N.W., Washington, D.C. 20009. All correspondence will be sent to the first-named author unless otherwise specified. Papers that are not accompanied by the appropriate cover letter, including the copyright transfer statement, will not be reviewed until such a statement is received.

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The length of submitted material should not exceed the following specifications unless a special arrangement has been made with the Editor. Regular articles—3,800 words or the equivalent, including references, tables, and figures (about 15 manuscript pages). Brief Communications—2,500 words or equivalent (about 10 manuscript pages). Clinical and Research Reports—1,000 words, 10 references, 1 table (no figures can be used in this section). Letters to the Editor—500 words, 5 references. The number of words, tables, and figures should be noted on the title page.

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Overview articles attempt to bring together important relevant information on a topic of general interest to psychiatry. They are usually written at the invitation of the Editor; authors who have ideas for overview articles are advised to check with the Editor to ensure that a similar work is not in preparation. Overviews should not exceed 25 double-spaced pages (about 7,500 words) and should have no more than 100 references. All overviews, including those written by invitation, are given the same peer review received by other papers.

Regular Articles and Brief Communications

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This section contains very brief articles (maximum of 1,000 words, 10 references, 1 table, and *no figures*) reporting new research findings, including preliminary data from pilot

studies, and case reports that 1) describe new syndromes, 2) cast a new light on established ones, 3) indicate a new therapeutic procedure of potential value, or 4) describe adverse effects or previously unreported complications of drugs or therapeutic interventions. Because of the stringent criteria for this section, authors can expect more rapid publication than is possible in other sections. Submitted papers that substantially exceed the stated maximum length or contain figures will be returned to the author unreviewed.

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Abbreviations. All abbreviations (other than those for metric units) should be explained the first time they are used. Idiosyncratic abbreviations should be avoided: overuse of abbreviations may hinder rather than facilitate readability.

Drugs. Generic rather than trade names of drugs should be used; trade names may be given parenthetically if necessary.

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References should be restricted to closely pertinent material; a complete review of the literature is rarely desirable. *Accuracy of citation is the author's responsibility.* References should conform exactly to the original spelling, accents, punctuation, etc. Authors should be sure that all references listed have been cited in text; no bibliographies can be used.

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1. Berne E: Principles of Group Treatment. New York, Oxford University Press, 1966, p 26
2. Blackwell B, Marley E, Price J, et al: Hypertensive interactions between monoamine oxidase inhibitors and foodstuffs. Br J Psychiatry 113:349-365, 1967
3. Gold PW, Goodwin FK: Urinary free cortisol in depression and mania. Presented at the 130th annual meeting of the American Psychiatric Association, Toronto, Ont, Canada, May 2-6, 1977
4. Brosin H: Communication systems of the consultation process, in The Psychiatric Consultation. Edited by Mendel W, Solomon P. New York, Grune & Stratton, 1968

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The May 1979 issue of

The American Journal of Psychiatry

will feature

- **Research on the Psychosocial Treatment of Schizophrenia: A Summary Report**

  By Loren R. Mosher and Samuel J. Keith



Lithane® 300 mg Tablets
(lithium carbonate)

WARNING

Lithium toxicity is closely related to serum lithium levels, and can occur at doses close to therapeutic levels. Facilities for prompt and accurate serum lithium determinations should be available before initiating therapy.

DESCRIPTION: Lithium carbonate is a white, light, alkaline powder with molecular formula Li_2CO_3 and molecular weight 73.89. Lithium is an element of the alkali-metal group with atomic number 3, atomic weight 6.94, and an emission line at 671 mμ on the flame photometer.

ACTIONS: Preclinical studies have shown that lithium alters sodium transport in nerve and muscle cells and effects a shift toward intraneuronal metabolism of catecholamines, but the specific biochemical mechanism of lithium action in mania is unknown.

INDICATIONS: Lithium carbonate is indicated in the treatment of manic episodes of manic-depressive illness. Maintenance therapy prevents or diminishes the intensity of subsequent episodes in those manic-depressive patients with a history of mania.

Typical symptoms of mania include pressure of speech, motor hyperactivity, reduced need for sleep, flight of ideas, grandiosity, elation, poor judgment, aggressiveness, and possibly hostility. When given to a patient experiencing a manic episode, lithium may produce a normalization of symptomatology within 1 to 3 weeks.

WARNINGS: Lithium should generally not be given to patients with significant renal or cardiovascular disease, severe debilitation or dehydration, or sodium depletion, and to patients receiving diuretics, since the risk of lithium toxicity is very high in such patients. If the psychiatric indication is life-threatening, and if such a patient fails to respond to other measures, lithium treatment may be undertaken with extreme caution, including daily serum lithium determinations and adjustment to the usually low doses ordinarily tolerated by these individuals. In such instances, hospitalization is a necessity.

Lithium toxicity is closely related to serum lithium levels, and can occur at doses close to therapeutic levels (see DOSAGE AND ADMINISTRATION).

Outpatients and their families should be warned that the patient must discontinue lithium carbonate therapy and contact his physician if such clinical signs of lithium toxicity as diarrhea, vomiting, tremor, mild ataxia, drowsiness, or muscular weakness occur.

Usage in Pregnancy: Adverse effects on nidation in rats, embryo viability in mice and metabolism *in vitro* of rat testis and human spermatozoa have been attributed to lithium, as have teratogenicity in submammalian species and cleft palates in mice. Studies in rats, rabbits, and monkeys have shown no evidence of lithium-induced teratology.

There are lithium birth registries in the United States and elsewhere: however there is at the present time insufficient data to determine the effects of lithium carbonate on human fetuses. Therefore, at this point, lithium should not be used in pregnancy, especially the first trimester, unless in the opinion of the physician; the potential benefits outweigh the possible hazards.

Usage in Children: Since information regarding the safety and effectiveness of lithium carbonate in children under 12 years of age is not available, its use in such patients is not recommended at this time.

COMBINED USE OF HALOPERIDOL AND LITHIUM: An encephalopathic syndrome (characterized by weakness, lethargy, fever, tremulousness and confusion, extrapyramidal symptoms, leucocytosis, elevated serum enzymes, BUN and FBS) followed by irreversible brain damage has occurred in a few patients treated with lithium plus haloperidol. A causal relationship between these events and the concomitant administration of lithium and haloperidol has not been established; however, patients receiving such combined therapy should be monitored closely for early evidence of neurological toxicity and treatment discontinued promptly if such signs appear.

PRECAUTIONS: The ability to tolerate lithium is greater during the acute manic phase and decreases when manic symptoms subside (see DOSAGE AND ADMINISTRATION).

The distribution space of lithium approximates that of total body water. Lithium is primarily excreted in urine with insignificant excretion in feces. Renal excretion of lithium is proportional to its plasma concentration. The half-elimination time of lithium is approximately 24 hours. Lithium decreases sodium reabsorption by the renal tubules which could lead to sodium depletion. Therefore, it is essential for the patient to maintain a normal diet, including salt, and an adequate fluid intake (2500–3000 ml) at least during the initial stabilization period. Decreased tolerance to lithium has been reported to ensue from protracted sweating or diarrhea and, if such occur, supplemental fluid and salt should be administered.

In addition to sweating and diarrhea, concomitant infection with elevated temperatures may also necessitate a temporary reduction or cessation of medication.

Untoward reactions to simultaneous use of lithium and other psychoactive drugs have not been reported. However, the possibility of adverse interaction with other anti-psychotic medications remains.

ADVERSE REACTIONS: Adverse reactions are seldom encountered at serum lithium levels below 1.5 mEq/L, except in the occasional patient sensitive to lithium. Mild to moderate toxic reactions may occur at levels from 1.5–2.5 mEq/L, and moderate to severe reactions may be seen at levels from 2.0–2.5 mEq/L, depending upon individual response to the drug.

Fine hand tremor, polyuria, and mild thirst may occur during initial therapy for the acute manic phase, and may persist throughout treatment. Transient and mild nausea and general discomfort may also appear during the first few days of lithium administration.

These side effects are an inconvenience rather than a disabling condition, and usually subside with continued treatment or a temporary reduction or cessation of dosage. If persistent, a cessation of dosage is indicated.

Diarrhea, vomiting, drowsiness, muscular weakness, and lack of coordination may be early signs of lithium intoxication, and can occur at lithium levels below 2.0 mEq/L. At higher levels, giddiness, ataxia, blurred vision, tinnitus, and a large output of dilute urine may be seen. Serum lithium levels above 3.0 mEq/L may produce a complex clinical picture involving multiple organs and organ systems. Serum lithium levels should not be permitted to exceed 2.0 mEq/L during the acute treatment phase.

The following toxic reactions have been reported and appear to be related to serum lithium levels:

Neuromuscular: Tremor, muscle hyperirritability (fasciculations, twitching, clonic movements of whole limbs), ataxia, choreo-athetotic movements, hyperactive deep tendon reflexes.

Central Nervous System: Blackout spells, epileptiform seizures, slurred speech, dizziness, vertigo, incontinence of urine or feces, somnolence, psychomotor retardation, restlessness, confusion, stupor, coma.

Cardiovascular: Cardiac arrhythmia, hypotension, peripheral circulatory collapse.

Gastrointestinal: Anorexia, nausea, vomiting, diarrhea.

Genitourinary: Albuminuria, oliguria, polyuria, glycosuria.

Dermatologic: Drying and thinning of hair, anesthesia of skin, chronic folliculitis, xerosis cutis.

Autonomic Nervous System: Blurred vision, dry mouth.

Miscellaneous: Fatigue, lethargy, tendency to sleep, dehydration, weight loss, transient scotomata.

Thyroid Abnormalities: Euthyroid goiter and/or hypothyroidism (including myxedema) accompanied by lower T_3 and T_4. ^{131}I uptake may be elevated. Underlying thyroid disorders do not constitute a contraindication to lithium treatment; careful monitoring of thyroid function during lithium stabilization and maintenance allows for correction of changing thyroid parameters, if any. They can be treated by prescribing thyroid in these cases.

EEG Changes: Diffuse slowing, widening of frequency spectrum, potentiation and disorganization of background rhythm.

EKG Changes: Reversible flattening, isoelectricity or inversion of T-waves.

Miscellaneous Reactions Unrelated to Dosage: Transient electroencephalographic and electrocardiographic changes, leucocytosis, headache, diffuse nontoxic goiter with or without hypothyroidism, transient hyperglycemia, generalized pruritus with or without rash, cutaneous ulcers, albuminuria, worsening of organic brain syndromes, excessive weight gain, edematous swelling of ankles or wrists, and thirst or polyuria, sometimes resembling diabetes insipidus. A single report has been received of the development of painful discoloration of fingers and toes and coldness of the extremities within one day of the starting of treatment of lithium. The mechanism through which these symptoms (resembling Raynaud's syndrome) developed is not known. Recovery followed discontinuance.

DOSAGE AND ADMINISTRATION: Acute Mania: Optimal patient response to lithium carbonate usually can be established and maintained with 600 mg t.i.d. Such doses will normally produce an effective serum lithium level ranging between 1.0 and 1.5 mEq/L. Dosage must be individualized according to serum levels and clinical response. Regular monitoring of the patient's clinical state and of serum lithium levels is necessary. Serum levels should be determined twice per week during the acute phase, and until the serum level and clinical condition of the patient have been stabilized.

Long-Term Control: The desirable lithium levels are 0.6 to 1.2 mEq/L. Dosage will vary from one individual to another, but usually 300 mg t.i.d. or q.i.d. will maintain this level. Serum lithium levels in uncomplicated cases receiving maintenance therapy during remission should be monitored at least every two months.

Patients abnormally sensitive to lithium may exhibit toxic signs at serum levels of 1.0 to 1.5 mEq/L. Elderly patients often respond to reduced dosage, and may exhibit signs of toxicity at serum levels ordinarily tolerated by other patients.

NB: Blood samples for serum lithium determinations are drawn 8–12 hours after the previous dose when lithium concentrations are relatively stable. Total reliance must not be placed on serum levels alone. Accurate patient evaluation requires both clinical and laboratory analysis.

OVERDOSAGE: The toxic levels for lithium are close to the therapeutic levels. It is therefore important that patients and their families be cautioned to watch for early toxic symptoms and to discontinue the drug and inform the physician should they occur. Toxic symptoms are listed in detail under ADVERSE REACTIONS.

Treatment: No specific antidote for lithium poisoning is known. Early symptoms of lithium toxicity can usually be treated by reduction or cessation of dosage of the drug and resumption of the treatment at a lower dose after 24 to 48 hours. In severe cases of lithium poisoning, the first and foremost goal of treatment consists of elimination of this ion from the organism.

Treatment is essentially the same as that used in barbiturate poisoning: 1) lavage, 2) correction of fluid and electrolyte imbalance and 3) regulation of kidney functioning. Urea, mannitol, and aminophylline all produce significant increases in lithium excretion. Infection prophylaxis, regular chest X-rays, and preservation of adequate respiration are essential.

HOW SUPPLIED: Lithane (lithium carbonate) is available as scored tablets containing 300 mg of lithium carbonate in bottles of 100 and 1000.

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George Frederick Handel
(1685-1759), known for his swings from depression to mania, composed his majestic *Messiah* oratorio in only six weeks. If he were living today, lithium would probably control his symptoms.

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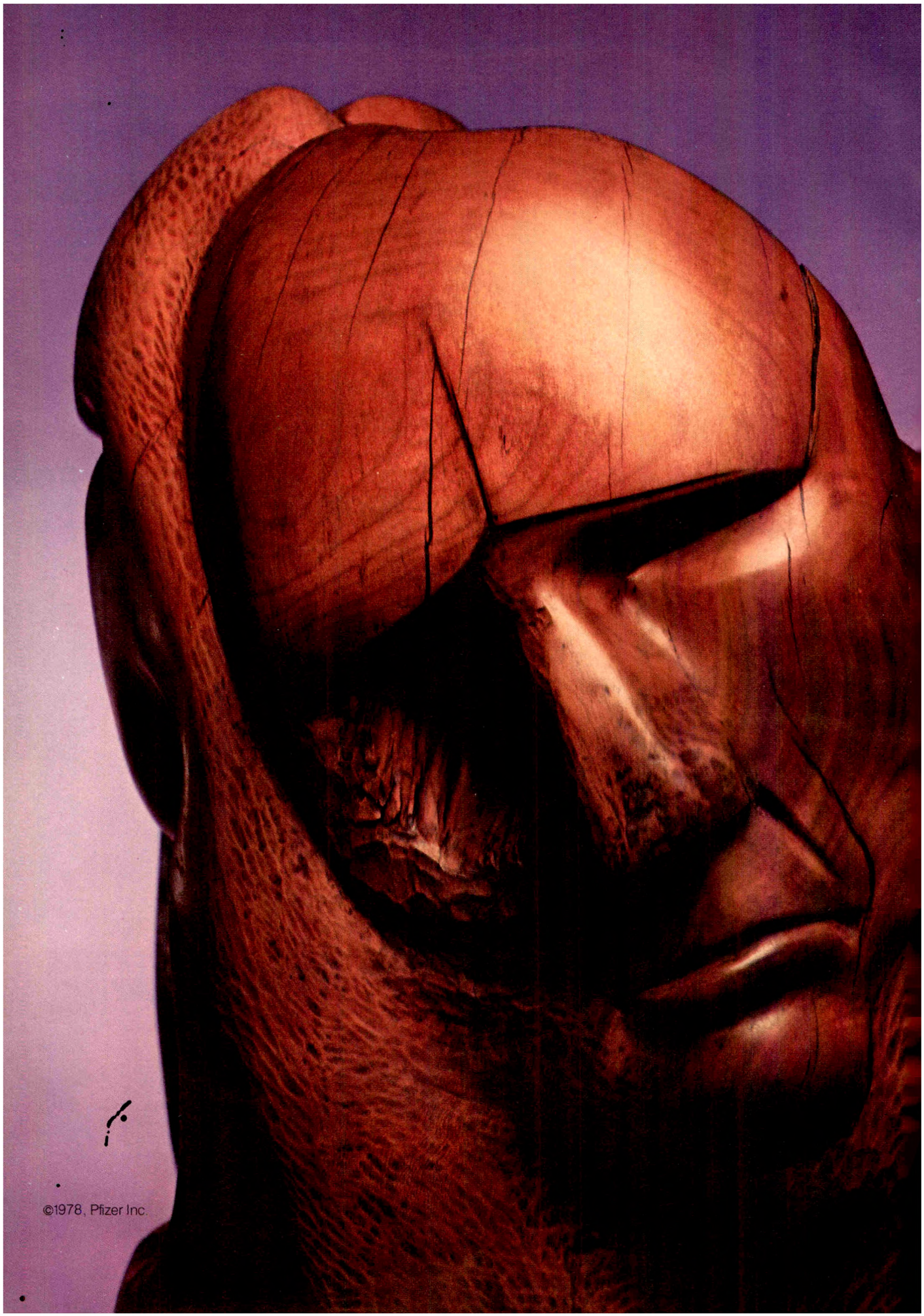
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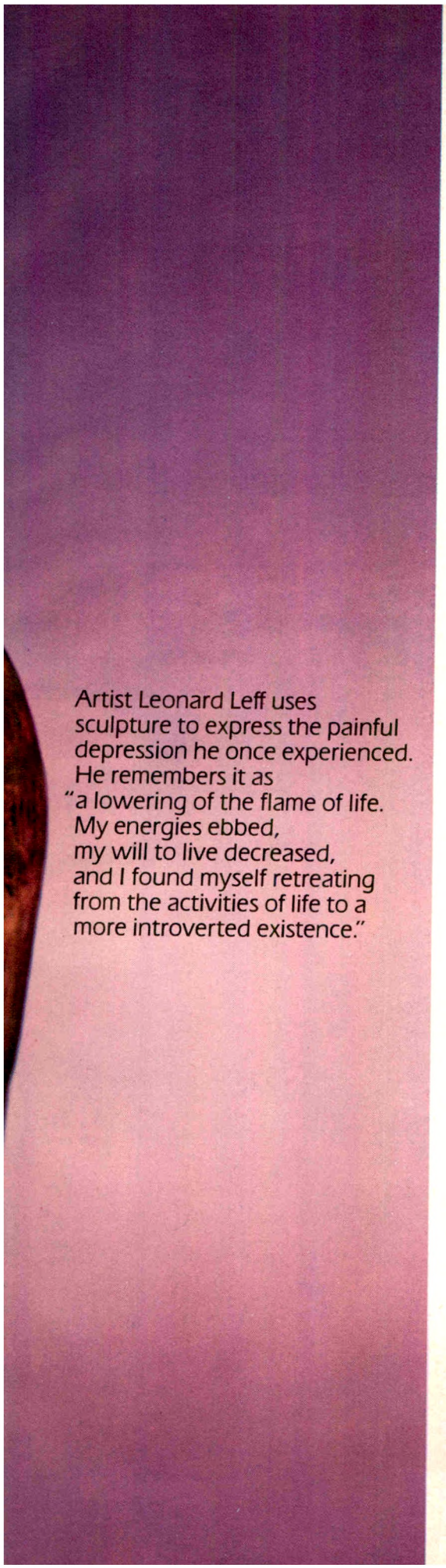
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Artist Leonard Leff uses sculpture to express the painful depression he once experienced. He remembers it as "a lowering of the flame of life. My energies ebbed, my will to live decreased, and I found myself retreating from the activities of life to a more introverted existence."

WHEN DEPRESSION EXPRESSES ITSELF

SINEQUAN®
(DOXEPIN HCl)

ANTIDEPRESSANT EFFECTIVENESS with convenient once-a-day *h.s.* dosage*

150-MG CAPSULE†

Also available in:
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*The total daily dosage of Sinequan, up to 150 mg, may be administered on a once-a-day schedule without loss of effectiveness.

† The 150-mg capsule strength is intended for maintenance therapy only and is not recommended for initiation of treatment.

See Brief Summary on next page for information on contraindications, warnings, precautions and adverse reactions.

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CONVENIENT ONCE-A-DAY *h.s.* DOSAGE

which may improve patient compliance. The total daily dosage, up to 150 mg per day, may be given on a once-a-day schedule without loss of effectiveness. Sinequan may also be given on a divided dosage schedule, up to 300 mg per day.

PROMINENT SEDATIVE EFFECT

which may help to relieve the difficulty in falling and staying asleep, and the early-morning awakening often associated with depression.

ESTABLISHED ANTIANXIETY ACTIVITY

to help alleviate the anxiety which often accompanies clinical depression.

USUALLY WELL TOLERATED

At doses up to 150 mg per day, Sinequan does not generally affect the antihypertensive activity of guanethidine and related compounds. Tachycardia and hypotension have been reported occasionally. Drowsiness is the most commonly observed side effect. Dry mouth, blurred vision, constipation and urinary retention have been reported.

EXTENDED RANGE OF DOSAGE STRENGTHS

for flexibility in individualizing therapy.

BRIEF SUMMARY
SINEQUAN® (doxepin HCl) Capsules/Oral Concentrate

Contraindications. Contraindicated in individuals who have shown hypersensitivity to the drug, and in patients with glaucoma or a tendency to urinary retention. These disorders should be ruled out, particularly in older patients. Possibility of cross sensitivity with other dibenzoxepines should be kept in mind.

Warnings. The once-a-day dosage regimen of SINEQUAN (doxepin HCl) in patients with intercurrent illness or patients taking other medications should be carefully adjusted. This is especially important in patients receiving other medications with anticholinergic effects.

Usage in Geriatrics: The use of SINEQUAN on a once-a-day dosage regimen in geriatric patients should be adjusted carefully based on the patient's condition.

Usage in Pregnancy: Reproduction studies performed in animals have shown no evidence of harm to the animal fetus. Since there is no experience in pregnant women receiving this drug, safety in pregnancy has not been established. There are no data with respect to the secretion of the drug in human milk and its effect on the nursing infant.

Usage in Children: Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

MAO Inhibitors: Serious side effects and even death have been reported following the concomitant use of certain drugs with MAO inhibitors. Therefore, MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with this drug. The exact length of time may vary and is dependent upon the particular MAO inhibitor being used, the length of time it has been administered and the dosage involved.

Usage with Alcohol: It should be borne in mind that alcohol ingestion may increase the danger inherent in any intentional or unintentional SINEQUAN overdosage. This is especially important in patients who may use alcohol excessively.

Precautions. Since drowsiness may occur with the use of this drug, patients should be warned of that possibility and cautioned against driving a car or operating dangerous machinery while taking this drug.

Patients should also be cautioned that their response to alcohol may be potentiated.

Since suicide is an inherent risk in any depressed patient, and may remain so until significant improvement has occurred, patients should be closely supervised during the early course of therapy. Prescriptions should be written for the smallest feasible amount.

Should increased symptoms of psychosis or shift to manic symptomatology occur, it may be necessary to reduce dosage or add a major tranquilizer to the dosage regimen.

Adverse Reactions. NOTE: Some of the adverse reactions noted below have not been specifically reported with SINEQUAN use. However, due to the close pharmacological similarities among the tricyclics, the reactions should be considered when prescribing SINEQUAN.

Anticholinergic Effects: Dry mouth, blurred vision, constipation, and urinary retention have been reported. If they do not subside with continued therapy, or become severe, it may be necessary to reduce the dosage.

Central Nervous System Effects: Drowsiness is the most commonly noticed side effect. This tends to disappear as therapy is continued. Other infrequently reported CNS side effects are confusion, disorientation, hallucinations, numbness, paresthesias, ataxia, and extrapyramidal symptoms and seizures.

Cardiovascular: Cardiovascular effects including hypotension and tachycardia have been reported occasionally.

Allergic: Skin rash, edema, photosensitization, and pruritus have occasionally occurred.

Hematologic: Eosinophilia has been reported in a few patients. There have been occasional reports of bone marrow depression manifesting as agranulocytosis, leukopenia, thrombocytopenia, and purpura.

Gastrointestinal: Nausea, vomiting, indigestion, taste disturbances, diarrhea, anorexia, and aphthous stomatitis have been reported. (See anticholinergic effects.)

Endocrine: Raised or lowered libido, testicular swelling, gynecomastia in males, enlargement of breasts and galactorrhea in the female, raising or lowering of blood sugar levels have been reported with tricyclic administration.

Other: Dizziness, tinnitus, weight gain, sweating, chills, fatigue, weakness, flushing, jaundice, alopecia, and headache have been occasionally observed as adverse effects.

Dosage and Administration. For most patients with illness of mild to moderate severit starting daily dose of 75 mg is recommended. Dosage may subsequently be increase decreased at appropriate intervals and according to individual response. The usual optim dose range is 75 mg/day to 150 mg/day.

In more severely ill patients higher doses may be required with subsequent gradual increas 300 mg/day if necessary. Additional therapeutic effect is rarely to be obtained by exceedir dose of 300 mg/day.

In patients with very mild symptomatology or emotional symptoms accompanying orga disease, lower doses may suffice. Some of these patients have been controlled on doses as as 25-50 mg/day.

The total daily dosage of SINEQUAN (doxepin HCl) may be given on a divided or once-a- dosage schedule. If the once-a-day schedule is employed the maximum recommended dos 150 mg/day. This dose may be given at bedtime. **The 150 mg capsule strength is intended maintenance therapy only and is not recommended for initiation of treatment.**

Antianxiety effect is apparent before the antidepressant effect. Optimal antidepressant ef may not be evident for two to three weeks.

Overdosage.

A. Signs and Symptoms

1. Mild: Drowsiness, stupor, blurred vision, excessive dryness of mouth.
2. Severe: Respiratory depression, hypotension, coma, convulsions, cardiac arrhythmias tachycardias.

Also: urinary retention (bladder atony), decreased gastrointestinal motility (paralytic ile hyperthermia (or hypothermia), hypertension, dilated pupils, hyperactive reflexes.

B. Management and Treatment

1. Mild: Observation and supportive therapy is all that is usually necessary.
2. Severe: Medical management of severe SINEQUAN overdosage consists of aggres supportive therapy. If the patient is conscious, gastric lavage, with appropriate precautior prevent pulmonary aspiration, should be performed even though SINEQUAN is rapidly absor The use of activated charcoal has been recommended, as has been continuous gastric lav with saline for 24 hours or more. An adequate airway should be established in comatose pati and assisted ventilation used if necessary. EKG monitoring may be required for several d since relapse after apparent recovery has been reported. Arrhythmias should be treated with appropriate antiarrhythmic agent. It has been reported that many of the cardiovascular and symptoms of tricyclic antidepressant poisoning in adults may be reversed by the slow ir venous administration of 1 mg to 3 mg of physostigmine salicylate. Because physostigmir rapidly metabolized, the dosage should be repeated as required. Convulsions may respor standard anticonvulsant therapy; however, barbiturates may potentiate any respiratory dep sion. Dialysis and forced diuresis generally are not of value in the management of overdos due to high tissue and protein binding of SINEQUAN.

Supply. SINEQUAN is available as capsules containing doxepin HCl equivalent to: 10 mg, 75 and 100 mg doxepin: bottles of 100, 1000, and unit-dose packages of 100 (10 x 10's). 25 mg 50 mg doxepin: bottles of 100, 1000, 5000, and unit-dose packages of 100 (10 x 10's). 150 doxepin: bottles of 50, 500, and unit-dose packages of 100 (10 x 10's). SINEQUAN Concentrate (10 mg/ml) is available in 120 ml bottles with an accompanying dropper calibr at 5 mg, 10 mg, 15 mg, 20 mg, and 25 mg. Each ml contains doxepin HCl equivalent to 1 doxepin. Just prior to administration, SINEQUAN Oral Concentrate should be diluted approximately 120 ml of water, whole or skimmed milk, or orange, grapefruit, tomato, prur pineapple juice. SINEQUAN Oral Concentrate is not physically compatible with a numb carbonated beverages. For those patients requiring antidepressant therapy who ar methadone maintenance, SINEQUAN Oral Concentrate and methadone syrup can be m together with Gatorade®, lemonade, orange juice, sugar water, Tang®, or water; but not grape juice. Preparation and storage of bulk dilutions is not recommended.

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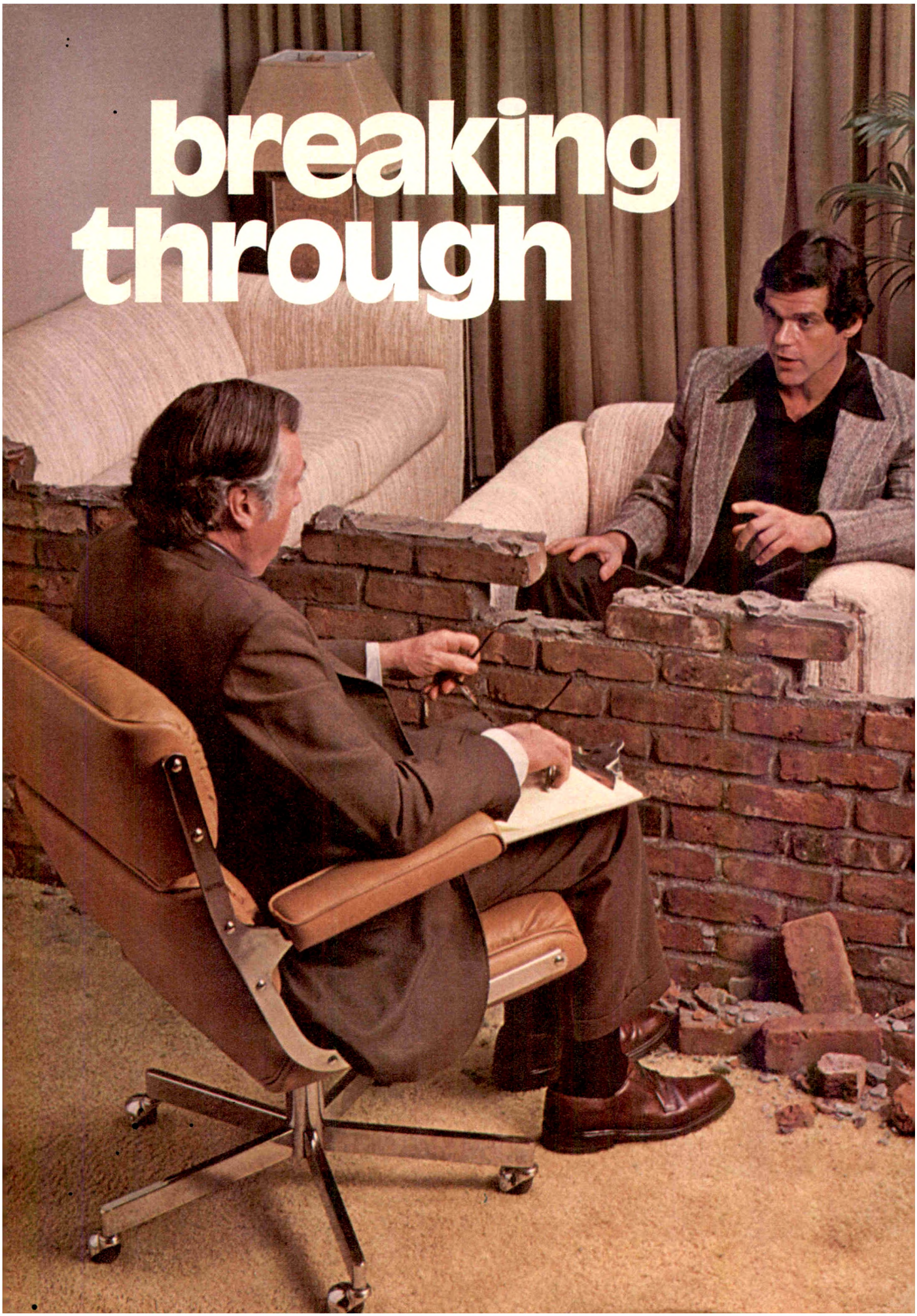
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CONTRAINDICATIONS: Central nervous system depression from drugs (barbiturates, alcohol, narcotics, analgesics, antihistamines); evidence of bone marrow depression; known hypersensitivity to phenothiazines or amitriptyline. Should not be given concomitantly with a monoamine oxidase inhibitor since hyperpyretic crises, severe convulsions, and deaths have occurred from such combinations. When used to replace a monoamine oxidase inhibitor, allow a minimum of 14 days to elapse before initiating therapy with TRIAVIL. Therapy should then be initiated cautiously with gradual increase in dosage until optimum response is achieved. Not recommended for use during acute recovery phase following myocardial infarction.

WARNINGS: TRIAVIL should not be given concomitantly with guanethidine or similarly acting compounds since TRIAVIL may block the antihypertensive action of such compounds. Use cautiously in patients with history of urinary retention, angle-closure glaucoma, increased intraocular pressure, or convulsive disorders. Dosage of anticonvulsive agents may have to be increased. In patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely. Tricyclic antidepressants, including amitriptyline HCl, have been reported to produce arrhythmias, sinus tachycardia, and prolongation of conduction time, particularly in high doses. Myocardial infarction and stroke have been reported with tricyclic antidepressant drugs. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. May impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle. In patients who use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage. Not recommended in children or during pregnancy.

PRECAUTIONS: Suicide is a possibility in depressed patients and may remain until significant remission occurs. Such patients should not have access to large quantities of this drug.

Perphenazine: Should not be used indiscriminately. Use with caution in patients who have previously exhibited severe adverse reactions to other phenothiazines. Likelihood of some untoward actions is greater with high doses. Closely supervise with any dosage. The antiemetic effect of perphenazine may obscure signs of toxicity due to overdosage of other drugs or make more difficult the diagnosis of disorders such as brain tumor or intestinal obstruction. A significant, not otherwise explained, rise in body temperature may suggest individual intolerance to perphenazine, in which case discontinue.

If hypotension develops, epinephrine should not be employed, as its action is blocked and partially reversed by perphenazine. Phenothiazines may potentiate the action of central nervous system depressants (opiates, analgesics, antihistamines, barbiturates, alcohol) and atropine. In concurrent therapy with any of these, TRIAVIL should be given in reduced dosage. May also potentiate the action of heat and phosphorous insecticides. There is sufficient experimental evidence to conclude that chronic administration of antipsychotic drugs which increase prolactin secretion has the potential to induce mammary neoplasms in rodents under the appropriate conditions. There are recognized differences in the physiological role of prolactin between rodents and humans. Since there are, at present, no adequate epidemiological studies, the relevance to human mammary cancer risk from prolonged exposure to perphenazine and other antipsychotic drugs is not known.

Amitriptyline: In manic-depressive psychosis, depressed patients may experience a shift toward the manic phase if they are treated with an antidepressant. Patients with paranoid symptomatology may have an exaggeration of such symptoms. The tranquilizing effect of TRIAVIL seems to reduce the likelihood of this effect. When amitriptyline HCl is given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required. Paralytic ileus may occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs.

Caution is advised if patients receive large doses of ethchlorvynol concurrently. Transient delirium has been reported in patients who were treated with 1 g of ethchlorvynol and 75-150 mg of amitriptyline HCl.

Amitriptyline HCl may enhance the response to alcohol and the effects of barbiturates and other CNS depressants.

Concurrent administration of amitriptyline HCl and electroshock therapy may increase the hazards associated with such therapy. Such treatment should be limited to patients for whom it is essential. Discontinue several days before elective surgery if possible. Elevation and lowering of blood sugar levels have both been reported. Use with caution in patients with impaired liver function.

ADVERSE REACTIONS: Similar to those reported with either constituent alone.
Perphenazine: Extrapyramidal symptoms (opisthotonus, oculogyric crisis, hyperreflexia, dystonia, akathisia, acute dyskinesia, ataxia, parkinsonism) have been reported and can usually be controlled by the concomitant use of effective antiparkinsonian drugs and/or by reduction in dosage, but sometimes persist after discontinuation of the phenothiazine.

Tardive dyskinesia may appear in some patients on long-term therapy or may occur after drug therapy with phenothiazines and related agents has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. Symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw. Involuntary movements of the extremities sometimes occur. There is no known treatment for tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms. It is advised that all antipsychotic agents be discontinued if the above symptoms appear. If treatment is reinstituted, or dosage of the particular drug increased, or another drug substituted, the syndrome may be masked. Fine vermicular movements of the tongue may be an early sign of the syndrome. The full-blown syndrome may not develop if medication is stopped when lingual vermiculation appears.

Other side effects are skin disorders (photosensitivity, itching, erythema, urticaria, eczema, up to exfoliative dermatitis); other allergic reactions (asthma, laryngeal edema, angioneurotic edema, anaphylactoid reactions); peripheral edema; reversed epinephrine effect; hyperglycemia; endocrine disturbances (lactation, galactorrhea, gynecomastia, disturbances of menstrual cycle); altered cerebrospinal fluid proteins; paradoxical excitement; hypertension, hypotension, tachycardia, and ECG abnormalities (quinidine-like effect); reactivation of psychotic processes; catatonic-like states; autonomic reactions, such as dry mouth or salivation, headache, anorexia, nausea, vomiting, constipation, obstipation, urinary frequency or incontinence, blurred vision, nasal congestion, and a change in pulse rate; other adverse reactions reported with various phenothiazine compounds, but not with perphenazine, include grand mal convulsions, cerebral edema, polyphagia, pigmentary retinopathy, photophobia, skin pigmentation, and failure of ejaculation.

The phenothiazine compounds have produced blood dyscrasias (pancytopenia, thrombocytopenic purpura, leukopenia, agranulocytosis, eosinophilia); and liver damage (jaundice, biliary stasis).

Pigmentation of the cornea and lens has been reported to occur after long-term administration of some phenothiazines. Although it has not been reported in patients receiving TRIAVIL, the possibility that it might occur should be considered.

Hypnotic effects, lassitude, muscle weakness, and mild insomnia have also been reported.

Amitriptyline: Note: Listing includes a few reactions not reported for this drug, but which have occurred with other pharmacologically similar tricyclic antidepressant drugs and must be considered when amitriptyline is administered. *Cardiovascular:* Hypotension; hypertension; tachycardia; palpitation; myocardial infarction; arrhythmias; heart block; stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth; blurred vision; disturbance of accommodation; increased intraocular pressure; constipation; paralytic ileus; urinary retention; dilatation of urinary tract. *Allergic:* Skin rash; urticaria; photosensitization; edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis; leukopenia; eosinophilia; purpura; thrombocytopenia. *Gastrointestinal:* Nausea; epigastric distress; vomiting; anorexia; stomatitis; peculiar taste; diarrhea; parotid swelling; black tongue. Rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido; elevated or lowered blood sugar levels. *Other:* Dizziness, weakness; fatigue; headache; weight gain or loss; increased perspiration; urinary frequency; mydriasis; drowsiness; alopecia. *Withdrawal Symptoms:* Abrupt cessation after prolonged administration may produce nausea, headache, and malaise. These are not indicative of addiction.

OVERDOSAGE: All patients suspected of having taken an overdosage should be admitted to a hospital as soon as possible. Treatment is symptomatic and supportive. However, the intravenous administration of 1–3 mg of physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage of physostigmine should be repeated as required particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine. On this basis, in severe overdosage with perphenazine-amitriptyline combinations, symptomatic treatment of central anticholinergic effects with physostigmine salicylate should be considered.

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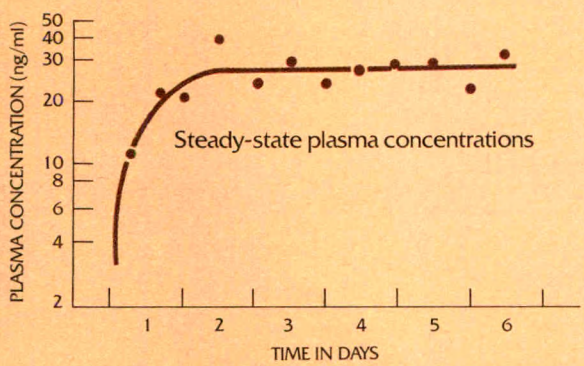
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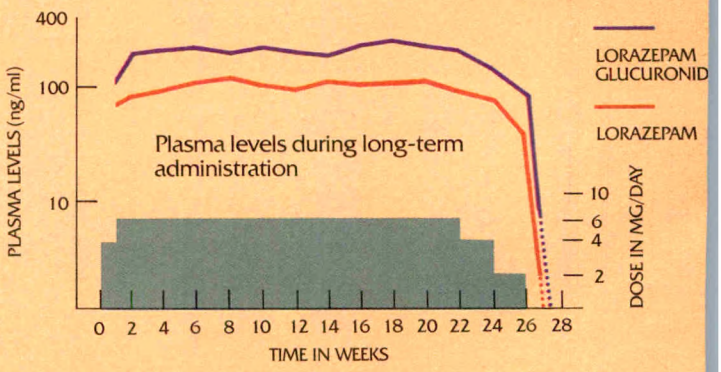
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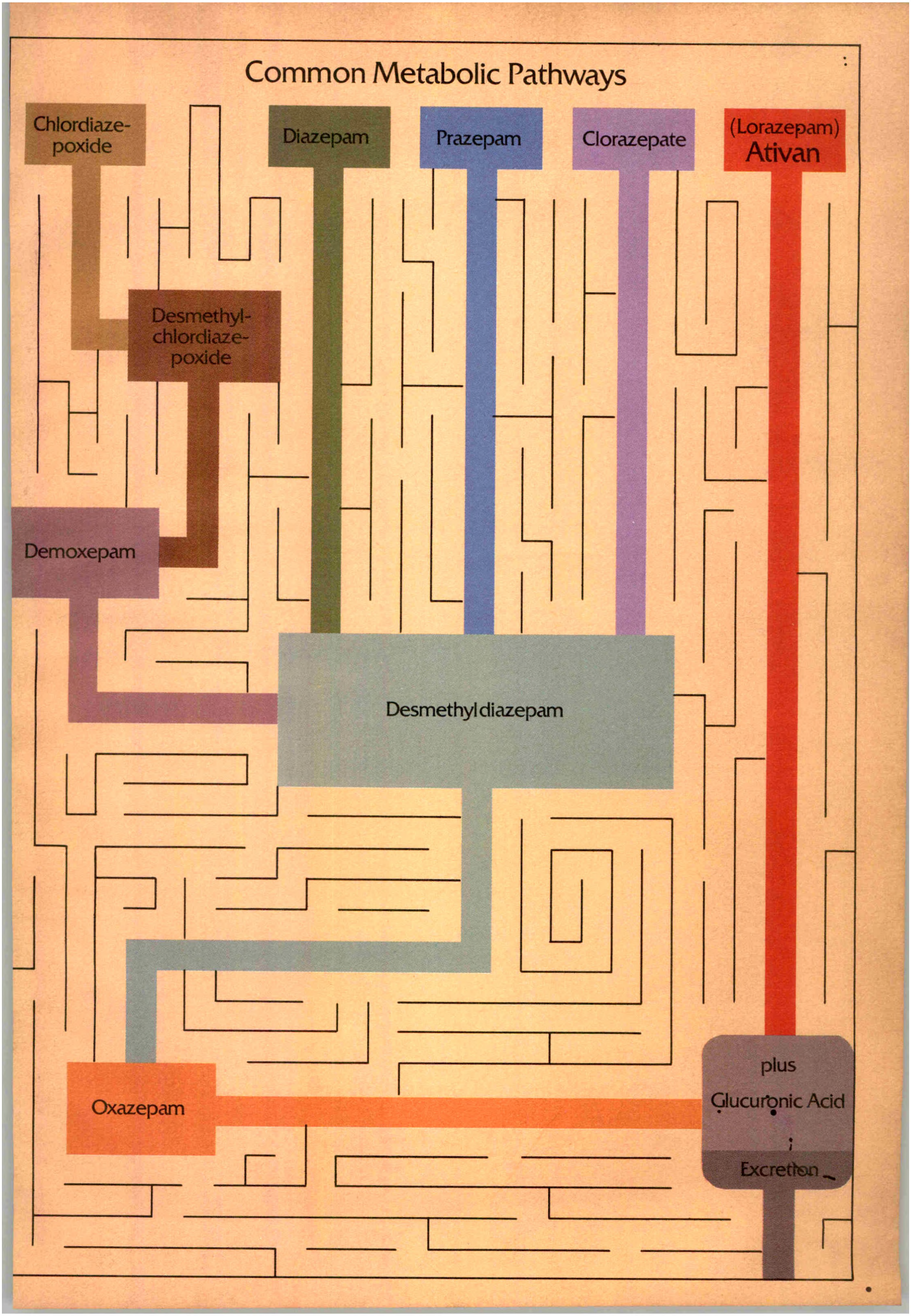


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Contraindications: Ativan is contraindicated in patients with known sensitivity to the benzodiazepines or with acute narrow-angle glaucoma.

Warnings: Ativan is not recommended for use in patients with a primary depressive disorder or psychosis. As with all patients on CNS-acting drugs, patients receiving lorazepam should be warned not to operate dangerous machinery or motor vehicles and that their tolerance for alcohol and other CNS depressants will be diminished.

Physical and Psychological Dependence: Withdrawal symptoms similar in character to those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepine drugs. These symptoms include convulsions, tremor, abdominal and muscle cramps, vomiting and sweating. Addiction-prone individuals, such as drug addicts and alcoholics, should be under careful surveillance when receiving benzodiazepines because of the predisposition of such patients to habituation and dependence.

Precautions: In patients with depression accompanying anxiety, a possibility for suicide should be borne in mind.

For elderly or debilitated patients, the initial daily dosage should not exceed 2 mg in order to avoid oversedation.

Ativan dosage should be terminated gradually since abrupt withdrawal of any antianxiety agent may result in symptoms similar to those for which patients are being treated: anxiety, agitation, irritability, tension, insomnia and occasional convulsions.

The usual precautions for treating patients with impaired renal or hepatic function should be observed.

In patients where gastrointestinal or cardiovascular disorders coexist with anxiety, it should be noted that lorazepam has not been shown to be of significant benefit in treating the gastrointestinal or cardiovascular component.

Esophageal dilation occurred in rats treated with lorazepam for more than one year at 6 mg/kg/day. The no-effect dose was 1.25 mg/kg/day (approximately 6 times the maximum human therapeutic dose of 10 mg per day). The effect was reversible only when the treatment was withdrawn within two months of first observation of the phenomenon. The clinical significance of this is unknown. However, use of lorazepam for prolonged periods and in geriatric patients requires caution, and there should be frequent monitoring for symptoms of upper G.I. disease.

Safety and effectiveness of Ativan in children of less than 12 years have not been established.

Essential Laboratory Tests: Some patients on Ativan have developed leukopenia and some have had elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function tests are recommended for patients on long-term therapy.

Clinically Significant Drug Interactions: The benzodiazepines including Ativan produce CNS depressant effects when administered with such medications as barbiturates or alcohol.

Carcinogenesis and Mutagenesis: No evidence of carcinogenic potential emerged in rats during an 18-month study with Ativan. No studies regarding mutagenesis have been performed.

Pregnancy: Reproductive studies in animals were performed in mice, rats, and two strains of rabbits. Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastroschisis, malformed skull and microphthalmia) were seen in drug-treated rabbits without relationship to dosage. Although all of these anomalies were not present in the concurrent control group, they have been reported to occur randomly in historical controls. At doses of 40 mg/kg and higher, there was evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lower doses.

The clinical significance of the above findings is not known. However, an increased risk of congenital malformations associated with the use of minor tranquilizers (chlordiazepoxide, diazepam and meprobamate) during the first trimester of pregnancy has been suggested in several studies. Because the use of these drugs is rarely a matter of urgency, the use of lorazepam during this period should almost always be avoided. The possibility that a woman of child-bearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant, they should communicate with their physician about the desirability of discontinuing the drug.

In humans, blood levels obtained from umbilical cord blood indicate placental transfer of lorazepam and lorazepam glucuronide.

Nursing Mothers: It is not known whether oral lorazepam is excreted in human milk like the other benzodiazepine tranquilizers. As a general rule, nursing should not be undertaken while a patient is on a drug since many drugs are excreted in human milk.

Adverse Reactions: Adverse reactions, if they occur, are usually observed at the beginning of therapy and generally disappear on continued medication or upon decreasing the dose. In a sample of about 3,500 anxious patients, the most frequent adverse reaction to Ativan is sedation (15.9%), followed by dizziness (6.9%), weakness (4.2%) and unsteadiness (3.4%). Less frequent adverse reactions are disorientation, depression, nausea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, eye function disturbance, together with various gastrointestinal symptoms and autonomic manifestations. The incidence of sedation and unsteadiness increased with age.

Small decreases in blood pressure have been noted but are not clinically significant, probably being related to the relief of anxiety produced by Ativan.

Overdosage: In the management of overdosage with any drug, it should be borne in mind that multiple agents may have been taken.

Manifestations of Ativan overdosage include somnolence, confusion and coma. Induced vomiting and/or gastric lavage should be undertaken followed by general supportive care, monitoring of vital signs and close observation of the patient. Hypotension, though unlikely, usually may be controlled with Levarterenol Bitartrate Injection, U.S.P. Caffeine and Sodium Benzoate Injection, U.S.P. may be used to counteract CNS depressant effects. The usefulness of dialysis has not been determined.

Dosage and Administration: Ativan is administered orally. For optimal results, dose, frequency of administration and duration of therapy should be individualized according to patient response. To facilitate this, scored 1.0 and 2.0 mg tablets are available.

The usual range is 2 to 6 mg/day given in divided doses, the largest dose being taken before bedtime, but the daily dosage may vary from 1 to 10 mg/day. For anxiety, most patients require an initial dose of 2 to 3 mg/day given b.i.d. or t.i.d.

For insomnia due to anxiety or transient situational stress, a single daily dose of 2 to 4 mg may be given, usually at bedtime.

For elderly or debilitated patients, an initial dosage of 1 to 2 mg/day in divided doses is recommended, to be adjusted as needed and tolerated.

The dosage of Ativan should be increased gradually when needed to help avoid adverse effects. When higher dosage is indicated, the evening dose should be increased before the daytime doses.

How Supplied: Ativan (lorazepam) is available in scored 1.0 and 2.0 mg tablets in bottles of 100.

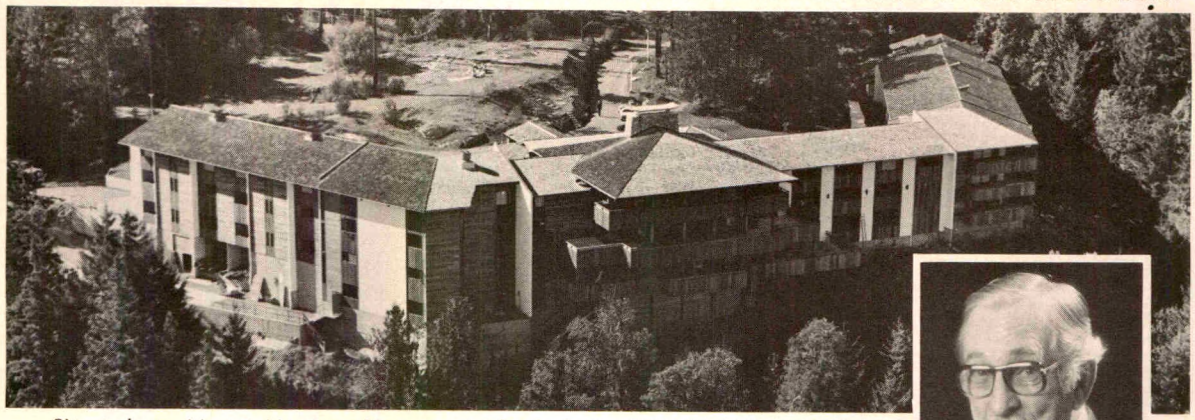
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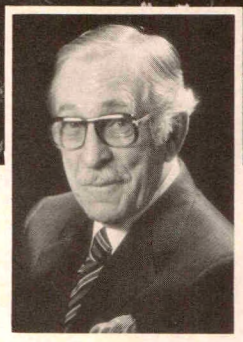
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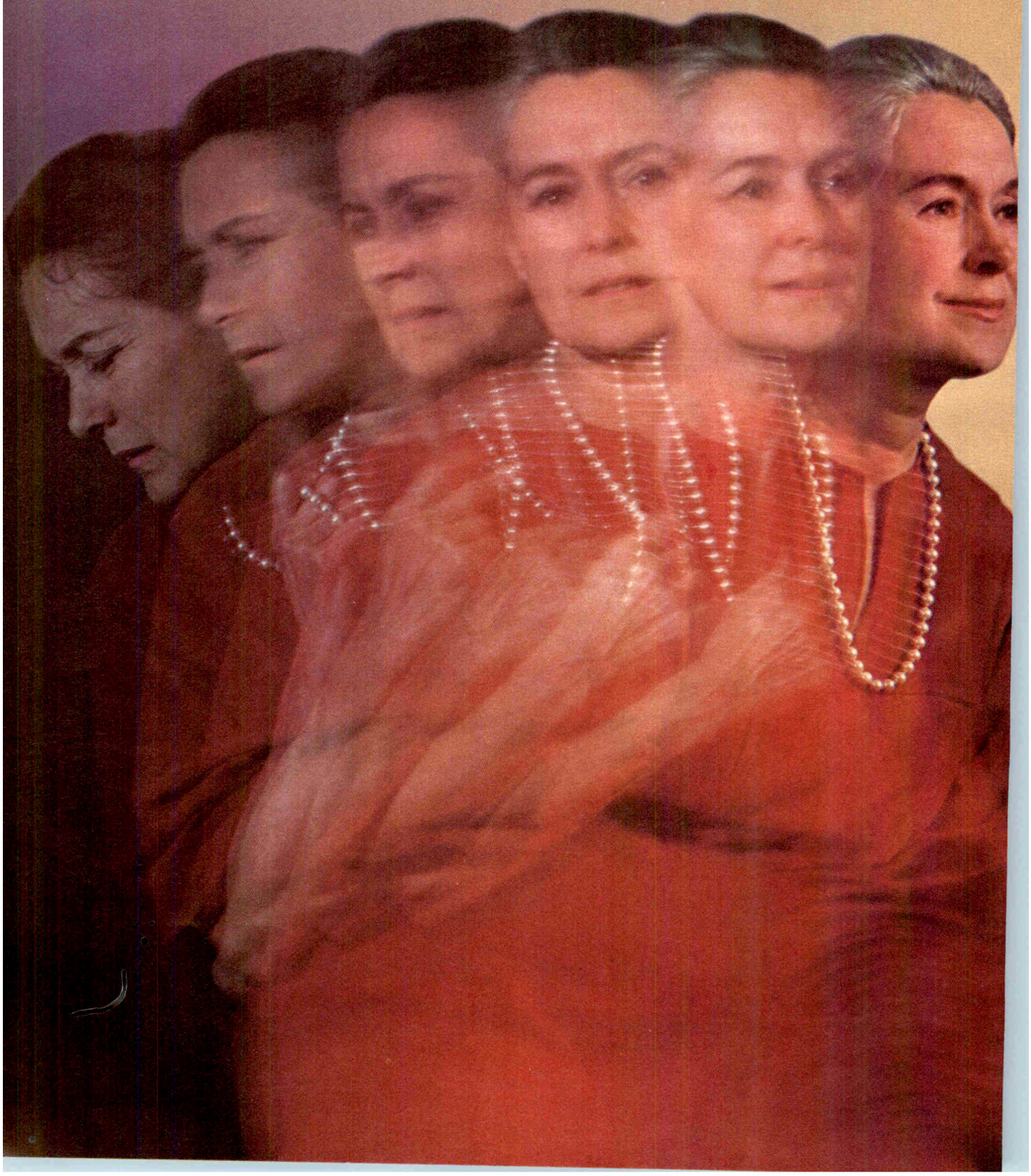
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Chlorprothixene	Moderate
Thiothixene	Moderate
Haloperidol	High
MELLARIL® (thioridazine)	Low

*Based on antipsychotic dosage ranges

References

1. Van Putten T: The rising rehospitalization rate of psychiatric patients. Scientific Exhibit, American Psychiatric Association, 130th Annual Meeting, Toronto, Canada, May 2–6, 1977.

2. Byck R: Drugs and the treatment of psychiatric disorders, in Goodman LS, Gilman A (eds): *The Pharmacological Basis of Therapeutics*, ed. 5. New York, Macmillan Publishing Co, Inc, 1975, pp 170–171.

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It should be noted that efficacy, indications and untoward effects have varied with the different phenothiazines. It has been reported that old age lowers the tolerance for phenothiazines; the most common neurologic side effects are parkinsonism and akathisia, and the risk of agranulocytosis and leukopenia increases. The following reactions have occurred with phenothiazines and should be considered whenever one of these drugs is used. *Autonomic Reactions*—Miosis, obstipation, anorexia, paralytic ileus. *Cutaneous Reactions*—Erythema, exfoliative dermatitis, contact dermatitis. *Blood Dyscrasias*—Agranulocytosis, leukopenia, eosinophilia, thrombocytopenia, anemia, aplastic anemia, pancytopenia. *Allergic Reactions*—Fever, laryngeal edema, angioneurotic edema, asthma. *Hepatotoxicity*—Jaundice, biliary stasis. *Cardiovascular Effects*—Changes in terminal portion of electrocardiogram, including prolongation of Q-T interval, lowering and inversion of T-wave, and appearance of a wave tentatively identified as a bifid T or a U wave have been observed with phenothiazines, including Mellaril (thioridazine); these appear to be reversible and due to altered repolarization, not myocardial damage. While there is no evidence of a causal relationship between these changes and significant disturbance of cardiac rhythm, several sudden and unexpected deaths apparently due to cardiac arrest have occurred in patients showing characteristic electrocardiographic changes while taking the drug. While proposed, periodic electrocardiograms are not regarded as predictive. Hypotension, rarely resulting in cardiac arrest. *Extrapyramidal Symptoms*—Akathisia, agitation, motor restlessness, dystonic reactions, trismus, torticollis, opisthotonus, oculogyric crises, tremor, muscular rigidity, and akinesia. *Persistent Tardive Dyskinesia*—Persistent and sometimes irreversible tardive dyskinesia, characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and sometimes of extremities may occur on long-term therapy or after discontinuation of therapy, the risk being greater in elderly patients on high-dose therapy, especially females; if symptoms appear, discontinue all antipsychotic agents. Syndrome may be masked if treatment is reinstituted, dosage is increased, or antipsychotic agent is switched. Fine vermicular movements of tongue may be an early sign, and syndrome may not develop if medication is stopped at that time. *Endocrine Disturbances*—Menstrual irregularities, altered libido, gynecomastia, lactation, weight gain, edema, false positive pregnancy tests. *Urinary Disturbances*—Retention, incontinence. *Others*—Hyperpyrexia; behavioral effects suggestive of a paradoxical reaction, including excitement, bizarre dreams, aggravation of psychoses, and toxic confusional states; following long-term treatment, a peculiar skin-eye syndrome marked by progressive pigmentation of skin or conjunctiva and/or accompanied by discoloration of exposed sclera and cornea; stellate or irregular opacities of anterior lens and cornea; systemic lupus erythematosus-like syndrome.

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For brief summary, please see preceding page.

Am J Psychiatry 136:4B, April 1979

THE AMERICAN JOURNAL OF PSYCHIATRY

Editorial: Diverse Research Strategies for Depression and Alcoholism

MOST OF US vaguely recall the poem derived from a Hindu fable that begins, "It was six men from Indostan, to learning much inclined who went to see the elephant (though all of them were blind), that each by observation might satisfy his mind." Each man encountered a single part of the elephant and each described the elephant totally in terms of what he "saw." Hence they "disputed loud and long, each in his opinion exceeding stiff and strong, though each was partly in the right and all of them were wrong."

Recently, when rereading the poem I learned that it was written by John Saxbe, an American poet in the mid 19th century. It was not a child's poem but rather a satire upon parochial religiosity, for it ends with a moral: "So, oft in theologic wars the disputants, I ween, rail on in utter ignorance of what each other mean, and prate about an elephant not one of them has seen."

The moral remains pertinent to our times and to our profession. The elephant we encounter is man with all of his genetic and cultural heterogeneity, and we face the task of understanding his functioning or malfunctioning as an individual and as a member of a family and of a large number of overlapping and sometimes conflicting social groups. It is not likely that any of us can yet fully visualize the elephant, much less understand him completely.

We have advanced considerably beyond the blind men from Indostan insofar as we have new technologies that amplify our senses and scientific methodology with which we can test and replicate our findings. Our actual progress will depend on the degree to which we listen to each other's findings, respect them, and try to integrate them into a comprehensible whole.

Probably no mental illnesses have been studied more thoroughly, with more diverse methods, and with greater profit to our patients than the affective disorders. Yet problems remain and many of these are addressed in the 20 papers on depression presented in this issue. Six employ epidemiological methods, 2 deal with the problems of diagnosis in the very young and the aged, 4 with treatment, 4 with the use of lithium, and 4 with aspects of the biology of depression. There are also 10 papers on alcoholism: 3 deal with the relationship of alcoholism to depression, 1 with the nongenetic familial transmission of alcoholism, 2 with neurophysiological deficits, 2 with issues related to treatment, and 2 with special problems of alcoholic women. Most of the papers are multiauthored and many are the product of interdisciplinary team efforts.

An epidemiological study of depression by Warheit obtained interview data on mental and physical well-being, family and social resources, health and human services utilization, and related social and demographic variables from a large adult population. Three years later he determined through reinterviews the degree to which depressive symptomatology appeared. He found that the best predictor of ensuing depression was the degree to which it existed initially. This suggests that depressive symptomatology may be a trait; whether this is biological, characterological, or both is not known but it seems to predispose such individuals to life events that may exacerbate their depression. Not so surprisingly, persons in low socioeconomic groups are most vulnerable; they have fewer resources to begin with and experience more losses. The study documents the degree to which per-

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sonal, familial, and interpersonal resources can mitigate some of the adverse effects of losses. Motto's epidemiological research generates two models of people at especially high risk for suicidal behavior. He interviewed 3,000 patients admitted to a psychiatric inpatient unit because of a depressive or suicidal state. This population has an expected suicide rate of 1%–2% a year. Two groups were found that committed suicide at a rate above 4% during the follow-up period. One of these was males under 40, the other consisted of individuals who were stable in their health, work, and marriage but who had imposed on them a significant loss or threat of loss. These two models fall far short of the number required to include the bulk of the very high-risk suicidal persons, but it is an encouraging beginning and demonstrates a research method that may be continually useful in generating still other models. Dunner and coworkers examined the question of the degree to which life events can precipitate episodes of illness in patients with bipolar disease. Fifty percent of such patients report significant life events within three months of their first episode of illness. Once the pattern of the illness was established it seemed to achieve its own autonomy, and life events were not significantly related to subsequent episodes. It might be expected that the 50% who failed to report external precipitants might be more heavily genetically loaded, but Dunner's study of the relatives of these patients showed this not to be the case.

Goldberg and coworkers addressed the question of whether depressed mood is a precursor of serious illness. Over a two-year period a large number of subjects from two areas of the country were sampled to assess their physical health and degree of depression. A year later the subjects were reinterviewed. Persons who died or developed serious physical illness requiring hospitalization had no more depressive symptoms before becoming ill than the controls who remained well.

The two remaining epidemiological papers deal with more specialized populations. Helzer and coworkers conducted a followup study of Viet Nam combat veterans who had been depressed a year after their return and found the depression to be short-lived. Preservice experience apparently plays a stronger role in long-term psychological adjustment. Beckman and coworkers found that depression occurs in the wives of nuclear submarine personnel whose husbands are essentially incommunicado while at sea for three-month intervals twice yearly. The problem is important not only to the Navy but is of considerable theoretic interest because the phases of maladaptation that lead to depression among these women are described.

Depressive symptomatology may differ from the usual in both the elderly and the very young. Cavenar and coworkers offer brief case histories of four patients with depression mimicking organic brain syndrome that responded dramatically to pharmacotherapy. Philips describes the symptoms of depression in infancy, preschool years, and the early school years. Acknowledging the diversity of etiological factors that may contribute to childhood depression, he highlights the interpersonal ones. Depressed parents are unable to meet the needs of the child and this may result in depression in the child.

In the four papers dealing with therapy, Rivinus and Harmatz report on manic-depressive illness in the institutionally retarded. The age of occurrence, the frequency in their families, and their response to lithium is similar to that of patients with normal intelligence. Nelson and coworkers report that tricyclics may exaggerate the existing delusions of unipolar delusional depressed patients. They respond well to the addition of phenothiazines or to ECT. Avery and Lubrano reconsider data from a large Italian study that compares ECT with imipramine in high doses. These data show that ECT is superior for delusional depressions and for the most severe clinical depressions; it is inferior for neurotic and reactive depressions. The paper brings to mind the thought that the usual indications for ECT are patients who have had drug failures, those whose occupations or professions do not permit prolonged hospitalization, the severely depressed and suicidal, the aged, and those who are too medically ill to withstand the adverse medical effects of drugs. We tend to avoid ECT because we assume that the risks outweigh the benefits, but if this really is the case, why do we use it on the old and frail? The risk benefit ratio for modern ECT compared with drugs requires reevaluation.

The article by Weissman and coworkers on the efficacy of combined pharmacotherapy and psychotherapy in the treatment of acute depressive disorders is most stimulating. Common sense suggests that combined treatment should be more effective than either component alone because a biological diathesis for depression is well established, antecedent psychosocial stresses are frequently found, and coexisting problems in interpersonal relationships are the rule in depressed patients. The efficacy of drug treatment has been well established in a large number of controlled clinical trials. The efficacy of insight-oriented psychotherapy has not been as rigorously documented. Recently there have been claims that cognitive therapy is more effective than imipramine in depressed outpatients, but it is not yet clear whether this would be true for patients whose illness is of the type or severity to require hospitalization. While combined therapy is commonly used, pharmacotherapy and psychotherapy remain competitive ideologically. Moreover, there is surprisingly little empirical evidence that the effects are additive or synergistic.

In this 16-week study by Weissman, amitriptyline, weekly interpersonal psychotherapy that focused on the social context of the depression, and their combination was compared with psychotherapy, which was not scheduled but was made available if the patient called. All of the active treatments were better than the nonscheduled treatment. By all criteria the combined treatment group did better than either treatment alone. We can hope that there will be more well-designed research with other populations, other illnesses, other drugs, and other forms of psychological treatment alone and in combination. Research on the combination of cognitive therapy and pharmacotherapy for depression is certainly in order.

Among the 4 papers dealing with biology, that by Oliveau and Willmuth finds that noninvasive electromyography of the facial muscles is a sensitive indicator of affect-laden memories and images. It is, however, unable to detect differences between depressed and nondepressed subjects. Lykouras and coworkers find a correlation between plasma levels of cyclic adenosine monophosphate and mood in manic-depressive patients but note that since other factors such as anxiety may also influence these levels the exact association warrants more detailed exploration. Garfinkel and coworkers employed a peripheral decarboxylase inhibitor (carbidopa) to separate central nervous system metabolites of the biogenic amines from peripheral metabolites. In a carefully controlled metabolic study they obtained data that suggest a distinct profile of indolamine and catecholamine metabolism in unipolar depressed subjects but not in bipolar patients. They submit the hypothesis that the fundamental defect in these patients may lie in the reduced capacity of their brains to take up tryptophan, the amino acid precursor of serotonin. They suggest that lithium, which increases brain tryptophan uptake, might be combined with tryptophan in clinical trials of the antidepressant effectiveness of amino acid. A neuroendocrinological study by Brown and coworkers confirms previous findings that depressed patients show resistance to the suppression of cortisol secretion following dexamethasone. Their data show a general trend for an association between a good response to tricyclic treatment and the capacity to escape dexamethasone suppression.

Among the four papers dealing with the use of lithium, that of Steinberg suggests, on the basis of retrospective studies of lithium responders, that there is an MMPI profile that may help to prospectively predict them. The article by Belmaker and coworkers demonstrates that lithium diminishes the accelerated heart rate which usually accompanies mental arithmetic tasks without affecting the resting heart rate. They hypothesize that this correlates with the finding that lithium does not inhibit basal adenylcyclase activity but does inhibit noradrenaline-induced rises. They suggest that lithium may be useful in case of paroxysmal atrial tachycardia in patients with healthy hearts. The paper by Kocsis and Stokes retrospectively examines the quality of the outcome in a large number of patients maintained on lithium for a mean of somewhat more than four years. Among other things they find that there are lower plasma lithium levels in patients who experience both depressive and manic episodes. They suggest that this may reflect the practice of some patients to decrease lithium when depression occurs, thus creating a vicious cycle with rebound mania. A longitudinal case report by Ereshefsky

and coworkers suggests that red blood cell lithium concentrations correlate better with suppression of tardive dyskinesia, neurotoxicity, and changes in manic symptomatology than does plasma lithium. Further study of the utility of lithium for the distressing problem of tardive dyskinesia based on erythrocyte levels seems warranted.

There are many reasons for suspecting a relationship between alcoholism and depression. Keeler and coworkers studied the prevalence of depression after alcohol withdrawal in a population of 35 male alcoholics about a week after admission to a VA alcoholism treatment service. They found that the prevalence depended on the method used for diagnosis. On clinical grounds 8.6% were considered depressed, with the Hamilton Rating Scale it was 28%, the MMPI depression scale 43%, and the Zung Self-Rating Scale 66%. These authors emphasize that the diagnosis should be made solely on clinical grounds and that rating scales can be used to measure change once the diagnosis has been made. This is important because if depression is overdiagnosed in alcoholics, tricyclic antidepressants and lithium can be overused for treatment and prophylaxis. The study of Hamm and coworkers deals with military personnel referred solely because of excessive drinking. They employed a Hamilton Rating Scale and a self-rating depression scale and found the level of depression to be much lower than that reported by others for alcoholics. They emphasize that their patients were all on active duty and were not referred because of psychiatric problems. They suggest that the low incidence of depression in their alcoholics may more closely resemble that of alcoholics in the population at large because the majority of such patients do not seek treatment. Dunner and coworkers studied alcoholism in a large population of well-defined manic-depressives. About 20% of the men had alcohol-related problems but none of the women did. The relatives of manic-depressive patients were more prone to alcoholism than are those of the control group. Although genetic data for transmission of alcoholism are sound, it is quite clear that genetics alone cannot account for the transmission. Roland and coworkers found that families which maintained their patterns and rituals even in the face of a serious parental alcoholic problem are less likely to transmit alcoholism into the children's generations than those whose patterns are disrupted.

Hill and coworkers used a CT scanner and the Halstead-Reitan Neuropsychological Battery and found surprisingly little evidence of ventricular enlargement and cerebral atrophy but, nonetheless, evidence of neuropsychological damage. Fine and Steer examined 100 men arrested for drunken driving several days after they had stopped drinking and found evidence of short-term spatial memory deficits that might influence their capacity to remember road signs, traffic alerts, or emergency signals. Thirty percent had previously been arrested for drunken driving, but only 10% had been treated for alcoholism.

Two papers by Schuckit and Morrissey address some problems of alcoholic women. Fourteen percent have primary affective disease and many abuse other drugs. Two-thirds of these women had received prescriptions of hynoptic and antianxiety drugs while actually abusing the drugs. They urge that hypnotics, stimulants, and antianxiety drugs should almost never be prescribed to outpatient alcoholics.

Two papers deal with treatment: Mathew and coworkers suggest that the craving of sober alcoholics for liquor is largely a conditioned response to environmental stress, that it diminishes with continued abstinence, and that it can be relieved by psychological methods. The article by O'Leary and coworkers emphasizes that the degree to which the staff rates patients as high on interpersonal attractiveness is the major determinant in their acceptance or rejection for continued treatment.

This brief summary and comments hardly do justice to the quality and diversity of the 30 papers. I hope it transmits the sense of vigor with which research is being conducted. Psychiatry, so long dominated by lofty and competing theoretical structures, requires honest brick-laying to create the complex home in which we can comfortably work, live, and evolve.

I conducted an informal and nonscientific survey of my colleagues and was unable to find any who had ever read every article in a journal issue. Nor had I, until confronted with the task of writing this editorial. I not only profited from

learning about work in areas that I have tended to ignore but even enjoyed it. I suggest that the readers of the journal do the same for at least one issue. It might get to be a profitable habit. There is much to be learned by listening to each other.

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Life Events, Coping, Stress, and Depressive Symptomatology

BY GEORGE J. WARHEIT, PH.D.

The author presents data on the relationships between life events, coping resources, and depressive symptomatology. A probability sample (N=517) was interviewed three years apart. Respondents with high life-event scores had significantly more depressive symptomatology than those with low scores. Those with personal, familial, and interpersonal resources had significantly less depressive symptomatology than those without such resources in both the low- and high-life-event groups. The best predictor of Time 2 depressive symptom scores was Time 1 symptom scores; losses and resources were also statistically significant factors. These findings demonstrate the complex interrelatedness of life events, coping resources, and depressive symptomatology.

THE RELATIONSHIPS between social, psychological, and other environmental factors and illness have been of research interest to medical and social scientists for a long time. This interest has accelerated in recent decades as investigators from a wide range of disciplines have attempted to identify the processes by which stressors act as precursors to physical and/or mental disorders. Although there have been almost as many definitions of stress as there have been researchers, there is a common theme in them. Stress is generally conceptualized as the altered state of an organism produced by agents in the psychological, social, cultural, and/or physical environments. It is assumed that this altered state, when unmitigated, produces deleterious effects on the physical and/or mental well-being of affected individuals.

Among medical scientists, the early work of Cannon (1) was very influential. His pioneering efforts to detail the relationships between emotional states such as fear, anger, pain, and anxiety and changes in body function provided a model for early scientific inquiry. In psychiatry, Adolf Meyer (2) emphasized the role of life events in the development of physical and mental disorders, and Selye's work (3, 4) has made very important theoretical contributions to our understanding of the physiological adaptations to stress. Hinkle (5, 6), Wolff (7), and their colleagues working in the area of psychosomatic medicine also made early contributions as they investigated the links between stressors and illness. Recently, many researchers have attempted to establish the qualitative and quantitative relationships between particular classes of life events and illness behavior. The efforts of Rahe and Holmes (8, 9), Paykel and associates (10, 11), Dohrenwend and Dohrenwend (12, 13), Myers and associates (14), and Brown and associates (15) are particularly well known.

The research findings dealing with stress and illness are voluminous, comprehensive, and broadly published. The number of empirical investigations reporting on coping behaviors as they relate to stress and illness are quite limited, although an increasing number of publications reflect a growing interest in the issue (16–21).

The first purpose of this paper is to report empirical data on life events, coping resources, stress, and depressive symptomatology. The second objective is to analyze these same variables within the context of longitudinal data that permit comparisons of depressive symptomatology before and after the occurrence of 23 loss-related life events.

THEORETICAL MODELS

Early research relating life events to psychosocial stress and illness tended to regard the relationships in a simple stimulus-response, unicausal manner. This oversimplified model has more recently been refined to include coping and adaptation variables that are seen as intervening processes, buffering the individual from the damaging effects of stress-producing life events.

Although stress-illness models have become more sophisticated in the past decade, many of them still have serious theoretical deficiencies, e.g., stressful events, coping, and illness continue to be conceptualized as static and occurring within a closed system. As such, these models do not provide a theoretical understanding of the dynamic interactions of an organism as it influences and is influenced by its environments. The scientific problems posed by closed models are not

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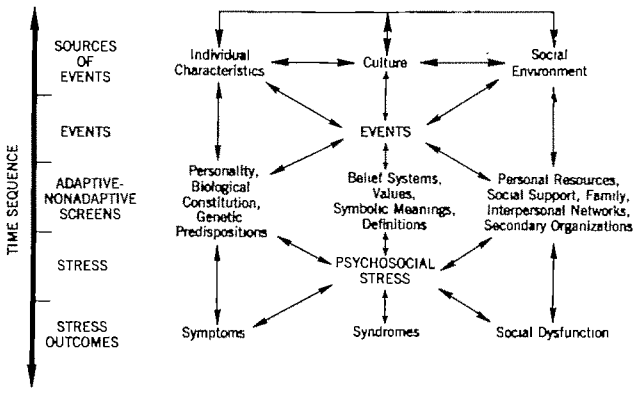
confined to their theoretical inadequacies; they have inherent analytic weaknesses as well. Statistical models that assume unidirectional causality, e.g., path analysis, are basically inadequate since they do not provide for the testing of reciprocal interactions between the variables being analyzed. As a consequence, they do not take into account the systemic relationships inherent in the real world as organisms affect and are affected by their psychological, social, ecological, and cultural milieus.

The limitations of these early models led to the formulation shown in figure 1. This model constituted the theoretical framework that guided our efforts to test the relationships between life events, coping resources, stress, and depressive symptomatology. Events are seen as arising from the following three sources: the individual's psychological and biological constitution, the culture, and the social environment. The events section of the model includes the specific events commonly associated with the literature on stress and crisis events.

The adaptive-nonadaptive screens represent the coping resources available to individuals as they attempt to meet the demands placed on them by life events. These screens include the individual's personality, biological constitution, and genetic predispositions. They also include coping screens provided by the culture. Parsons (22) described some of these as supra-empirical referents and suggested that their function was to afford culturally defined and transmitted systems of belief that give persons sources of *explanation* for events that cannot be accounted for by a society's logic or science. The culture also provides symbolic definitions that attach *meaning* to events. The personal resources available to individuals confronted by life events are seen as their socioeconomic statuses, families (both nuclear and extended), interpersonal networks, and the secondary organizations provided by the broader social environment.

Life events, stress, coping behaviors, and illness are most usefully studied as a process. Characteristically, when a crisis event occurs, we hypothesize that the individual's first line of defense is his or her psychological, physical, and genetic makeup. If the individual is unable to deal with the demands occasioned by the event, he or she then customarily seeks to extend his or her sources of support, most commonly calling for assistance from spouse, children, parents, or other family members. If these resources are unavailable or inadequate, the individual often turns to interpersonal networks, that is, to friends in whom he or she can confide and go to for help. The theory suggests, further, that when the person's individual and personal resources are insufficient to meet life-event demands, the next step involves seeking assistance from professional persons or agencies. If these prove to be inadequate, the individual may then turn to his or her culturally provided beliefs, values, and symbols. In practice, of course, when confronted with crisis-related demands, individuals normally seek to maximize all available resources simultaneously and in a complementary manner.

FIGURE 1
Life Events: Sources, Adaptations, and Outcomes



In the model, stress is conceived as an altered state of an organism and occurs when the demands on an individual exceed his or her capabilities to respond. The degree of stress experienced is a function of the number, frequency, intensity, duration, and priority of the demands viewed in apposition to coping resources. The demand-capability ratio is seen as dynamic (and, as such, fluctuating), multisystemic, and temporal. As shown in figure 1, psychosocial stress is conceptualized as being different from the events that may precipitate it. Events are seen as agents, and stress is viewed as an altered state of the organism. Stress is different from its outcomes, which are viewed as being symptoms, syndromes, and/or social dysfunction.

The theoretical model presented in figure 1 is designed to reflect the systemic nature of life events, coping mechanisms, stress, and stress outcomes as they occur in a temporal context.

DESIGN OF THE STUDY

The data reported are from a major two-stage epidemiological study of mental health needs and services that is being conducted in the southeastern United States. A statistical probability sample of 1,645 adults was interviewed by means of a 317-question interview schedule. The schedule included an extensive inventory of items, scales, and indexes designed to obtain data on mental and physical well-being, health, the utilization of health and other human service agencies, personal, familial, and social resources, and related social and demographic variables. A 517-person subsample of the original respondents was reinterviewed three years after the initial contact. The two interview schedules were essentially the same; one exception was the inclusion of a slightly modified version of the Paykel life event items in the follow-up instrument (10, 11).

In this research the independent variables were loss events and Time 1 depression scores. The intervening variables were personal coping resources, consisting of spouse, family, friends, socioeconomic status (SES), and professional persons and/or agencies. The dependent variable was Time 2 depression scores.

The depression scores were determined by use of a factor analytic scale conceptualized as containing five dimensions: affective symptoms related to lowered mood; somatic symptoms; altered patterns of psychobiological reactivity such as sleep, appetite, and libido; negative self-evaluation including lowered self-esteem, self-blame, suicidal ideation, and guilt; and an existential dimension typified by pessimism, despair, and a gloomy outlook of the future. Using this conceptualization, an 18-item scale was developed. Cronbach's alpha (as reported in 1951) was computed as a test of reliability; the alpha coefficient was 0.8399, well above the limit accepted for scalability. Since its development the scale has been administered to probability samples of approximately 5,000 adults living in the southeastern United States. Analysis of scores for various social and demographic groups in these samples also suggests high levels of scale reliability (23–25).

Data on coping resources were obtained as part of each survey. Both interviews included a large number of questions related to personal, familial, social, and cultural resources and their use.

The SES scores were calculated following the methodology suggested by the Bureau of the Census and modified by Nam and associates (26). The SES score was the average of ranks of an individual's education, income, and occupation scores. For purposes of this paper the sample scores were arranged in quintiles and were then placed into one of two categories. Those in the lowest three quintiles were put in the low SES group, and those in the top two quintiles in the high group.

Levels of loss were determined by scores on 23 items from the Paykel inventory. Only events occurring between the Time 1 and Time 2 interviews were included. The items selected were all associated with loss, a condition highly related to depressive symptoms and syndromes. The items included events such as the death of a child, spouse, and/or close family members; marital separation; and retirement. The 23 items were weighted using the method provided by Paykel and Uhlenhuth (11). Each respondent was assigned a total loss score on the basis of the number and kinds of losses. The weighted mean of the loss scores for the sample was 28.31. After preliminary analysis the sample was dichotomized into a low/moderate-loss group and a high-loss group; those with scores of 0–32.99 were placed in the former category, and those with scores of 33 or above into the latter group. Approximately one-third of the 517 respondents were in the high-loss group.

The methods of analysis included analysis of variance, t tests, and multiple regression equations.

FINDINGS

Relationship Between Depression Scores, Resources, and Losses

The findings relating depression scale score means, resources, and level of loss as determined by the 23 Paykel life-event items are presented in table 1. In every instance those in the high-loss category, regardless of the presence or absence of personal resources, had significantly higher depression scale scores than those in the low/moderate-loss group. The data also suggest that the presence of resources significantly mitigates the impact of losses associated with life events. All persons in the high-loss category had significantly higher depression scores than those in the low-loss group. Personal resources were shown to significantly lessen the impact of loss except when the resource was close relatives nearby.

The utilization of physical health services as determined by visits to physicians, clinics, and agencies was significantly greater for those in the high-loss group. The one exception was for those utilizing mental health services; score differences between the two groups were not statistically different. However, the numbers are quite small, and this finding requires cautious interpretation.

The presence of resources as determined by SES was significant within and between groups. The highest depression scale scores in the entire sample were found among those in the high-loss group without friends nearby to help and among those in the high-loss, low-SES groups. The data also show that individuals with low SES experienced more life-event losses than those with high SES. Approximately two-thirds (64.7%) of all those with low SES were in the high-loss group; by contrast, only 31.2% of those with high SES were in the high-loss group.

Estimation of Depression Scores from Losses and Resources

When the data were analyzed by means of a regression equation designed to estimate depression scores from losses and resources (including SES), the overall regression was significant at the $p<.001$ level. Low SES, the absence of a spouse, and high loss scores were most significantly correlated with high depression scores. The presence of friends was a significant variable ($p<.01$), as was the utilization of psychiatric services ($p<.01$). The cross-product interaction term formed from loss and SES was also significant ($p<.01$); the other interaction terms were not significant. The overall variance accounted for by the terms in the equation was 27.2%.

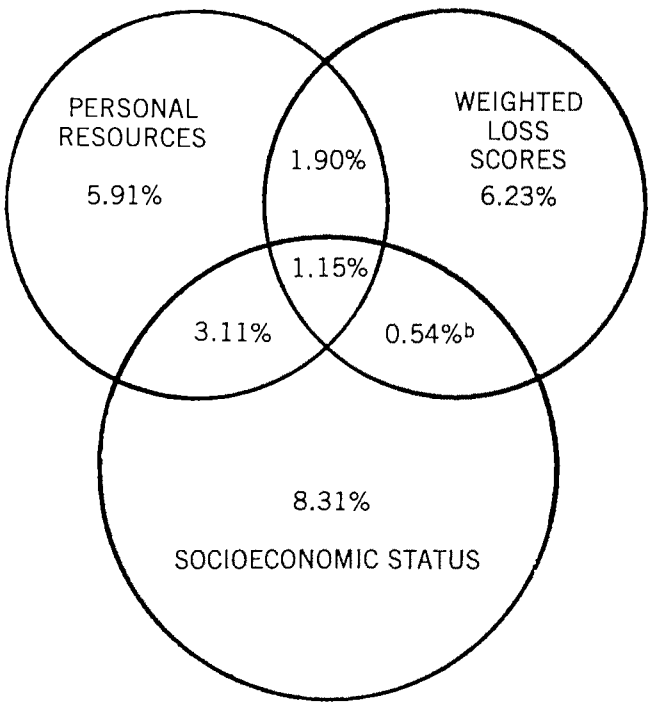
Figure 2 displays findings from a series of regressions that include terms for losses, resources, and SES. It shows that SES accounted for the greatest amount of variance; this variable accounted for 13.11% of the total explained variance. Of this total, 4.80% was shared with other resources and losses. The presence of resources accounted for a total of

TABLE 1
Mean Depression Scores, by Presence or Use of Resources and by Level of Loss Expressed (N=517)

Resource/Utilization	Low/Moderate Loss			High Loss			Significance of Low Versus High Loss
	N	Mean	SD	N	Mean	SD	
Spouse present							
Yes	248	12.4	7.9	99	17.7	10.4	.001
No	98	18.4[a]	10.2	72	22.1[b]	11.9	.05
Close relative nearby[c]							
Yes	246	14.6	9.3	134	20.0	11.3	.001
No	99	12.8	8.2	37	18.2	11.2	.01
Friends to talk about problems with[c]							
Yes	215	13.8	8.8	110	17.8	10.4	.001
No	124	14.6	9.1	59	23.0[d]	12.3	.001
Close friends nearby to help[c]							
Yes	268	13.9	8.5	121	17.6	9.9	.001
No	68	15.4	10.6	43	25.5[a]	13.4	.001
Physical health visits							
None	68	13.5	8.8	20	18.9	10.6	.05
1-3	174	12.8	7.9	67	17.3	9.7	.001
4 or more	104	16.6[b]	10.4	84	21.4	12.3	.01
Mental health utilization							
No	339	13.9	8.9	156	19.1	11.3	.001
Yes	7	20.6[b]	12.0	15	24.0	9.7	n.s.
SES[c]							
Low	43	17.8[a]	10.0	79	24.8[a]	12.4	.001
High	203	11.5	7.2	92	15.1	7.8	.001

[a]$p<.001$.
[b]$p<.05$.
[c]Ns do not total 517 in all cases since responses were not received for all subjects.
[d]$p<.01$.

FIGURE 2
The Explanation of Time 2 Depression Scores Provided by a Regression Model with Terms for Losses, Resources, and Socioeconomic Status[a]



[a]The entries represent percentages of Time 2 Depression score variance explained by the terms in the model. No control for race or Time 1 Depression scores is included.
[b]An interaction term for Socioeconomic Status × Loss is included in the model.

12.07% of the variance, with 6.16% of this being shared with losses and SES. The loss scores accounted for 9.82% of the variance, with 3.59% of this total shared with resources and SES. The data in figure 2 indicate that resources were more powerful predictors of depression scores than were the life-event-loss scores.

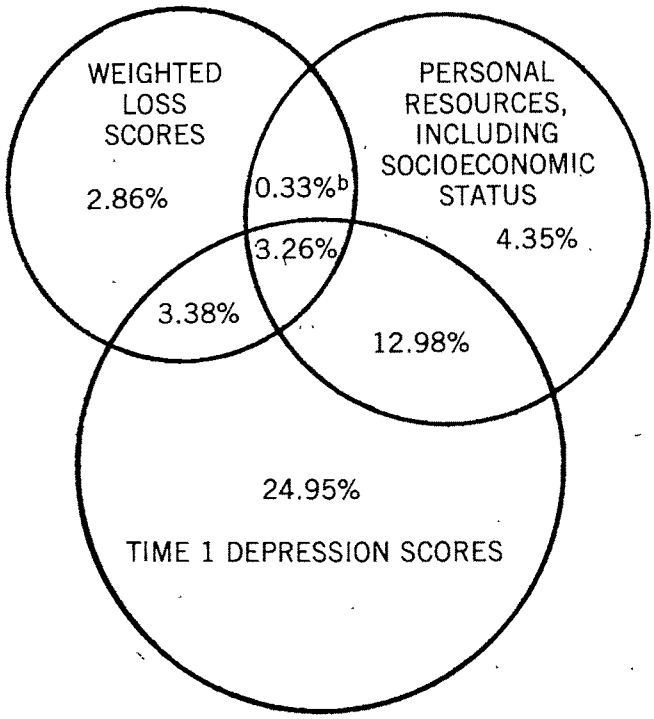
Relationship Between Time 1 and Time 2 Depression Scores, Losses, and Resources

Since there were depression data on each respondent at two points in time, it was possible by means of regression analysis to examine the relationships between Time 1 and Time 2 depression scores, losses, and resources. The findings indicate that the Time 1 depression scores accounted for about half of the variance associated with Time 2 scores. High-loss scores were statistically significant in the equation ($p<.001$); receiving psychiatric care and SES variables were also significant ($p<.01$), but they accounted for much less of the explained variance. Presence of friends was not significant. The overall equation was significant ($p<.001$). The total variance explained was 52.11%.

As figure 3 illustrates, the Time 1 depression scores accounted for 44.57% of the explained variance, with 16.24% of this being shared with resources (12.98%) and losses (3.38%). Resources, including SES, accounted for 20.92% of the variance, 4.35% of which was unique. Losses contributed 9.83% to the variance explained; 2.86% was a unique contribution.

The findings presented in figure 3 have significant theoretical implications. The data indicate that much

FIGURE 3
The Explanation of Time 2 Depression Scores Provided by a Regression Model With Terms for Depression, Losses, and Resources[a]



[a]The entries represent percentages of total Time 2 Depression score variance explained by the terms of the model. No control for race is included. A total of 52.11% of the Time 2 Depression scores is explained by this model.
[b]An interaction term for Socioeconomic Status × Loss is included in the model.

depressive symptomatology is stable over time and that it is relatively independent of both coping resources and life-event losses. The availability of resources and the occurrence of loss-related life events are not discounted by the findings; they are statistically significant in the equation. However, the amount of unique variance accounted for by each is much smaller than that predicted by the Time 1 depression scores. Finally, the interrelatedness of depression scores, resources, and loss events empirically corroborate the theoretical model that guided the research.

SUMMARY AND CONCLUSIONS

This paper has presented data on life-event losses, coping resources, and depression scores, as well as data on a second issue relating the stability of depressive symptomatology over time to losses and resources. The major findings of the research can be summarized as follows.

1. Persons having high loss scores had higher depression scores than those with low/moderate loss scores.

2. The presence of a spouse was significantly correlated with lower depression scores for both the low/moderate- and high-loss groups. The presence of other relatives nearby was not significant.

3. The availability of friends was significantly correlated with lower depression scores for the high-loss group; no significant differences were found for the low-loss group.

4. Significantly more of the high-loss group were using the services of physicians and clinics than were those with low losses.

5. Those utilizing mental health services in both loss groups had higher depression scores than the nonutilizers.

6. Low socioeconomic status was significantly correlated with higher depression scores in both loss groups. It was also found that 64.8% of those in the low-SES group were in the high-loss group, compared with 31.1% of those in the high-SES group. The data suggest that low SES places persons in double jeopardy; they have fewer resources with which to cope and experience more losses.

7. A series of regression equations showed that losses and the absence of resources were significant variables when used to predict high depression scores.

8. The Time 1 depression scores accounted for more explained variance in Time 2 depression scores than did losses or resources.

9. There was considerable overlapping (interrelatedness) of variance explained by losses, resources, and Time 1 depression scores as predictors of Time 2 depression scores.

The data presented illustrate the complex nature of the interrelationships between life events, coping resources, and depressive symptomatology. The findings suggest that while life-event losses and the absence of personal and social resources are related to high depression scale scores, other factors are statistically (and probably theoretically) more important sources of explanation of depressive symptomatology. For example, the finding that Time 1 depression scores were the best predictor of Time 2 scores suggests that for some persons, at least, depressive symptomatology is a trait condition that may predispose them to life events which in turn exacerbate their preevent levels of psychiatric distress. The data also indicate that life-event losses are mitigated somewhat by the availability of personal, familial, interpersonal, and other resources; this finding has implications for early therapeutic intervention designed to assist those experiencing significant life-event losses.

Finally, the theoretical and analytical model used to guide the research appears to have utility for investigators seeking to understand other dimensions of the phenomenon of life events, coping resources, and illness.

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Life Events at the Onset of Bipolar Affective Illness

BY DAVID L. DUNNER, M.D., VIJAYALAKSHMY PATRICK, M.D., AND RONALD R. FIEVE, M.D.

The authors assessed the occurrence of stressful life events before the initial or subsequent episodes of affective illness in a group of 79 bipolar I manic-depressive patients who were attending a lithium clinic. About half of the patients recalled a life event in the 3-month interval before their initial episode; postpartum onset was prominent among these events. Few patients recalled life events for subsequent episodes. Finally, history data did not differentiate patients who recalled life events and those who did not. These data may be useful in assessing environmental antecedents of populations at risk for bipolar illness.

THE ROLE of stressful life events in the etiology of affective episodes has been speculated on and studied by many psychiatrists. Environmental events considered as possible precipitants of affective episodes have included interpersonal relationships (1); bereavement and social factors (2); arguments with spouse, marital separation, starting a new type of work, change in work conditions, and personal illness (3, 4); and problems in job performance and increased work responsibility (5). Some studies have noted that stressful environmental events are noted more frequently before initial affective episode than subsequent episodes (4, 5), and one study reported stressful life events before the initial episodes of two rapid-cycling patients (4). There has been no consistency among these studies regarding the temporal relationship between the life event and the onset of the affective episode. This time interval has ranged up to 3 years (2).

Hudgens and associates (6) found that 10 of 40 affectively ill patients in a controlled study had a precipitating life event within 6 months before the index episode. Similar associations for previous episodes of illness were rare. In addition, they found that the observed rate of life events before an affective attack in these 40 patients was not significantly greater than that in a control group of hospitalized patients. However, they noted more frequent changes of residence and a higher incidence of reported interpersonal discord in the group with affective disorders. These occurred during the year before admission but after the onset of affective symptoms. The authors concluded that when the illness seemed to begin soon after a stressful event, the temporal relationship might be purely random.

We attempted to study the relationship of life events to the onset of illness and to subsequent episodes in bipolar patients. We also analyzed family history data from these patients to ascertain if the subjects who reported life events associated with illness onset showed less familial incidence of affective illness than patients who did not report such events. We were also interested in assessing the frequency of postpartum onset and postinfluenza onset as specific possible precipitants.

METHOD

The sample consisted of 79 patients (41 men and 38 women) from the Lithium Clinic of the New York State Psychiatric Institute. All patients were evaluated by two psychiatrists and met the criteria for primary affective disorder (7). All had been hospitalized at least once for mania and were classified as bipolar I (8). Eleven of these bipolar I patients were "rapid cyclers," i.e., they had four or more affective episodes a year (9). All patients voluntarily consented to participate in this study.

All patients were requested to complete a questionnaire that included a list of life events. This list was prepared by assessing the records of over 200 former clinic patients and determining life stresses that were recorded as being of possible relevance to their affective episodes. Any events that occurred during the 3 months before the initial affective episode were noted as possible precipitating factors. The events included 1) accidents, operations, or physical illness, 2) occupational and financial changes, 3) illness, death, accidents, or separations in the family, 4) interpersonal conflict at home, school, or work, 5) marital changes, 6) pregnancy, postpartum state, or menopause, 7) change of residence or country, and 8) patients' spontaneous assessments of any other impor-

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tant or stressful occurrence preceding their illness. Life events relating to subsequent episodes were also noted. Concurrently, each patient was interviewed by a psychiatrist, and care was taken to exclude events that were reported during the illness and those that had occurred more than 3 months before the episode or onset. The age at onset of illness and the nature of the initial episode were also determined.

Family history data were obtained from clinic records, research records, and through direct interviews of probands' relatives. Most patients had participated in genetic studies that included structured interviews (10) of relatives, performed by individuals who were blind to proband diagnosis. Of 259 first-degree relatives (parents and siblings), 63 had been interviewed. Of the remainder, 49 were deceased. In addition, other relatives (e.g., children and spouses) of many patients had been interviewed and gave information about psychiatric illness in other first-degree relatives of the proband. Assessment of family history was performed independently of and blind to the subject's life event status.

TABLE 1
Demographic Data for Bipolar Patients With and Without Life Events

Group	N	Age		Age at Onset	
		Mean	SD	Mean	SD
Men					
With life event	21	43.5	10.7	29.1	8.8
Without life event	20	51.2	14.9	25.1	13.6
Women					
With life event	20	44.4	15.0	26.1	8.1
Without life event	18	44.9	13.6	24.7	8.5

TABLE 2
Life Events Reported at Onset of Illness by Nature of Onset

Group	Type of Onset	
	Depression	Mania
Men[a]		
Reported life events	5	16
Did not report life events	14	6
Women		
Reported life events	16	4
Did not report life events	9	9

[a]For the distribution for men $\chi^2=7.1$, $p<.01$ (Yates' correction applied).

RESULTS

About half of the patients described life events before the onset of their initial episode. There were no significant differences in the mean age of patients or the mean age at onset of illness for patients who described life events and those who did not (table 1).

Thirty-five of the 79 patients had initial manic episodes, and 44 patients had depressive onsets (see table 2). For patients whose first episode was manic, a greater percentage of men than women reported life events at onset of illness. The reverse was true when the initial episode was depressive. There was a significant difference in type of onset between men who reported life events and those who did not. However, similar results were not shown for women.

The frequency of life events at the onset of illness is presented in table 3. Several patients noted multiple events. Difficulties at work, marital problems, interpersonal conflict, and postpartum events ranked among the most frequently reported. Difficulties at work and interpersonal conflicts were reported more frequently by those who had mania as their first episode. Postpartum state and surgery as preceding life events were reported only by patients with depressive onsets. It is worth noting that 7 patients with manic onsets described increased responsibilities at work before the onset of their manic episode. Influenza as an event preceding the first episode was reported by only 1 patient in our study. Two patients related their first episodes to appendectomy and thyroidectomy.

There were 3 men and 8 women rapid cyclers. None of the men reported life events before their onset of illness, whereas 5 women had a preceding event. In 3 of these 5 patients the onset of illness occurred postpartum. However, a postpartum onset was not significantly more frequent among those who were rapid cyclers than those who were not.

TABLE 3
Frequency of Life Events at Onset of Bipolar Illness

Life Event	Type of Onset		Total
	Mania (N=20)[a]	Depression (N=21)[a]	
Difficulties at work	8	5	13
Marital problems	4	5	9
Interpersonal conflicts	8	3	11
Postpartum	0	8	8
Pregnancy	2	0	2
Illness in family	2	0	2
Accident	1	1	2
Operation	0	2	2
Influenza	1	0	1

[a]Some subjects reported more than one life event.

Subsequent Episodes

Twelve patients reported life events that occurred before subsequent affective episodes; seven of them had not reported events at the onset of illness. Two of these patients reported marital problems. Difficulties at work, interpersonal conflicts, postpartum episodes, illness in the family, and flu were reported by one patient each.

Family History Studies

Family history data were available for 68 patients. Family histories were recorded as positive for affective disorder if a parent or sibling 1) had been hospitalized or treated for mania or depression or 2) had depressive symptoms and had not been treated but met criteria for cyclothymic personality or depressive

personality (11). Of patients who reported life events at onset of illness, 72.7% had a positive family history for affective disorder compared with 68.7% of patients who did not report onset events. This difference was not statistically significant. Morbidity risks for bipolar and unipolar illness among these first-degree relatives were calculated and found to be similar for patients with life events at onset of illness and those without. Even when illness in relatives was considered present only if treatment had been received, the morbid risks in relatives for bipolar I, bipolar II, and unipolar illness were similar for the two groups.

DISCUSSION

The population we selected for this report has rather strictly defined bipolar manic-depressive illness and has been extensively studied from many points of view. Contrasts between bipolar I patients and other affectively ill patients on diverse measures such as symptomatology, clinical course, pharmacologic response, and biochemical characteristics have been reported (11, 12). Of interest is the high familial loading of affective illness, particularly bipolar illness, among relatives of bipolar I probands. The increased risk for affective illness in relatives has supported the notion of a genetic component in the etiology of bipolar I illness (13).

Thus in view of this presumably large genetic component in the etiology of bipolar I illness, our finding that about half of these patients had a life event at the onset of their illness which could be related to etiology suggests several possibilities. There may be a genetic-environmental interaction in the onset of symptomatology of bipolar illness, or the life events might be randomly related to onset or symptomatic of the illness. Some stresses, such as increased interpersonal conflict, could represent symptoms of illness (a view taken by Hudgens and associates [6]). Other stresses, such as influenza and surgery, occurred so infrequently that their relationship to the onset of bipolar illness was probably one of chance. However, childbirth is a specific event that can be accurately dated in relation to onset of affective symptoms. Our data confirm a study by Baker and associates (12) relating childbirth as an important factor in the onset of bipolar depression among women. It seems to be more difficult to demonstrate events of similar importance in men that relate to onset of illness.

If a form of bipolar illness had more of an environmental and less of a genetic component in its etiology, then family histories of affective illness should be less positive among "reactive" bipolar patients. We did not find this to be the case; positive family histories occurred equally often among patients who had life events and those who did not. Furthermore, when we limited affective illness in relatives to rare (and presumably inherited) forms—such as bipolar I and bipolar II illness—we did not find differences in the morbid risk for these disorders among relatives of bipolar probands who described life events and those who did not. Thompson and Hendrie (14) reported that "life-change" scores were similar for an index episode among two groups of patients with primary depressive illness (unipolar plus bipolar), one with positive and one with negative family history. Their findings support our data, although our population is more strictly defined.

Leff and associates (4) studied two rapid-cycling manic-depressives and noted life events before the initial episode but not for subsequent attacks. In contrast, we found that only about half of our rapid cyclers described life events at the onset of illness and that this percentage was similar to those who were not rapid cyclers.

In general, a higher percentage of patients reported life events before the initial episode than subsequent episodes. Our data are in agreement with the study of Okuma and Shimoyama (5) regarding this point.

The frequency of life events we found can be influenced by several factors. Although we interviewed patients to limit the time period of this study (three months before onset), it is possible that patients' recall of the relationship of the events to onset of illness may have been faulty. It should be noted that patients were usually interviewed several years after the onset of illness, although the mean ages and mean ages at onset of illness were similar for patients reporting events and those not reporting events. Furthermore, about half of another sample of patients interviewed within 2 years of onset of illness reported life events, suggesting that recall for such events might be stable over extended periods of time (D.L. Dunner, unpublished data).

Current research strategies for the study of affective illness relate to genetic-environmental interaction in the onset of symptomatology. Our data suggest that environmental stresses should be carefully evaluated in research on areas such as prospective studies of offspring of affective patients. Furthermore, since the postpartum state is clearly associated with onset of illness, we suggest that further research in postpartum depression might be helpful in determining biological factors associated with bipolar illness.

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Childhood Depression: Interpersonal Interactions and Depressive Phenomena

BY IRVING PHILIPS, M.D.

The author discusses the impact of parental disability, particularly parental depression, on children. Depression in children, which takes different forms according to the child's developmental level, may mirror or be a response to similar affect in a parent. Clinicians need to take note of the relationship between child and caretaker and of the various manifestations of childhood depression in order to avoid underdiagnosing this disorder.

CHILDHOOD DEPRESSION tends to be underdiagnosed in clinical practice. Its manifestations differ from those of depression in adults. The symptoms depend on age, psychosocial development, cognitive integration, and attachment behavior. If clinicians consider the child a miniature adult and expect symptoms similar to those seen in adult depression, many children will be misdiagnosed and their problems neglected.

Childhood depression does not refer to transitory moments of sadness or disappointment but rather to frank disorder that affects development and interferes with the fullest realization of innate potential. There may be a host of etiologic factors that have not yet been differentiated and clarified. Although the biochemical factors found in adults have not been confirmed in children, they may exist as either primary or secondary features. Nevertheless, the nature and intensity of interactional factors need delineation and description. This paper will highlight interpersonal factors between parent and child that may have significance in the expression of childhood depression in ways that are congruent with the psychic and cognitive developmental level of the child.

The effects of parental behavior on child development are obvious. Parental depression interferes with the ability of a parent to best meet the needs of the child, and the result may be subsequent psychopathology in the child, in the form of depression or its equivalents.

UNAVAILABILITY OF THE ATTACHMENT FIGURE

Bowlby (1), in a classic description of attachment behavior and the consequences of separation and loss on the developing child, widened our horizons regarding the influence of normal attachment and "anxious attachment" on child development. In normal development during the first year, children form close ties with their mothers. The availability of responsive and helpful persons to the child is basic to the development of self-reliance. When such a figure is available, the individual will learn that he can deal with alarming situations and that he may seek help without hesitation, feelings of anguish, or humiliation. When attachment figures are not available enough and do not respond

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enough in a warm and consistent manner, such learning may not take place, and the result is what Bowlby called anxious attachment. To quote Bowlby,

> Yet a further difficulty turns on the fact that a mother can be physically present but "emotionally" absent. What this means, of course, is that although present in body, mother may be unresponsive to her child's desire for mothering. Such unresponsiveness can be due to many conditions—depression, rejection, preoccupation with other matters—but, whatever its cause, so far as her child is concerned she is no better than half present. Then again, a mother can use threats to abandon a child as a means of disciplining him, a tactic that probably has an immeasurably greater pathogenic effect than is yet recognized. . . . Whether a child or an adult is in a state of security, anxiety, or distress, is determined in large part by the accessibility and responsiveness of his principal attachment figure. (1, p. 23)

A child confronted with such a situation goes through a typical sequence: protest, whereby he tries to recover his mother; despair, when he remains preoccupied with her and vigilant for her return; and, finally, detachment, when he becomes emotionally uninterested in her as a defense against painful feelings. The period of protest is related to his anxiety about separation, and the period of despair to grief and mourning.

Szurek (2) described mothers who were unresponsive or severely ambivalent toward their children. The earliest expressions of these children's sensual impulses were thwarted and repressed when met by the conflicted tension of the person upon whom the child depended for the satisfaction of his needs. A parent often will find it difficult to help his child experience basic needs if his own needs were frustrated during development. Thwarting of such impulses leads to frustration and inevitable rage. The child's repressed impulses undergo transformation and may express in a distorted and often caricaturized fashion the parental psychopathology that influenced the initial behavior.

Although the occurrence of depression in childhood has been debated, clinicians have recognized affective disturbance in the developing child of sufficient severity to indicate, if not frank depression, at least the precursors of adult psychopathology. Anna Freud noted,

> It was known in psychoanalysis long before such infant observations that depressive moods of the mother during the first two years after birth create in the child a tendency to depression (although this may not manifest itself until many years later). What happens is that such infants achieve their sense of unity and harmony with the depressed mother not by means of their developmental achievements but by producing the mother's mood in themselves. (3, p. 87)

Retrospective analysis has contributed to our understanding of parental loss through death or abandonment and its effects on subsequent development as well as on adult behavior. A more common occurrence is partial loss, when the caretaker, because of neuroticisms awakened by life events, cannot be emotionally available as much as he would like or as the infant needs. The parent may threaten desertion or abandonment or may shame the child sufficiently that he is afraid to seek expression of his wishes and impulses.

The varied manifestations of depression may depend on the developmental level of the child. It is obvious, as Malmquist noted, that "the response is contingent upon the child's developmental stage. The reactions to a depressed mother in an infant will vary from those in the child during the height of oedipal feelings as well as in the child during middle latency" (4, p. 956). Not only the psychic structure but also the cognitive level of the child and his ability to express painful affect by language or symbolic representations are significant. Anthony commented,

> If one accepts this close relationship between cognitive and affective development as advocated by Piaget, it would stand to reason that the experience of depression in late childhood will be radically different from the earlier experience. It is possible to go further and suggest that depression itself undergoes development throughout the child's development and that its characteristics are determined by parallel developments in symbolism, representation, language, and logical operations. The question whether depression does or does not occur during childhood, therefore, can no longer stand by itself. (5, p. 235)

CASE REPORTS

The following clinical experiences encountered in the treatment of children and families reveal incidents in which the child and a caretaker were experiencing similar affect, but which each expressed in different modes because of their different levels of psychic maturity.

Case 1. Consultation was requested at a well-baby clinic for a 17-year-old mother and her 9-month-old infant, who had failed to thrive. The mother, who was self-depreciatory and had frequent crying spells and suicidal ideas, indicated she had little time for herself or the infant. The infant was unresponsive and quite difficult to arouse. His face had a withdrawn and distant look, he was undernourished, and his growth rate was retarded.

Case 2. Bill, a 4-year-old, was abandoned by his father at birth. He was one of 3 children. His 32-year-old mother frequently spanked him (sometimes a bit too hard) to control difficult behavior. The mother's life pattern was punctuated by frequent depressive episodes; she had been hospitalized once, shortly after Bill's birth. Bill was a problem in nursery school, alternating between aggressive outbursts and hurting smaller and younger children and passively remaining by himself, refusing to enter into activities.

Case 3. George, an 8-year-old, was referred because of encopresis. When George entered the office the psychiatrist's pinched nose reflected the symptoms. George remarked, "Doctor, I have troubles. I stink inside and out." His

mother had been repeatedly hospitalized for psychotic depression since his birth, and his father was a chronic alcoholic. George was a highly intelligent youngster who was failing in school because of daydreaming and isolating behavior. His teacher remarked that she had never known a sadder youngster. When barely scolded he broke into tears.

Case 4. June, a 7-year-old, refused to go to school because of overwhelming fear. Her mother felt incapable of helping her attend. When June was 7 months old, the mother had had a severe depression that necessitated hospitalization for a 3-month period. Her father, with whom she felt unusually close, was an immature underachiever who had frequent job changes and dreams of great success that was soon to appear on the horizon.

Case 5. Jean, a 10-year-old, was devastated by the sudden death of her father when she was 9. After 2 days she rarely mentioned him again, but she wore his oversized parka wherever she went. When asked about him she responded with little emotion. Her mother, an ambitious, successful attorney, denied her grief and ambivalent feelings toward her departed husband. She lamented her new role in life. She was highly critical of her daughter, demanding high scholastic performance, a full social life, and after-school work. The daughter responded with compliant behavior but alternated between obesity and anorexia, elation and depression. Soon after the daughter entered treatment and began to rebel against her mother's unrealistic demands, the mother sought psychiatric help for herself because of progressively deteriorating behavior and severe depression.

These five examples of childhood disorder are merely paradigms of the expression of depression at various developmental levels. The symptoms of failure to thrive, passive aggression, overt sadness, school avoidance, and psychosomatic problems served to express pervasive depressive affect and clinical depression in children at different developmental levels.

DEVELOPMENTAL LEVEL AND DEPRESSIVE SYMPTOMS

At the Langley Porter Institute, University of California at San Francisco, work with each parent is part of the diagnostic evaluation of all children. The understanding of the family gained through an evaluation of both the parents and the child with the presenting complaints gives a fuller perspective on the disorder of the child. A State of California regulation required that if a parent was seen in treatment along with the child a chart be opened and a diagnosis established, and a review of the institute's records from September 1969 to December 1975 revealed 38 cases of depression in the parent and 10 cases of depression in the children, plus 1 additional report made in pediatric consultation. The relationship of these parents and the subsequent development of their children was recorded. Five of these cases were described in the previous section.

In all of these cases, the mother was the depressed parent. Eight boys and three girls were included in this series. Children with adolescent depression were not included. The predominant response to caretaker depression was passive or active aggression. The response to caretaker disability was aggression directed outward or inner passivity and withdrawal. The underlying themes seem related to efforts at compensation and expression of bereavement by whatever means was available to the child. In these cases the experiences were continuous, the bereavement unresolved, and the result was frank disorder—in these examples, depression. Although this sample is small and certainly not completely representative, it indicates that more children might be identified by clinicians if they were more aware of parental distress and its influence on the child.

In this series the clinical depression of the mother was the significant variable. In each case the history of the mother influenced her care of the child. It was evident that what she had not received in her childhood she could not give to her child, and the attachment behavior of the child was distorted.

It is likely that disorders other than parental depression may result in anxious attachment, with subsequent manifestations of depression in childhood. An increasingly frequent situation is the discharge of a mother who is heavily dosed with psychoactive drugs from a psychiatric hospital after an acute illness. Obviously this interferes with the ability of the parent to provide for optimal development of young children and interferes with their attachment behavior. The parent is not only separated from the child through hospitalization but on return finds it difficult to care for him. A variety of symptoms may result, depending on the nature of attachment between parent and child, the developmental level of the child, and intercurrent life events. A survey of phases of the growth cycle indicates various manifestations of response to interpersonal experience.

Infancy

The classic studies of Spitz (6) called attention to the institutionalized child who receives little attention from personnel, with resultant anaclitic depression. In these situations the maternal figure is not present and there are no other opportunities for attachment. The infant fails to thrive, withdraws, becomes apathetic, and in some cases dies. It became clear from a number of studies that similar affects occur in infants who remain in their own homes and fail to thrive because of maternal depression. Mothers who experience severe depression, disinterest, or apathy react to their infants in a distorted fashion. The descriptions of Fischhoff (7) and those of Fraiberg and associates (8) in the classic "Ghosts in the Nursery" emphasize this point. The infants in these studies were similar to those described by Spitz. Both caretaker and infant were depressed, each expressing the disorder through symptoms consistent with his own developmental level. Fischhoff (7) brought to our attention that deprivation may not be the only factor in failure of development and that there exists a multitude of possible factors. He noted,

> Recently Cravioto (1973) reported an ongoing study, now in its seventh year, of 300 children in a Mexican village, where malnutrition of some degree is prevalent among all the children because of "low socioeconomic and cultural backgrounds," but severe malnutrition is a "seemingly random occurrence." The investigators concluded that the development of severe "third degree" malnutrition is strongly associated with the lack of social, emotional and cognitive stimulation within the home. (7, p. 223)

The separation and resultant despair that reflect caretaker disability may be the infantile expression of depression. Szurek (2) reported that "only one child among 264 psychotic children . . . seen at Langley Porter between 1946 and 1961 was in any appreciable degree malnourished and definitely physically underdeveloped. This patient at age three years presented a picture of a severe agitated depression, restless, wringing her hands with an extremely tense facial expression and speaking little and often not at all, and showed little sign of attachment to any staff member" (p. 255). The history of the family was not presented.

A similar patient was admitted to the Langley Porter Institute children's ward. The child at age 3 was severely undernourished, refused to eat, and was cachectic in appearance. She wrung her hands so severely that she had caused tissue damage to her wrists. She refused to speak but had good eye contact. Her posture and gait were stiff and awkward. Repeated neurological examinations were within normal limits. After 3 years of residential treatment her condition had changed very little. She was below the first percentile in height and weight. Her early years had been chaotic, with her mother so constantly frightened and under threat from a paranoid husband that she had little time for her daughter. Early normal development receded into the more pathological picture described above.

Are these two examples of what may be called psychotic depression expressions of the vagaries of the depressive phenomena in infancy? It is intriguing to speculate as to whether the most severe disorders of childhood, such as the psychotic maldevelopments, may not be prototypes of severe depressive episodes occurring early in the child's development.

Preschool (2 Years-6 Years)

In the preschool period the child's language increases considerably. With cognitive development he gains the capacity for observational learning, with increased emphasis on mental representations through imitation, but he is still quite egocentric and tends toward direct action. The structuralization of his mental process becomes more formal, but the ability to think conceptually is not achieved until he moves closer to 6 or 7 years of age. He is only partially able to conceptualize ideas such as death. It is in this period that the child is vulnerable in his quest for autonomy and initiative and may identify with a depressed mother. The manifestations of depression may be varied. The reactions of children to parental disability are frequently regressive reactions—severe separation anxieties, hyperactivity, learning disorders, somatizations, and others. Depression in childhood may be best diagnosed by the analysis of the caretaker-child dyad and their reactions to each other.

School Age (6 Years-11 Years)

The beginning of operational thinking is a major turning point in the child's cognitive development. The child now learns how to organize the immediate situation in terms of a more general reality. In the structural sense, this is the time of formation of the panoply of defenses and consolidation of the superego. The loss of a parent is now understood in conceptual form, although understanding of death in the abstract sense must wait a few more years. The symptoms of depression take more nearly the form of adult depressive reaction and mirror the depression of the caretaker. The child is overly sensitive and easily hurt. Self-depreciatory feelings begin to emerge. He seeks relationships but fears them; the threat of abandonment remains in the air. Consequently, his relationships are tenuous or tentative. He vacillates between harsh self-criticism and blaming others for their faults. Observation reveals a sense of sadness and unhappiness. He may chronically complain about physical or psychological hurts. In school he may daydream or be the class clown, and for his efforts to find acceptance and reward he is scolded and sometimes punished. Seldom is he recognized as needing consolation; rather, his acting-out behavior is annoying and results in further reinforcement of his negative feelings. His suffering is unrecognized by teacher or parent, and he may go unnoticed, never receiving the attention he craves and so desperately needs. Malmquist (4), who listed symptoms of depression in middle childhood, described this type of child very well. Although the child's symptoms are more focused, they mirror those of his parent.

Finally, separation through divorce, an ever-increasing phenomenon in contemporary society, must be mentioned. The work of Wallerstein and Kelly (9) indicated the acute effects on children of such an event. Their descriptions are of depressed children, although they are not diagnosed as such. Their findings are consistent with those of clinicans who work with such families. The affect of such children is obvious and mirrors further the affects of each of the separated parents, now attempting a new life apart. In divorce the family continues, although the marriage has died.

Table 1 summarizes the disorder of depression as it appears in the developing child.

CONCLUSIONS

Childhood depression is underdiagnosed in clinical practice. There may be many factors, both biological and psychological, that contribute to its development. The role of biochemical factors has not been as clearly delineated in children as in adults. This paper has emphasized some of the psychological interactions that

TABLE 1
Developmental Chart of Childhood Depression

Age	Psychosocial	Cognitive	Mode	Symptoms	Caretaker Problems and Life Events[a]
Infancy	Trust vs. mistrust Attachment vs. "anxious" attachment	Sensorimotor phase	Autonomic nervous system Distorted ego differentiation	Anaclitic depression Failure to thrive Psychophysiologic manifestations	Parental depression Rejection Abuse-neglect (physical and psychological)
Preschool (2–6)	Autonomy vs shame and doubt	Preoperational thought	Premature superego development Autonomic nervous system	Depressive equivalents Passive aggression Psychophysiologic manifestations Separation anxiety, etc.	Incapacity and inability Separation Death Hospitalization Divorce Chronic illness
Early school age	Initiative vs. guilt	Stage of concrete operations	Organization of defenses	School refusal Psychophysiologic manifestations Variety of affective symptoms Aggression Learning problems, hyperactivity, etc.	
Late school age	Industry vs. inferiority	Stage of concrete operations	Superego consolidation	Depression similar to adult picture	

[a]Throughout all ages.

may be involved in the etiology of childhood depression. It has emphasized that parental disability, whatever its origin but especially if it is related to depression, interferes with the parent's capacity to provide the relationship needed by the child. The child, in turn, is inhibited from achieving his innate potential. A close study by the clinician of the dyad of child (with attention to his developmental level) and caretaker will increase the accuracy of diagnosis and make possible a greater understanding of the manifestations of depression in childhood.

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The Psychopathology of Suicide: A Clinical Model Approach

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The author hypothesizes that certan types of persons at high risk for suicide constitute definable clinical models, that the process of arriving at a suicidal outcome involves elements unique to each model, and that these elements can be considered manifestations of psychopathology. This approach is investigated using two models: "Males Under Forty" and "Stable with Forced Change." The results indicate statistically reliable differences between the models in the distributions of estimated risks for persons who subsequently committed suicide compared with those who did not. The implications of the associated high-risk variables as reflections of psychopathologic processes are considered.

THE MANY factors involved in suicidal behavior call for an approach that can account for this behavior in a systematic way while considering a bewildering array of biological, social, and psychological influences. Because of this complexity, efforts to characterize the "suicide-prone person" have not produced a generally accepted formulation, although some persuasive concepts have been developed.

A large part of our dilemma involves the uniqueness of each individual; a full understanding of the suicidal impulse in a given person can only be reached by a thorough knowledge of that person. This uniqueness prevents us from readily translating our understanding into a valid generalizable principle. Further, the recognized universal characteristics of the suicidal state tend to be highly subjective processes that are often difficult to explore. The number of exceptions to specific universal elements in completed suicides is also troubling.

We are exploring a middle ground, with the hypothesis that certain kinds of people constitute clinical models in that they tend to respond to a given constellation of stresses in a similar way with regard to suicidal behavior. By defining the model and identifying the constellation of elements associated with completed suicide for that model, we may be able to reduce the disadvantages of focusing on either individual uniqueness or universal inclusiveness. Without challenging the validity of traditional psychopathologic constructs, we can acknowledge that clinical experience tells us that certain kinds of people seem to reach the suicidal act by a similar route.

If the use of models is a valid approach, the more specific the model (i.e., the more homogenous the persons involved), the more effective it should be as a predictive tool. In this report two models will be discussed, a broad one, "Males Under Forty," and a specific one, "Stable with Forced Change." The number of possible models is limited only by how subtle one wishes to be and how many subjects can be found for study and subsequent validation.

We have proceeded on the premise that identification of the person at high risk for suicide is not enough. Rather, those within the high-risk group who are most likely to complete the act must be recognized. This permits a more efficient use of treatment time and facilities when intensive efforts are needed and avoids the false sense of security that may be generated by simply screening "suicidal" from "nonsuicidal" populations.

METHOD

Subjects

Detailed psychosocial data were gathered by extensive evaluative interviews of 3,006 persons admitted to a psychiatric inpatient unit because of a depressive or suicidal state; such a population is known to be at high risk for subsequent suicide. We then determined at yearly intervals which individuals had committed suicide; the expected rate was 1%–2% per year. The dependent variable considered in this study was occurrence of suicide within 2 years of the initial evaluation. From this pool of subjects, those meeting the criteria for two models were identified and followed for 2 years.

Subjects were required to meet three criteria for inclusion under the Stable with Forced Change model: 1) rating of moderate or high on overall stability (this rating considers stability of emotional health, work, school, marriage, relationships with others, and geo-

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TABLE 1
Distribution of Suicides in Index and Validation Sets of Two Clinical Models

Model	Number of Subjects	Outcome: Suicide N	Suicide %	Nonsuicide N	Nonsuicide %
Males Under Forty	858	41	4.8	817	95.2
Index set	566	27	4.8	539	95.2
Validation set	292	14	4.8	278	95.2
Stable with Forced Change	782	35	4.5	747	95.5
Index set	469	20	4.3	449	95.7
Validation set	313	15	4.8	298	95.2

graphic location), 2) absence of psychosis, and 3) a significant loss or threat of loss associated with the initial high-risk situation.

Of the 858 subjects who met the single criterion for inclusion in the Males Under Forty model, 41 (4.8%) committed suicide during the follow-up period. Of the 782 subjects who met the three criteria for the Stable with Forced Change model, 35 (4.5%) committed suicide during the follow-up period.

Index and Validation Sets

Subjects satisfying the criteria for each model were divided randomly into an index set and a validation set, with the constraint that the validation set include at least 14 suicides (a figure determined by statistical methods to be necessary for validation purposes). The final breakdown is shown in table 1.

Variable Screening

For the two index sets, 183 psychological and social variables elicited in the initial interview were examined for their individual association with the outcome variable, suicide within 2 years. The procedures used have been previously outlined in detail by Heilbron and Bostrom (1). This screening process identified 64 variables in the Males Under Forty model and 46 variables in the Stable with Forced Change model that showed a significant association ($p<.05$) with suicidal outcome.

Model Development

The variables selected in the screening process were entered into a stepwise linear discriminant analysis. The resulting discriminant functions contained 13 variables for the Males Under Forty model and 11 variables for the Stable with Forced Change model.

The variables selected by the discriminant analysis were then analyzed in a series of linear logistic regressions, in which the estimated values of p can be interpreted as estimates of risk of suicide within 2 years. After fitting a logistic model, the variable with the weakest coefficient was eliminated and the model fitted again until all remaining coefficients were significant at the .05 level. For the Males Under Forty model 1 variable was eliminated in this way, leaving a total of 12, while 2 variables were eliminated from the Stable with Forced Change model, leaving a total of 9.

TABLE 2
Predictor Variables for Males Under Forty Model

Variable	High-Risk Factors	Number of Subjects	Suicides N	Suicides %	p
Marital status	Divorced or separated[a]	64	7	10.9	<.02
Residence	Hotel, small[a] or medium[a]	79	8	10.1	<.02
Occupation	Higher executive[a] or semi-skilled	129	12	9.3	<.01
Help sought after attempt	Actively sought help	74	7	9.5	<.05
Seriousness of injury from attempt	Required intensive care	72	9	12.3	<.01
Living arrangement	With nonfamily mixed group/ opposite sex	88	9	10.2	<.02
Stress, other	Severe	174	13	7.5	<.04
From broken home	Death of father or divorce with mother departed	68	9	13.2	<.005
Emotional disorder in family	Alcoholism	121	11	9.1	<.02
Attitude toward interviewer	Mixed or negative[a]	209	16	7.7	<.02
Degree of psychosis	Moderate or severe[a]	124	11	8.9	<.02
Degree of religious activity	Low or moderate[a]	105	10	9.5	<.02

[a]Number of observations too few to be reliable unless pooled with other indicated categories.

RESULTS

Variables Associated with High Risk and Suicide

The characteristics most clearly associated with high-risk subjects in the index set of the broad Males Under Forty model who committed suicide within 2 years are indicated in table 2. The characteristics associated with suicide in the high-risk subjects in the index set of the more specific Stable with Forced Change model are indicated in table 3.

Three variables are common to both models: marital status, occupation, and seriousness of injury during the present suicide attempt. With the exception of one category of marital status (separated), which contained only 2 persons in one model and 1 in the other, the high- and low-risk category groupings are identical in the two models for these 3 common variables. The weighting of marital status is approximately the same for both models, while the Stable with Forced Change model gives a slightly greater weight to the high-risk occupational categories and the Males Under Forty model gives greater weight to the high-risk category of seriousness of injury from present attempt.

TABLE 3
Predictor Variables for the Stable with Forced Change Model

Variables	High-Risk Factors	Number of Subjects	Suicides N	Suicides %	p
Marital status	Divorced	78	7	9.0	<.04
Religious affiliation	Protestant, Jewish,[a] Fundamentalist,[a] other,[a] or no affiliation	312	18	5.8	<.02
Occupation	Higher executive[a] or semi-skilled[a]	76	8	10.5	<.01
Last change in occupation	Within 1 month[a] or no change	262	17	6.5	<.01
Seriousness of injury from attempt	Required intensive care	88	8	9.1	<.02
Severe loss other than significant person	Any but academic loss	209	17	8.1	<.001
Physical health in past year	Severe disability[a] or progressively worse	165	13	7.9	<.01
Crying during present illness	None or unable to cry	159	12	8.1	<.01
Personality pattern	Obsessive or schizoid	126	12	9.5	<.002

[a]Number of observations too few to be reliable unless pooled with other category or categories.

TABLE 4
Median Estimated Risk of Suicide Assigned to Subjects Who Did or Did Not Commit Suicide

Model	Risk: Nonsuicide	Risk: Suicide	p	Separation Index[a]
Males Under Forty				
Index set	.0078	.2448	<.0001	.926
Validation set	.0082	.0221	<.04	.661
Stable with Forced Change				
Index set	.0056	.2752	<.0001	.932
Validation set	.0056	.0288	<.01	.705

[a]The separation index, which reflects the separation of risk distributions for subjects who committed suicide and those who did not, is the proportion of suicide/nonsuicide pairs in which the suicide had a higher estimated risk.

Performance of the Models

The estimated risk of suicide can be compared with the actual suicidal outcome by showing that persons who subsequently committed suicide were assigned higher estimated risks than those who did not. The median estimated risks and the Mann-Whitney chi-square statistics for testing differences in these medians are shown in table 4. In addition, the proportion of suicide/nonsuicide pairs in which the suicide had the higher estimated risk is a related measure of the separation of the two distributions of risks; this proportion is indicated in the last column of table 4. This figure would be .50 if estimated risks did not, in general, differ between suicides and nonsuicides. Perfect separation would yield a value of 1.00. As postulated above, the more precise model (Stable with Forced Change) provides a clearer separation of the suicides and nonsuicides than the less precise one. Especially gratifying is the small proportion of nonsuicides (6%) who were classified as high risk under this model.

TABLE 5
Identification of Actual Suicides and Nonsuicides Using Optimal Criterion for High Risk

Outcome	Index Set: High Risk N	Index Set: High Risk %	Index Set: Not High Risk N	Index Set: Not High Risk %	Validation Set: High Risk N	Validation Set: High Risk %	Validation Set: Not High Risk N	Validation Set: Not High Risk %
Males Under Forty								
Suicide	25	93	2	7	9	64	5	36
Nonsuicide	99	18	440	82	94	34	184	66
Stable With Forced Change								
Suicide	15	75	5	25	6	40	9	60
Nonsuicide	36	8	413	92	18	6	280	94

All indices show a decrement between index and validation sets, as expected. However, the data for both models on the validation sets indicate real, statistically reliable differences in the distributions of estimated risks for those who subsequently committed suicide compared with those who did not.

Figure 1 shows the cumulative distributions of estimated risks for suicides and nonsuicides in the index and validation sets for each model. These are plotted to show how setting an arbitrary criterion for classifying a person as high risk affects the proportion of actual suicides and nonsuicides so classified. As this criterion is lowered (moved left on the scale), a progressively larger proportion of the actual suicides is identified, at the expense of increasing the proportion of nonsuicides also classified as high risk.

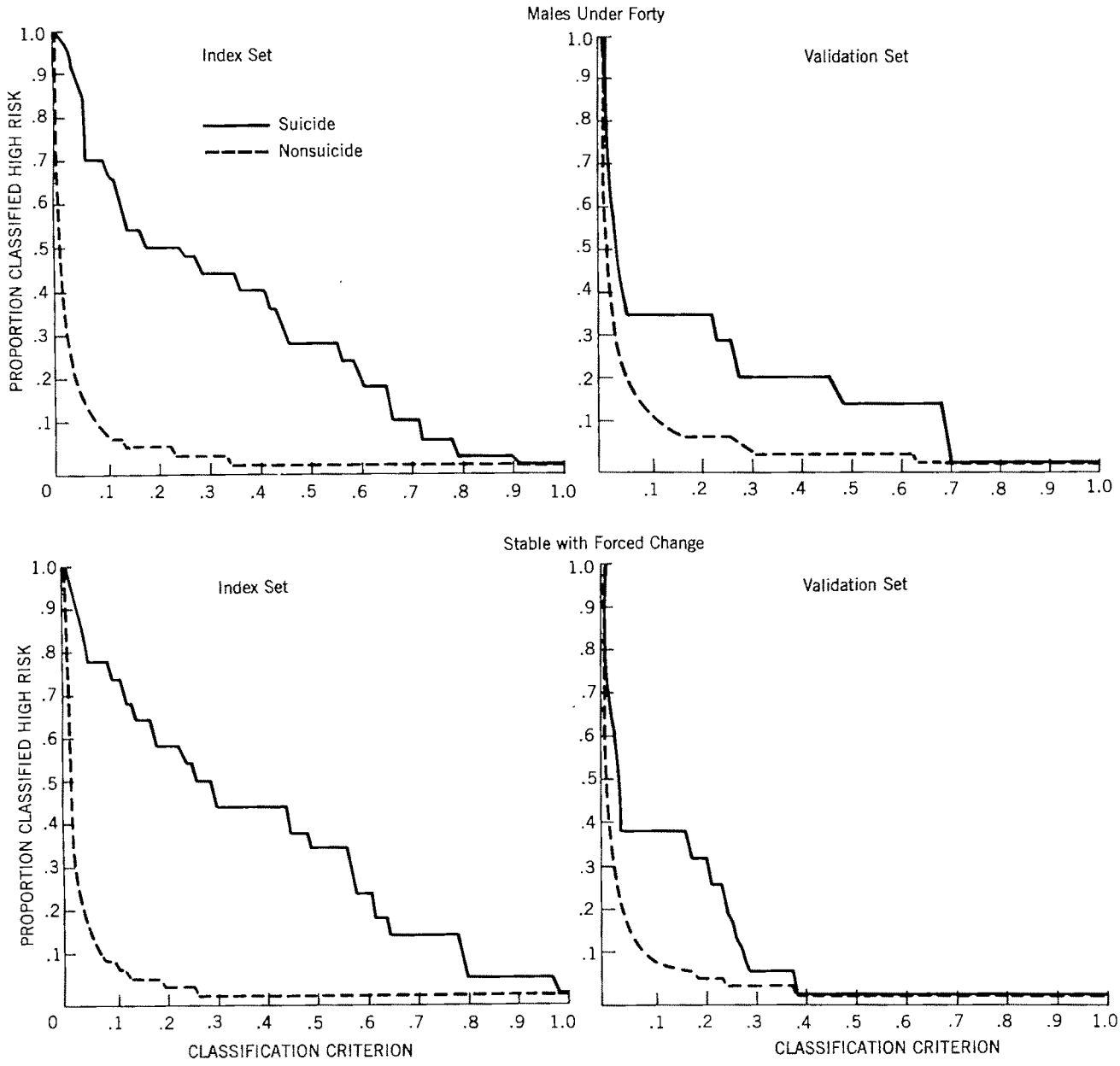
The point at which the two curves are most separated indicates the criterion value (or cutoff point for high risk) that yields the fewest total false positives and false negatives. Using this optimum criterion provides identification of actual suicides and nonsuicides compared with their estimate of risk, as indicated in table 5.

DISCUSSION

To use clinical models in studying the psychopathology of suicide is to acknowledge that, whatever the process leading to the suicidal act, high-risk persons who demonstrate a specific clinical pattern (model) appear to share observable indicators of the likelihood of suicide. These indicators can be construed as reflecting crucial intrapsychic processes that may respond to clinical intervention and—what is most important here—that will differ with different models.

This relationship of quantifiable data to underlying psychopathology can be seen as a function of the preciseness of the model. An age/sex-specific group such

FIGURE 1
Cumulative Distribution of Estimated Risk for Index and Validation Sets of Males Under Forty and Stable with Forced Change Models



as Males Under Forty allows for so varied a representation that the relationship can hardly be very close. As the model becomes more detailed, however, we can anticipate a progressively more direct translation of the correlates of high suicide risk into implications for psychopathology.

In the present instance, we can postulate that persons with relatively stable life patterns tend to exert a certain control over events in their life as a way of maintaining that stability. The capacity for control becomes a key dynamic in their adjustment and may be manifest clinically as obsessive personality traits. However, although crying may be controlled, events such as progressive worsening of physical health may not. A change in occupation can be necessitated by economic conditions, a spouse may leave, or an intercurrent stress be sustained. Without the support of spouse, colleagues, or strong religious affiliation, a suicidal state with a fatal outcome is highly probable. Although other persons who fit the Stable with Forced Change model may go through suicidal periods under stress, if they do not fall into the high-risk categories indicated their level of risk will be significantly lower. On the other hand, the presence of these high-risk factors may have relatively little significance in persons who do not fit the Stable with Forced Change model.

Applying this concept to the less precise Males Under Forty model necessarily involves a less direct translation of the correlates of high risk into intrapsychic process, as mentioned above. In this instance we can postulate that the presence of alcoholism in the family would be conducive to generating anxiety and instability of object relations, which are compounded if the family breaks up. This breakup would be especially handicapping emotionally for the young male if it occurred through the death of an alcoholic father because in this case the still unresolved process of identification and dependent-independent conflict with ambivalence toward the parent of the same sex is involved.

Continued instability is manifest in adult life in the form of marital conflict, transient forms of residence and living relationships, and significant thought disorder. When intercurrent stresses occur in the absence of a cohesive family or other support system, suicide may seem to be the only alternative. The still-unresolved dependent-independent struggle is seen in the person's behavior following serious attempts.

Such a formulation, based on the high-risk variables listed in tables 2 and 3, may seem to lean heavily on "psychologizing," but experienced clinicians and suicide prevention workers will recognize familiar markers at each step. Individual differences must still be considered in therapeutic efforts, of course, but those efforts can be more efficient if they are concentrated on persons in the 5%–6% per year fatality range than on those in the 1%–2% group. To date, the only demonstrated and validated means of objectively identifying such a high-risk population is by starting with models such as those discussed here. Earlier efforts to accomplish this task (2–5) contributed greatly to the development of the model concept, but they were limited by small sample size, retrospective design, or lack of validation.

An encouraging finding in the present study is the relatively small overlap of the high-risk variables applicable to the two models examined. This supports the hypothesis that the subgroups do represent different clinical entities that can be studied with a view toward developing treatment approaches most effective for each. Comparing the high- and low-risk categories for each variable in each model with the corresponding categories of the subjects in the other model, 11 are found to be in the same direction of risk and 10 in the opposite direction, giving further support to the independence of the two models.

In view of the poor results of rigorous validation efforts in the past (6), the performance of the predictor variables in the validation sets is most encouraging. This is especially so in view of the reduction of the variables to a number (12 for Males Under Forty, 9 for Stable with Forced Change) that can be applied easily in most clinical settings.

One source of ambiguity is the inevitable need for "other" categories in gathering data. In this study it was applied to religious affiliation to indicate membership in religious groups that are minorities in the United States, such as Mormon, Moslem, or Greek Orthodox churches. In reference to "loss" or "stress" the "other" category included an array of situations that defy classification, such as relinquishing a cherished goal, learning one is infertile, sale of a family home, an amputation, facing military draft, or loss of a feeling of independence or respectability. These stresses are pertinent to suicidal individuals but are difficult to apply generally to a high-risk population.

It remains to develop a variety of additional models in order to include the bulk of very high-risk persons in this approach. Although study of the many theoretic and clinical aspects of self-destructive behavior will never be an exact science, we are encouraged to believe that it is still possible to supplement the clinical and intuitive skills of health workers to minimize the number of unnecessary, premature, and tragic deaths from this cause.

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Depression Simulating Organic Brain Disease

BY JESSE O. CAVENAR, JR., M.D., ALLAN A. MALTBIE, M.D., AND LINDA AUSTIN, M.D.

The authors report four cases of depression manifesting as organic brain syndrome in adult nongeriatric patients. The correct diagnoses in three cases were made by a psychiatrist's or resident's empathic response to the patient, and in the fourth by the patient's history of depression. The authors state that only by maintaining a high degree of suspicion and trusting one's empathic response to the patient will unusual presentation of depression be recognized.

VERWOERDT (1) NOTED that depression in senescence is commonly overlooked because it does not always follow the same pattern or have the same symptoms as in the younger age group. The affective manifestations of depression, such as sadness, crying spells, guilt, and self-depreciation, may be absent. Impairment of memory, somatic symptoms, psychomotor retardation, and loss of initiative may be the more prominent symptoms and may be confused with an organic brain syndrome.

Although most gerontologists agree that depressive disorders in older patients may have an atypical clinical picture, this phenomenon has not been reported in younger patients. In fact, most authors suggest that it is not seen clinically. Cohen (2), in describing the depressive type of manic-depressive illness, stated, "There is no disorientation" (p. 683). Ford (3) noted that in involutional depressive reactions "the sensorium is clear" (p. 701).

The purpose of this communication is to report our experience with an unusual manifestation of depressive disorders in adult nongeriatric patients. Our work suggests that in certain patients a severe depressive disorder may have the clinical features of an organic brain syndrome and lack the usual depressive signs or symptoms. Our experience is illustrated by the following cases.

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The opinions expressed in this article are those of the authors and not those of the United States Veterans Administration.

CASE REPORTS

Case 1. Mr. A was a 50-year-old blue collar worker who was brought for emergency psychiatric evaluation by his wife. Over three months he had displayed increasingly bizarre behavior to the extent that he was rummaging through neighbors' garbage cans, refusing to go to work, and grossly confused as to time and place. His family noted that his disorientation seemed more pronounced at night. To the family's knowledge, his past physical health had been good and he had not been consuming alcohol.

Mr. A had been taken to his family physician several days before the emergency evaluation. This physician suspected that the patient had a brain neoplasm, but a thorough physical workup, including skull radiographs, neurological examination, and brain scan, were negative or normal. A mental health professional had evaluated the patient and had told the family that Mr. A had a severe organic brain syndrome and recommended institutional care.

The mental status examination revealed a dirty, unkempt man who appeared older than his stated age. He was grossly confused to time and place. His recent memory seemed impaired; he could not name the President or his own date of birth. His affect seemed to be one of bewilderment but was not depressed. The family reported that he had not seemed sad or depressed, slept well during the day, had had no crying spells, and had a good appetite.

Despite this seemingly clear organic picture and an absence of any signs of depression, the examining psychiatrist (J.C.), who is a psychoanalyst, believed the patient was depressed. Countertransference caused the psychiatrist to feel sad and many depressing thoughts came to his mind.

Amitriptyline was prescribed in therapeutic doses, and an appointment was scheduled for the following week. Mr. A came for that appointment neatly dressed and groomed; he reported that he had begun to sleep at night after taking the medication and that he was now "thinking clearly." He was oriented to time and place. His recent and remote memory were intact; his intelligence was estimated to be above average. His mood was normal. He showed no thought disorder and denied hallucinations or delusions. Mr. A's family confirmed that he was improved. He was seen on three subsequent visits and continued to appear completely recovered. Despite the fact that he was cautioned that he needed to continue the medication for six months, he neglected to return because he felt so well. He was subsequently lost to follow-up.

Fifteen months later Mr. A, accompanied by his family, appeared as a psychiatric emergency. He had again experienced increasing confusion over a period of several months and was grossly disoriented. He was dirty, unkempt, and to-

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tally disoriented to time and place but had no observable depressive signs. His family denied that any depressive symptoms had been present.

Mr. A was again treated with amitriptyline and seen one week later in follow-up. At that time he was well dressed, well groomed, and totally oriented to time and place. His cognitive function appeared to be intact, and his mood was normal. He remained well over the following seven months until he was again lost to follow-up.

This man appeared to have a classic organic brain syndrome characterized by confusion, disorientation to time and place, and memory disturbances. It was only through an empathic response in the examiner that depression was suspected; there were no signs or symptoms of depressive illness. No precipitating event was ever identified that might have led to the onset of the depression.

Case 2. Ms. B was a 22-year-old housewife who was seen as an emergency case because of confusion and disorientation. She had a six-month-old baby and had had an uneventful pregnancy and delivery. Two months before admission she had complained of "not thinking clearly." This had gradually worsened to the point where she was grossly confused and disoriented.

Her husband denied that she had experienced any anorexia, weight loss, crying spells, or sadness. She had been "restless" at night but had not had any significant sleep disturbance. Most disturbing was her disorientation in the evening and nighttime hours.

The mental status examination revealed that Ms. B was confused and disoriented. She did not appear to be depressed and specifically denied any sleep disturbance, weight loss, sadness, crying spells, or suicidal ideation. No delusions were noted and she did not appear to be hallucinating.

Ms. B had been seen by her family physician before psychiatric referral. A physical examination, neurological examination, brain scan, electroencephalogram, and skull radiographs were all negative or normal. She had not been febrile.

Ms. B seemed to be depressed, notwithstanding the fact that she denied any depressive symptoms and did not look depressed. The impression of depression was suggested by the fact that the psychiatrist found himself feeling dejected and blue after several minutes of talking with her. He prescribed therapeutic levels of amitriptyline.

When evaluated the following week, Ms. B appeared much better. She was oriented to time, place, and person and did not appear to be psychotic. She had some difficulty in recalling recent events and, particularly, names, but this was much less pronounced than it had been. Her medication dosage was increased and she was given an appointment for the following week.

At that appointment Ms. B's husband reported that she was "back to herself" in every way. She seemed totally oriented and demonstrated no memory deficits. She was outgoing, pleasant, and cheerful and showed no signs of psychopathology.

Ms. B's amitriptyline dosage was maintained and she was seen monthly for brief office visits over four months. Her family moved to another city at that time and contact with her was lost. However, during that time no other confusional episodes were noted.

Again, the examiner's countertransference and empathic response was significant in suggesting the proper diagnosis for this patient.

Case 3. Mr. C was a 51-year-old man who was hospitalized in September 1976 after a myocardial infarction. He made an uneventful recovery from the infarction but appeared depressed to the cardiologist. The cardiologist did not request psychiatric assistance, however, because the depression did not seem severe.

Mr. C requested psychiatric evaluation in February 1977 because of decreased energy and difficulty falling asleep at night. He denied depressive symptoms and had an excellent appetite.

Mr. C was a small, neatly dressed, pleasant, and cooperative man, who appeared 10 years older than his stated age. His speech was slow and halting, he performed mathematical calculations extremely poorly, and he quickly lost track of the task. There was no evidence of thought disorder, and he was not psychotic. His affect was labile and alternated between smiling appropriately and crying.

The patient was admitted to the psychiatry service for evaluation. Psychological testing for organic brain syndrome failed to show any organic central nervous system damage. An electroencephalogram was normal, as was the neurological examination. Mr. C appeared confused on the ward.

Antidepressant medication in therapeutic levels was begun because it was believed that Mr. C had a depressive disorder appearing as an organic brain syndrome. As Mr. C improved, he began to talk of his 22-year-old daughter who had died suddenly of anaphylactic shock in April 1976; his ambivalent feelings about her were the source of his depression. This information was not available, however, until Mr. C had improved to the point that he could communicate it to his physician. Most interesting was the fact that as the depression improved, the confusion cleared in direct proportion. The best clinical indicator of the extent of depression was Mr. C's ability to do serial subtractions of 7 from 100. At the end of a 6-week hospitalization, Mr. C could do mental arithmetic quickly and accurately. His lability of affect, confusion, staggering gait, and poor articulation all cleared as the depression improved.

Case 4. Mr. D was a 48-year-old disabled man who had been transferred from a chronic care psychiatric hospital where he had been hospitalized for over a year with the diagnosis of organic brain syndrome. Progressive confusion, memory loss, loss of energy, and his family's inability to care for him had led to the hospitalization. A thorough evaluation had been done, and there were no abnormal findings on the skull radiographs, brain scan, or electroencephalogram.

When evaluated upon transfer, Mr. D appeared to have a chronic organic brain syndrome. There was nothing on the mental status examination to suggest any depressive or psychotic disorder. An air of pessimism among the ward staff and psychiatrists was obvious; it appeared that nothing could be done.

Fortunately, a psychiatry resident in the first month of training started the patient on antidepressant medication. The resident could offer no objective reason or rationale as to why the medication was started; the patient's "organic brain syndrome" began improving, and within three months he completely recovered and was discharged.

Verwoerdt (1) noted, "All too often, an elderly patient who is essentially depressed receives the diagnosis of organic brain syndrome, after admission to the hospital, and then is treated accordingly. The result is that the patient begins to conform to the attitudes of his environment; i.e., he will start behaving more and more like a senile patient" (p. 46). Al-

though our patient was only 48 years old, it appeared that this was exactly what had happened.

DISCUSSION

The concept of a depressive disorder manifesting as an organic brain syndrome in young adult and middle-aged patients is vitally important to the clinician. As illustrated by the previous case reports, these patients may not experience the typical depressive symptoms.

The first two cases were diagnosed primarily by the psychiatrist's empathic and countertransference response to the respective patients. Kernberg (4) noted that countertransference may include the examiner's conscious and unconscious reactions to the patient and to the patient's reality situation; he suggested that countertransference is useful in understanding the patient. Kernberg stated that patients with borderline and psychotic levels of organization tend "by their intense, premature, and rapidly fluctuating transference, to evoke intense countertransference in the therapist which may at times give the most meaningful understanding" (p. 40). We believe that this is the situation in those patients who are depressed but appear to have an organic brain syndrome.

Mr. C's depression was diagnosed because of his history of depression. This would seem to suggest that the history of depressive disorder is vitally important to ascertain and may in fact be the only clue that a depression is present when the patient appears to have an organic brain syndrome.

In only one case were we able to elicit any material that might have led to a diagnosis of depression; this information was not available until after the patient (Mr. C) began to improve to the point where he could, or would, verbalize his concerns. The lack of losses or life circumstances that would tend to lead one to suspect a depressive disorder, plus the rapidity of response to antidepressant medication in the first two cases, suggests to us that depression was probably a biological phenomenon in these patients.

Mr. D's case is unusual in that a resident physician who was new to the field of psychiatry experienced an empathic response to the patient's depression; failing to realize that antidepressants are not indicated in organic brain syndromes, the resident's actions led to the proper treatment of the patient with subsequent recovery.

It is only through a high index of suspicion and one's empathic response to the patient that this unusual presentation of depressive disorders will be recognized. Because depressive illness is treatable and has a good prognosis, it is essential that it be differentiated from the organic brain syndromes. One can only wonder how many patients have been diagnosed as having organic brain syndrome and hospitalized for chronic care when in fact they are depressed, as was Mr. D.

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Depression in the Wives of Nuclear Submarine Personnel

BY KAREN BECKMAN, ANTHONY J. MARSELLA, PH.D., AND RUTH FINNEY, PH.D.

The authors compared levels of depression among 24 wives of nuclear submarine personnel under conditions of husband-presence and husband-absence in a crossover research design. The wives had significantly higher depression scores under conditions of husband-absence on both the Zung Depression Scale and the Multiple Affect Adjective Checklist. The results are discussed in terms of the tremendous stresses imposed on the wives as a result of the nuclear submarine deployment schedule and the failure of the Navy and the spouses to recognize and reward the wives for their efforts.

UNLIKE PERSONNEL in other branches of the military service, nuclear submarine personnel in the U.S. Navy Fleet Ballistic Missile program typically serve in 3-month rotational cycles; i.e., 3 months at sea, 3 months at home. This cycle is often continued through the entire tour of duty. For the wives of nuclear submarine personnel, this particular service pattern poses a severe stress. It requires a constantly shifting role pattern. During the husband's absence the wife often has complete responsibility for home and family; when the husband returns this role generally shifts back to him. In addition, the wife is faced with a repetitive grief response whenever the husband leaves.

For many submarine wives the difficulties of these two situations are exacerbated by the absence of any institutional resources for coping with the problems. Although counseling facilities are frequently available, the wife seeking help may be labeled "dependent" or "weak" by other wives, her husband's superiors, or even her own husband. The wife is faced with the omnipresent pressure to be a "good military wife"—to sacrifice for the good of her husband's career. However, even among those wives (and there are many) who are willing to fulfill the traditional military wife's role of accepting the burdens of military life, there are few supports. Thus both the resentful wife and the "good military wife" are often forced to keep their feelings regarding the role conflicts and the normal grief reactions to themselves.

Previous publications (1-3) have commented on the problems of the military family. Of special importance is the observation by Frances and Gale (1) that the stress of military life may frequently cause the nuclear family members to become more dependent on each other. What happens, then, when the family separates, as is the case with nuclear submarine personnel? A loss of support can precipitate depressive emotional states. In addition, the separation can signal the forthcoming stress from the additional responsibility and role conflict that the wife will probably experience as she assumes her husband's tasks and obligations (see references 4 and 5 for discussions of role problems among women).

Bermudes, a chaplain, noted that many wives of nuclear submarine personnel came to him with symptoms similar to those of his parishioners who had lost a husband through death (6). He administered a questionnaire to these women and found that the wives evidenced a distinct behavior pattern of "emotional highs and lows similar to the grief responses ordinarily observed in one who has lost a loved one through death" (p. 218). Bermudes identified five stages in the grief response. The last stage was one of despair and despondency.

The purpose of the present research was to examine the adjustment response of the wives of nuclear submarine personnel through an investigation of depression levels under conditions of husband-presence and husband-absence. It was hypothesized that the wives would score significantly higher on depression measures under the condition of husband-absence. The basic research strategy employed was a crossover design to control for possible order effects.

METHOD

Subjects

The sample consisted of 24 wives of nuclear submarine personnel. The mean age of the subjects was 26.3 years, and the mean number of years married was 6.1. The subjects' education level was 12.9 years. They had a mean of 1.6 children each, and the mean age of their spouses was 28.3 years. They were of the same socioeconomic level (only a few were married to

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The authors wish to express their deep gratitude to the wives who participated in this study.

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junior-grade officers; the majority were wives of noncommissioned officers). The subjects had lived in Hawaii for an average of 2.2 years, enough for a minimum of 4 sea-duty tours.

Procedure

The subjects completed 2 measures of depression: the Zung Depression Scale (7) and the Multiple Affect Adjective Checklist (MAACL) (8). These 2 measures were selected because they assess depression by different formats. The Zung scale uses a symptom frequency format, while the MAACL uses a mood adjective checklist. Thus it was believed that the 2 measures would cross-validate one another (9).

The depression measures were administered to 14 subjects whose husbands were on sea duty and to 10 subjects whose husbands were at home. During the next duty cycle the depression measures were readministered to the 14 subjects whose husbands were not on sea duty and the 10 subjects whose husbands were not at home. This type of research design permitted the subjects to serve as their own control for the comparison of levels of depression during both conditions. All measures were administered during the fifth week of the sea duty—home leave cycle. There were no marked differences between the 2 groups in age, education, number of children, or number of years married.

RESULTS

During the first 3-month cycle the wives whose husbands were absent had a mean Zung Depression Scale score of 54.29, while those whose husbands were present had a mean score of 38.50 ($t=3.61$, $df=23$, $p<.001$, t test for independent samples). The wives whose husbands were absent had a mean MAACL score of 15.71, while those whose husbands were present had a mean score of 8.50 ($t=2.52$, $df=23$, $p<.009$, t test for independent samples).

During the second 3-month cycle, in which the conditions were reversed, the wives whose husbands were then present had a mean Zung Depression Scale score of 46.43, while those whose husbands were absent had a mean score of 51.00 ($t=3.08$, $df=23$, $p<.003$). The wives whose husbands were present had a mean MAACL score of 10.21, while those whose husbands were not at home had a mean score of 16.40 ($t=3.29$, $df=23$, $p<.003$). Thus on both measures of depression the wives earned significantly higher scores under conditions of husband-absence.

To compare each subject's score under conditions of husband-presence and husband-absence regardless of the cycle, a t test for correlated samples was used. The mean score on the Zung Depression Scale for all of the subjects under conditions of husband-presence was 43.13, compared with a mean score of 52.92 under conditions of husband-absence ($t=3.51$, $df=23$, $p<.002$). According to Zung scale norms, the latter score suggests the presence of clinical levels of depression. A similar analysis of the MAACL scores for the 24 wives indicated that they had a mean score of 9.50 under conditions of husband-presence and a mean score of 16.00 under conditions of husband-absence ($t=4.26$, $df=23$, $p<.001$). Thus the scores on the different depression measures corroborated one another.

DISCUSSION

The results of this study provide an empirical demonstration of the development of clinical levels of depression among wives of nuclear submarine personnel during the course of their husbands' 3-month absence for sea duty. The depression was extremely responsive to the presence or absence of the husband; in this respect it appears to resemble strongly the grief reaction described by Bermudes (6) and Kübler-Ross (10). Bermudes concluded that there are 5 stages in the grief reaction. Interviews with the wives after the completion of testing revealed anecdotal support for this theory.

The wives reported that they started to withdraw from their husbands weeks before their departure (stage 1—shock). They often argued with their spouses over little things that had no bearing on their leaving. They also lost their patience with their children (stage 2—emotional release). When the husbands left, the wives reported that they withdrew. One wife said that she did not talk to her neighbors during this period because "they seemed silly." All of the wives stated that they felt an added burden of responsibility for the children and the home during their husbands' absence (stage 3—withdrawal). They also noted that they lost their tempers easily. Further, they became angry and resented the Navy (stage 4—anger). Eventually, they felt depressed and cried or slept much of the time (stage 5—depression).

Yet, through all of these cycles, which are repeated again and again in the course of a 4-year hitch (the careerman may keep up these cycles for several hitches), the wife survives, often without any support or recognition from the Navy. She takes care of the home and the children. She goes to work and tries to maintain the image of a "good military wife." She fears that her husband will not love her when he returns, that he will not approve of how she has managed things, and even that she may not love him as much.

The strain of the nuclear submarine service on the wife is great. The Navy should give careful attention to this type of deployment service because of its risks. There is truth behind the adage, "They also serve who only stand and wait." As one wife stated, "I can take all of this. I can even grow as a result of being on my own with all the responsibility. But, just once, . . . just once I would like some recognition for the support I do give to the Navy . . . and perhaps some recognition for being one hell of a woman to put up with this [kind of life]."

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Depression in Viet Nam Veterans and Civilian Controls

BY JOHN E. HELZER, M.D., LEE N. ROBINS, PH.D., ERIC WISH, PH.D., AND MICHI HESSELBROCK, M.S.W.

In order to investigate the long-term psychological consequences of Viet Nam combat, the authors located and personally interviewed a group of 571 randomly selected Viet Nam veterans and 284 matched civilian controls 3 years after the veterans returned to the United States. In the veterans they found a weak association between combat and subsequent depressive symptoms, but the association did not persist after controlling for preservice factors. The incidence of depressive symptoms and syndromes was similar when veterans were compared with nonveterans. Results are contrasted with a 12-month follow-up study of the same veterans in which a stronger association between combat and later depression was found.

IN A PREVIOUS publication (1) we discussed the occurrence of depressive syndromes in a group of Viet Nam veterans after their return to the United States. We found that postwar depression occurred significantly more often in men who had been exposed to combat in Viet Nam than in noncombatants. This relationship persisted after controlling for other relevant preservice and inservice variables.

The first follow-up investigation of our veterans sample was conducted 8 to 12 months after their return to the United States. We have now completed a second follow-up of this same group of veterans 3 years after their return and have interviewed a matched control group of nonveterans for comparison. In this second follow-up report, we will compare the occurrence of depressive symptoms in veterans and nonveterans in the 2 years prior to interview and examine the association of military combat with subsequent depressive syndromes.

METHOD

Approximately 13,760 U.S. Army enlisted men returned to the United States from Viet Nam in September 1971. With the assistance of the military, we selected a sample of 943 men for follow-up study. The sample was divided about evenly into two groups: 1) 470 subjects randomly chosen from all returnees, and 2) 495 subjects randomly selected from a group of men who had been identified as illicit drug users on the basis of a urine screening test at the time of departure from Viet Nam. There was an overlap of 22 men. The subjects were located and personally interviewed 8 to

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12 months after their return to the United States. In our previous study of postcombat depression, we used the data from the 470 "general sample" men only (1).

At the time of the second follow-up, 3 years after return, we reduced the size of the veteran sample by eliminating men from the least populous states and those inducted before 1969. This enabled us to select and examine a matched nonveteran control group. In order to maintain an adequate sample size of veterans, we combined data from the drug-positive and general samples for this second analysis. To avoid biasing the general sample with large numbers of men who used illicit drugs heavily in Viet Nam, we weighted the drug-positive sample to represent its true proportion in the general sample. There were 605 men in this combined veterans sample. We were able to locate and interview 571 or 94%.

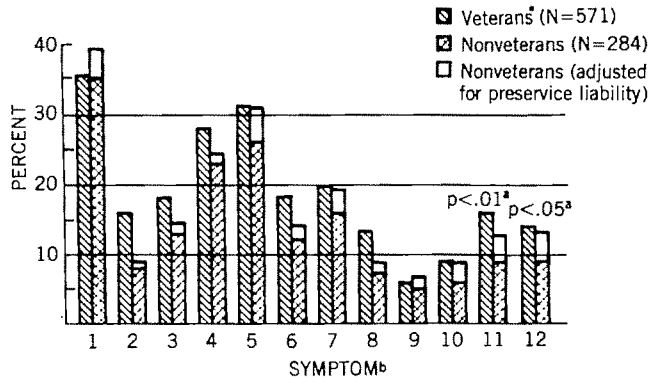
With the help of the Selective Service System, we identified 302 men who were matched to the general sample veterans on the basis of draft eligibility, size and location of predraft residential area, age, and education at the time of the veteran's entry into the military. These were all men who were eligible to serve in the military but did not, either because their lottery numbers were not drawn or they obtained deferments. We located and interviewed 284 (94%) of the controls.

At the first follow-up, we had inquired about depressive symptoms occurring at any time during the 8- to 12-month period since the veteran's return to the States. We did not investigate whether these symptoms occurred simultaneously, because the total interval was fairly brief. The second follow-up interview, covering a 2-year period, made this distinction. The definition of a depressive syndrome for the present study is dysphoria for at least 1 month, occurring at any time during the 2-year period since the first interview, and three or more simultaneous depressive symptoms among those shown in figure 1.

We defined combat in Viet Nam as positive responses to two or more of the three following interview items regarding combat experiences: 1) Did you go on combat patrols or have other very dangerous duty? 2) Were you ever under enemy fire? 3) Were you ever surrounded by the enemy? This also differs from the definition used in the first follow-up study in which combat was defined as a positive response to only the first of these three items. To test the validity of our current definition of combat, we examined two of its correlates. Veterans who met our definition of combat were 50 times more likely to have had friends killed in combat and 90 times more likely to have been wounded.

In the following analysis, we will compare veterans to nonveterans in terms of frequency of depressive symptoms and the depressive syndrome in years 2 and 3 since the veterans' return from Viet Nam. We will also examine whether combat contributes to any excess of depression among the veterans. Since we are interested in isolating the effect of combat as an antecedent stressful event, we will remove from our analysis men who were wounded in combat since physical injuries may profoundly influence psychological adjustment.

FIGURE 1
Depressive Symptoms Over a Two-Year Period



[a]Differences are significant only before adjusting for preservice differences.
[b]1=dysphoria at least 1 month; 2=insomnia; 3=anorexia and weight loss (8 lbs.); 4=loss of energy; 5=loss of interest; 6=feelings of guilt; 7=poor concentration; 8=worry about insanity; 9=crying spells; 10=thoughts of death or self-harm; 11=depressive syndrome; 12=depressive syndrome excluding 77 wounded veterans.

RESULTS

In figure 1, the cross-hatched bars show the frequency of depressive symptoms occurring over a 2-year period in all of the interviewed veterans and nonveterans. With the exception of dysphoric mood, which was equal for the two groups, substantially higher proportions of veterans reported each of the depressive symptoms, and several of these differences are statistically significant. More of the veterans met our criteria for a depressive syndrome occurring at some time during the 2-year follow-up (χ^2, $p<.01$). The difference in depressive syndrome continues to be significant after excluding the veterans who were wounded in Viet Nam ($p<.05$).

Despite our matching procedure, there were distinct differences between veterans and nonveterans for certain preservice variables such as antisocial behavior and drug use. Since many of these preservice factors were found to be powerful predictors of social adjustment and depressive symptoms after return from Viet Nam, it was obviously necessary to control for these before we could attribute postservice differences to Viet Nam or to the military experience. We constructed a preservice liability scale consisting of demographic descriptors, deviant behavior, and drug use (2). We then controlled for differences in preservice liability in the veterans and nonveterans by using an epidemiological method known as direct standardization (3). The open bars in figure 1 show the proportions of depressive symptoms in nonveterans after we adjusted for preservice differences. The veterans continued to exceed the nonveterans on most of the symp-

toms, but the differences were less striking. Differences in the frequency of the depressive syndrome were no longer significant.

Among men who met our criteria for a depressive syndrome, the severity of the disorder was similar for veterans and controls. Some symptoms were reported more frequently by the veterans, but most were reported more frequently by the nonveterans. A slightly higher proportion of the veterans reported four or more clustered symptoms (70% versus 66% for the weighted nonveterans), but more of the nonveterans saw a physician for depression.

The depressive syndrome, as we have defined it, was not a trivial problem. About one-third of both the depressed veterans and nonveterans had thoughts of death or self-harm. Almost half of each group reported anorexia, with an 8-pound weight loss, and about 60% had guilt feelings. About one-quarter of each group had to take time off from their regular activities because of depressive symptoms.

We also examined the relationship between the veterans' combat experience in Viet Nam and depressive syndromes 2 to 3 years after their return. Among the 494 nonwounded veterans whom we interviewed, 14% reported the occurrence of a depressive syndrome at some time during the 2-year follow-up period. Dividing the sample into combatants and noncombatants, we obtained proportions of 18% and 10%, respectively. This difference is significant ($p<.02$). In addition, there was a direct relationship between the number of combat events and the proportion of veterans reporting subsequent depressive syndromes.

An association of this type does not prove a causal relationship. There may be other pre-Viet Nam characteristics that predict both combat assignment and later depression which lead to a false impression that combat causes depression. In our previous study, we found a number of preservice variables that did indeed predict both combat assignment and depression, but we controlled individually for these and found that combat experience continued to predict a depressive syndrome in the first 8 to 12 months after return (1).

In this second follow-up, we found four preservice variables that predicted both combat assignment and depression: antisocial behavior, education, parental psychiatric care, and parental arrest. When we controlled for any one of these preservice variables, the relationship between Viet Nam combat experience and subsequent depression was no longer significant. Preservice antisocial behavior, for example, was quantified with a 10-point scale. We assigned up to 2 points for deviance in each of five areas: truancy, school dropout, drunkenness at a young age, fighting that led to trouble with authorities, and arrests. We then controlled for early antisocial behavior by examining the relationship between combat assignment and depression at various levels of preservice sociopathy. In every subgroup, with the exception of the least antisocial men, combatants reported more subsequent depression than did noncombatants, but this difference was not significant. The same was true for preservice education. At all levels of education, combatants reported a nonsignificant increase in subsequent depressive syndromes.

To see whether the apparent reduction in the importance of combat assignment as a predictor of later depression at the second follow-up was a function of time or an artifact of our change in sample, definitions, and analysis, we compared the predictive value of combat in the first and second follow-up periods. We used identical definitions of combat and depression, identical veterans samples (N=571 veterans), and identical methods of analysis. For both follow-up periods, we did a multivariate analysis (4) using the four preservice variables mentioned above, with depressive syndrome as defined in the first follow-up as the outcome variable. We then repeated these analyses with combat assignment added as a fifth predictor to see how much increase in explained variance there would be. In both instances, the addition of combat increased the explained variance by only a small amount, but the contribution of combat to depression was clearly greater at the first follow-up than at the second (4% versus 1%). Thus, we conclude that the development of a depressive syndrome was influenced by prior combat experiences at first follow-up but that these effects were largely dissipated by the time of the second interview.

There was little carry-over in depression from the first to the second follow-up. Only 28% of those who met our criteria for depression the first time did so the second. This represented only 2% of the entire nonwounded veteran sample. It would have been interesting to see if postcombat depressions were less persistent than those which were not combat related, but the numbers were too small for meaningful conclusions.

DISCUSSION

This investigation provides a unique opportunity to examine the effect of serving in the military and specifically the effect of the stress of combat on the long-term psychological adjustment of veterans. The sample is large and was randomly selected from men who served in Viet Nam at the height of the military campaign. Follow-up was by personal interview given by trained examiners using a structured instrument. The controls were matched for age, educational status, and other demographic characteristics. In comparing these carefully selected samples, we were unable to find much evidence of long-term depressive symptoms or syndromes that might be attributed to the military experience. It is true that the absolute proportions of veterans exceeded those of nonveterans for most of the depressive symptoms we inquired about. However, when we controlled for preservice factors that were clearly related to later psychological adjustment, these differences diminished. Preservice differences seem to play a much stronger role in psychological ad-

justment 2 to 3 years after return than do inservice variables.

We also found that combat experience played an insignificant role in the veterans' psychological adjustment 2–3 years after their return to the States. In a war like Viet Nam in which there were no "front lines" and even those stationed within major cities lived under the threat of bombardment, it is not as clear what constitutes a combat experience. It may be that combat as we have defined it is not an important predictor because it does not differentiate combatants from noncombatants in terms of the risk they were exposed to. However, the striking increase in the likelihood of being wounded or having friends killed in combat suggests that combat, as we have defined it, does identify those men particularly exposed to danger and risk during their Viet Nam experience.

Both the similarities in symptoms between veterans and nonveterans and the weak association between combat experience and depressive symptoms would suggest that the Viet Nam conflict produced few long-term psychological effects of the type we inquired about. There are no previous reports of large systematic comparisons of depressive symptoms in veterans and nonveterans. There are, however, previous studies of the relationship between combat and later depression, and the results appear to be consistent with our own. Nace and associates (5) recently reported a 2-year follow-up of over 200 veterans who had been hospitalized or were in a drug treatment center in Viet Nam. They administered the Beck Depression Inventory to their respondents and found a nonsignificant trend toward greater depression in combatants. Brill and Beebe (6), in their 5-year follow-up of World War II veterans, found that equal percentages of combatants and noncombatants were ill at the time of examination. Also consistent with our findings, they found that those with preservice personality disorders were 6 times more likely to be disabled at follow-up. Archibald and Tuddenham (7) found that stress reactions in World War II combat veterans were still present after 20 years; however, overall prevalence cannot be determined from their study, since their sample was selected from men under treatment at follow-up.

Because of differences in samples and definitions, our first and second follow-up studies are not entirely comparable. However, comparing the two as best we can, we find evidence at the first follow-up that combat was a predictor of a depressive syndrome and that this association did not disappear when preservice variables were controlled for. However, few of the men reported a depressive syndrome at both follow-up examinations. The association between combat and depression at the second follow-up was weak and did not continue to be significant after controlling for relevant preservice variables. The results of these two studies provide some evidence that combat as a stressful life event did predict depressive syndromes in Viet Nam veterans, but these depressions were short-lived, and the effect of combat as a predictor diminished over time.

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Depressed Mood and Subsequent Physical Illness

BY EVELYN L. GOLDBERG, SC.D., GEORGE W. COMSTOCK, M.D., DR.P.H., AND ROBIJN K. HORNSTRA, M.D.

The authors describe the results of a matched case-control analysis in which 82 pairs of subjects were selected from a large-scale community study carried out in two general populations. Cases were those of persons hospitalized during an interval between two interviews for an illness starting during that interval or persons who died during the interval. Controls were persons with no new illnesses or hospitalizations during the interval. No relationship between measures of depressive symptomatology or depressed mood and subsequent physical illness was shown. This result corroborates the findings of prospective studies in more selected populations and suggests that it is unlikely that depressed mood is related to subsequent physical illness.

DEPRESSIVE ILLNESSES are considered to be a major public health problem (1). They are very common, but increasingly effective methods of treatment are becoming available. To increase the success of these new therapeutic modalities, it is desirable to know the natural history of the disease so that treatment may be applied at points where its effectiveness is optimal.

An important and as yet unknown aspect of the natural history of depression is whether or not depressed mood is a precursor of serious illness. This is a difficult question to answer even when the outcome studied is limited to serious physical illness. Cross-sectional and case-control studies are unlikely to yield convincing answers because of the difficulty in determining whether depressed mood preceded the illness, accompanied it, or was caused by the illness. Prospective studies can establish that symptoms of depression did precede the onset of serious physical illness more often than expected by chance, and while they cannot establish a cause-and-effect relationship because of the possibility that depressed mood may be the first symptom of a physical illness that is not yet recognizable (2–4), a failure to find an association rules out an etiological relationship.

Among the few prospective studies of this nature that have been reported, some have looked at the health of persons already diagnosed as having a depressive illness and have found that these patients do have higher rates of disease and death than the general population. Heine (5) showed a relationship between depressive illness and subsequent hypertension in a follow-up study. Excess mortality for patients with various categories of depression has been reported in seven separate studies (6–12). Suicides accounted for some of the excess, especially in the Ciompi study (10), in which most of the lower life expectancy was due to suicides.

Following psychiatric interviews of the entire adult population of two adjoining parishes in Sweden during 1947, Hagnell restudied the 2,550 subjects 10 years later (13). Each individual had originally been rated on the personality dimension of stability on the basis of responses to questions and behavior during the interview. Women classified as having substable personalities, a rubric similar to Eysenck's extravert and Kretchmer's cyclothymic types, were found to have an excess risk of subsequent cancer; this was not true for men (14).

Thomas and her coworkers have been following a group of 1,130 white males who registered in medical school between 1948 and 1964. Among those with evidence of depression at the initial examination there has been an increased risk of suicide but as yet no increased risk from other causes (15).

A larger and longer study based on data routinely collected from students on entry into university programs has been reported by Paffenbarger and coworkers (16–22). Information was available from more than 13,500 men who were admitted to the University of Pennsylvania from 1931 to 1940. Included in the initial information were a number of psychological symptoms such as palpitation, nervousness, insomnia, exhaustion, worrying, moodiness, alternating gloominess and cheerfulness, self-consciousness, and inclinations to be secretive or seclusive. On follow-up 24 to 33 years later, it was found that a sense of exhaustion on initial interview was related to subsequent death

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from coronary heart disease. No association was found with the depression-related symptoms of moodiness or of alternating gloominess and cheerfulness (16). Another study (22) done 22 to 31 years later showed no association between depression-related symptoms of moodiness or cyclothymia and the incidence of peptic ulcer.

Because of the potential importance of the association of symptoms of depression and subsequent serious physical illness, the present paper reports the findings from a large prospective study expressly designed to measure the frequency of depressed mood in two distinct general populations and to assess its association with subsequent disease.

METHOD

Representative samples of dwelling units were selected in Kansas City, Mo., and Washington County, Md. Cluster sampling was used in Kansas City and systematic sampling from a geographically arrayed list in Washington County. A study subject was then randomly selected from persons over the age of 18 in each selected dwelling unit. In both communities, the sampling procedures were designed to yield successive monthly samples of adults who were representative of all community adults except for stratification by the number of adults in the household.

Interviews were conducted similarly in each community. The initial interviews were carried out between October 1971 and January 1973 in Kansas City and between December 1971 and July 1973 in Washington County. The response rates were 72% and 80%, respectively.

The initial interview schedule contained, in addition to demographic and general health questions, a number of scales designed to measure depression, happiness, and satisfaction with life. Symptoms of depression were assessed by the Center for Epidemiologic Studies Depression Scale (CES-D) (23) and depressed mood by the Lubin Depression Adjective Check List (DACL) (24, 25). Overall happiness was measured by scales developed by Bradburn (26, 27) and Gurin (28). The Cantril Ladder (29) provided an evaluation of the subjects' overall perceptions of their current life situations. In all instances, higher scores reflect more symptoms of depression, less happiness, or less satisfaction with present life situations. Cutting points were selected so that, in general populations, persons in the group with scores greater than the cutting point represent approximately 20% of the total.

The CES-D used 20 questions selected from five other depression questionnaires for their ability to discriminate between depressed and nondepressed persons (30). Duration of symptoms during the last week was rated on a scale from 0 through 3, yielding scores from 0 to 60. Cutting the scale between 15 and 16 has been shown to differentiate between depressed and nondepressed persons with considerable accuracy (31–33). The Lubin DACL is also designed to measure depressed mood. Results correlate well with those from the CES-D (31).

The Gurin Global Happiness Scale is derived from a single question relating to the subject's present happiness (28), while the Bradburn Happiness Week Balanced Scale uses several questions to assess the subject's mood (26, 27).

In using the Cantril Ladder, Present (29), the interviewer asks which rung of the "ladder of life" seems most appropriate to the subject's present condition—at the top (the best possible life), at the bottom (the worst possible life), or at some intermediate point on the scale from 0 (worst) to 10 (best). To keep the direction of the scale consistent with the others, these values were reversed by subtracting the value given by the subject from 10.

Reinterviews were restricted to a sample of the initial respondents. In Kansas City, the sample consisted of persons originally interviewed between July 1972 and December 1972; all were reinterviewed approximately 12 months after the initial survey. In Washington County, reinterviews took place between August 1973 and April 1974; the subjects were persons whose initial interviews had occurred 6 or 12 months earlier. There were 343 respondents to reinterviews in Kansas City and 660 in Washington County, representing response rates of 78% and 79%, respectively. A major purpose of the reinterviews was to ascertain illnesses that had occurred during the period since the initial interview. The portion of the questionnaire designed to elicit this information was patterned after questionnaires used in the health interview surveys conducted by the National Center for Health Statistics (34). Several different approaches were used to assure that illnesses were not forgotten or overlooked.

The subjects for the present analysis were Caucasians who completed interviews at both times and who responded to the initial interview but who died within the study areas during the study period. Nonwhites were excluded because of their small numbers in the final sample.

To expedite the analysis, a case-control approach was chosen even though the study was prospective in nature. Cases were the 66 subjects who had been hospitalized for an illness that started during the interval and the 16 who had died in the period between interviews, a total of 82 cases.[1] Controls were study subjects without new illnesses, hospitalization, or death between interviews. Each control was individually matched to a case by location, length of follow-up interval, sex, age (plus or minus five years), education, income, and employment status.

The findings were examined by matched-pairs anal-

[1]Results reported in an earlier paper (35) were based on data from 83 matched pairs. One of the original 83 matched pairs was eliminated from the present analysis because of a previous coding error; the omission of this pair made only trivial differences in the results found.

TABLE 1
Comparison of 82 Case-Control Pairs by Matching Variables

Characteristic	Cases (percent)	Controls (percent)
Location		
Washington County	56.1	56.1
Kansas City	43.9	43.9
Follow-up interval		
6 months	6.1	6.1
12 months	93.9	93.9
Sex		
Female	65.9	65.9
Male	34.1	34.1
Age		
18–44	39.0	41.4
45–64	34.1	29.3
65 and over	26.8	29.3
Educational status		
College degree	12.2	7.3
1–3 years college	17.1	15.9
High school degree	21.9	32.9
7–11 grades	36.6	35.4
0–6 grades	12.2	8.5
Employment status		
Working	47.6	50.0
Housewife	23.2	24.4
Retired/student	25.6	23.2
Unemployed	3.6	2.4
Income		
Less than $4,000	31.7	24.4
$4,000–7,999	17.1	18.3
$8,000–11,999	12.2	21.9
$12,000–15,999	21.9	20.7
$16,000 and over	9.8	9.8
Not stated	7.3	4.9

ysis, using McNemar's test to assess statistical significance (36). Median scores for each of the five scales were also looked at for cases and controls.

RESULTS

The final study group was composed of 82 matched pairs of cases and controls, 36 from Kansas City and 46 from Washington County. Their matched characteristics are compared in table 1. All of the differences between case and control groups could easily have occurred by chance. Because this was true both within and between each community, the results from the two locations were combined.

The categories of illnesses reported to have caused death or hospitalization are shown in table 2. All subjects but one were hospitalized for physical illnesses (one case was diagnosed as neurosis). Causes of death were ascertained from death certificates. For a sample of 23 subjects in Washington County, hospital records were examined to validate the diagnoses reported at reinterview. For 20 of the 23, the patients' statements and the hospital records agreed; for 2 others, there was agreement with respect to the general nature and location of the condition. One patient claimed to have had a stroke, while the hospital record referred only to an undiagnosed kidney lesion.

TABLE 2
Categories of Illness Causing Death or Hospitalization Among 82 Cases[a]

Illness Category[b]	Number
Gastrointestinal	19
Genitourinary	18
Neoplasms	11
Cardiovascular	10
Accidents (all types)	8
Respiratory	5
Musculoskeletal	5
Metabolic	5
Skin	4
Diseases of pregnancy	3
Bacterial and viral	2
Cerebrovascular	2
Dental	1
Neurosis	1
Nervous system	1
Undetermined cause of death	1

[a]Cause of death for 3 Kansas City subjects and 1 Washington County subject omitted due to inadequate data.
[b]ICD codes available from Dr. Goldberg on request.

TABLE 3
Matched-Pairs Analysis of Findings from Five Depression-Related Scales

	Cases and Controls				
	Concordant		Discordant		
Scale[a]	Both Normal	Both Abnormal	Case Abnormal	Control Abnormal	χ^{2b}
CES-D (16–60)	57	5	9	11	.05
DACL (11–34)	50	2	16	14	.03
Gurin Happiness (2)	68	0	9	5	.64
Bradburn Happiness (4–9)	47	5	16	14	.03
Cantril Ladder, Present (5–10)[c]	47	3	17	12	.55

[a]Numbers in parentheses represent "abnormal" values.
[b]Using McNemar's chi square (36).
[c]Analysis performed on only 79 pairs because of missing data for 1 member of each of 3 pairs.

Results of the matched-pair analyses for the five scales of interest are shown in table 3, along with scores on these scales considered "abnormal." In no instance is there any indication that symptoms of depression or feelings of unhappiness were associated with subsequent serious physical illness or death. Although more cases than controls assessed their present life situation as unsatisfactory, this difference was well within chance limits.

Median scores for each of the five scales are shown separately for cases and controls in table 4. There is little indication that the cases were more depressed prior to their illnesses than the controls. The very slight differences in median scores are consistent with the fact that controls were slightly better off with respect to education and income, both of which are negatively associated with depressed feelings (37).

TABLE 4
Median Scores on Five Depression-Related Scales for Cases and Controls

Scale[a]	Cases	Controls
CES-D (0-60)	7.2	5.8
DACL (0-34)	7.1	7.6
Global Happiness (0-2)	0.9	0.9
Bradburn Happiness (0-9)	2.3	1.8
Cantril Ladder, Present (0-10)	2.7	2.0

[a]Numbers in parentheses represent range of possible scores.

DISCUSSION

This study, using comparisons of cases and matched controls with prospectively gathered data, failed to show a relationship between physical illness and depressed mood measured in various ways prior to the onset of illness. The cases—persons who died or persons with a physical illness requiring hospitalization—had no more symptoms of depression or depressed mood before becoming sick than the controls who remained well. Measures of overall happiness and present satisfaction with life, both of which are correlates of depression, also failed to show significant differences between the two groups.

Because the illness was not present (or at least not known) at the time of the initial interview, there is little question that the history of depression-related findings was obtained prior to the onset of physical illness. Of course, if disease had been present but still unapparent at the initial interview, it might have affected the level of depression of the cases. Given the wide variety of illnesses, however, it seems unlikely that this could have happened often enough to be important. Because the disease was not known at the time of the initial interview, the depression scores could not have resulted from reactions to the diagnosis of an illness.

Physical illness and death are both commonly found to be more frequent among low socioeconomic groups (38-41). A similar association is also found for depression (37, 42-45). It is not surprising, therefore, that studies which did not make allowances for this common link found mortality to be increased among depressed persons (6, 7, 9, 10). Prospective studies such as those by Paffenbarger and associates (16, 22) and by Thomas (15) in effect adjusted for socioeconomic status by the fact that their study populations were white males who had attended private universities. Neither of these two studies found evidence of an association of depression-related symptoms with subsequent illness or death, except for deaths due to suicide.

In addition to controlling for a considerable number of demographic and socioeconomic characteristics, the present study also has the advantage of being replicated in two independent general populations, each with a broad range of demographic, socioeconomic, and personal characteristics. Its findings over the short term in these broad-based populations agree with the results of long-term observations by Paffenbarger and associates (16, 22) and Thomas (15) in more restricted populations. This agreement makes it appear unlikely that depressed mood is related in a causal way to subsequent physical illness.

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Depression: New Evidence in Support of Biological Differentiation

BY PAUL E. GARFINKEL, M.D., JERRY J. WARSH, M.D., PH.D., AND HARVEY C. STANCER, PH.D., M.D.

The authors studied monoamine metabolism in patients with bipolar (manic-depressive) and unipolar depressive illness and in normal control subjects under strict dietary conditions before and during administration of carbidopa, a peripheral decarboxylase inhibitor. They found that unipolar depressed patients excreted higher amounts of 3-methoxy-4-hydroxyphenylglycol (MHPG) and 5-hydroxyindoleacetic acid (5-HIAA) in the drug-free period and while receiving carbidopa and significantly less tryptamine only after carbidopa administration. Plasma tryptophan levels differed in the three groups. The authors state that their research design reveals changes in serotonin and norepinephrine metabolism in unipolar depressed subjects that distinguish this group from normal and bipolar depressed subjects, suggesting a reduced CNS uptake of tryptophan in unipolar depression.

THE DIFFERENTIATION of depressive syndromes has largely been based on clinical history. Exclusion and inclusion criteria to increase the reliability of diagnosis have also been described (1). Further, genetic studies and reports on responses to psychotropic medications have provided evidence to support the differentiation of primary depression into those conditions with a positive history of mania (bipolar, manic-depression) and those in which mania is absent (unipolar depression) (2–4). Recent reports suggested that diagnostic differentiation may be enhanced through the measurement of catecholamine metabolites (3-methoxy-4-hydroxyphenylglycol or MHPG) in urine (5–7) or serotonin (5-HT) metabolites (5-hydroxyindoleacetic acid or 5-HIAA) in cerebrospinal fluid (CSF) (8–10).

We have demonstrated the use of the selective peripheral decarboxylase inhibitor carbidopa to enhance the specificity of in vivo indexes of CNS biogenic amine function (11–13). This strategy has been used to reveal differences between unipolar and bipolar depressive patients and normal control subjects in both catecholamine and indoleamine metabolism. The biochemical profile thus obtained appears to distinguish a more homogeneous subgroup of patients with unipolar depression in which there may be a disturbance in CNS serotonin function secondary to the reduced availability of tryptophan.

METHOD

We studied 18 depressed patients (11 bipolar and 7 unipolar) with a diagnosis of primary affective disorder (1) and 10 control subjects on a clinical research ward where diet, medication, and activity were strictly controlled, as described in earlier reports (11–13). The bipolar patients were 6 men and 5 women whose mean age was 38.4 years (S.E.=3.7 years). The unipolar patients were 6 men and 1 woman; their mean age was 35.3±3.3 years. The control subjects were normal volunteers (6 men, 4 women) with no history of psychiatric disorder. Their mean age was 31.9±2.4 years. Informed consent was obtained from all of the subjects.

Following a 7-day drug-free period, each subject was studied over 10 days. This consisted of a 3-day baseline period followed by 7 days of receiving 100 mg of carbidopa three times daily. Urine was collected over 12-hour intervals (9 a.m. to 9 p.m. and 9 p.m. to 9 a.m.) for the entire 10 days for determination of 5-HIAA, MHPG, and tryptamine by methods described in earlier reports (11, 13). Free and total plasma tryptophan were determined on the first and second days of the baseline period and on the sixth and seventh days of carbidopa administration, as described in an earlier report (12). A multivariate analysis of variance was used for all 10 days and 10 nights for analysis of

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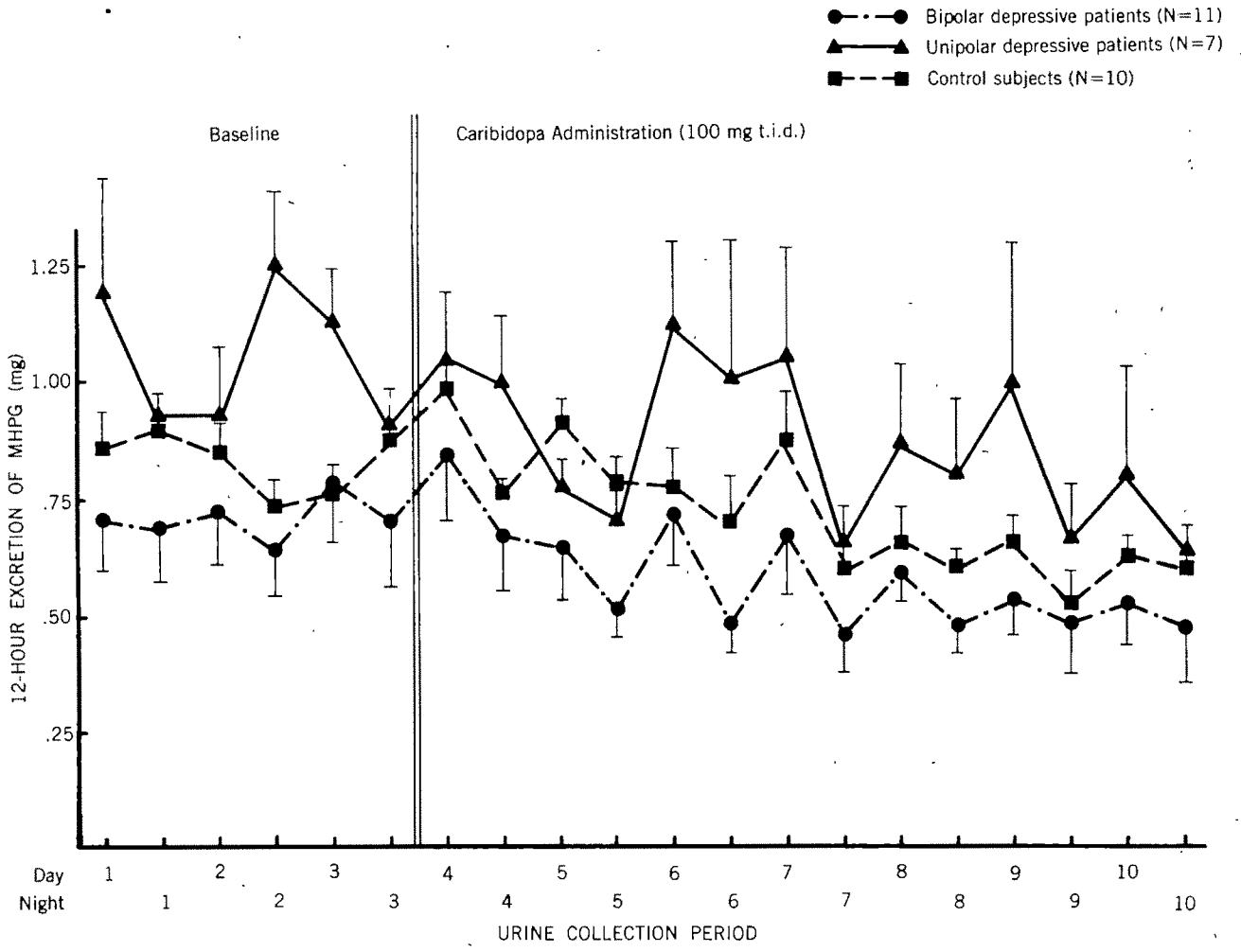
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FIGURE 1
Urinary 5-HIAA Excretion in Bipolar (Manic-Depressive) and Unipolar Depressed Patients and Control Subjects



MHPG and 5-HIAA data. For tryptamine, the multivariate analysis was redone using data from days 1, 2, 4, 9, and 10, including the 5-HIAA and MHPG data from these same days. Total and free plasma tryptophan were analyzed by univariate analysis of variance for days 1, 2, 9, and 10. Significant results from these analyses were followed by Duncan's Multiple Range Test for cell differences (14).

RESULTS

The results of the measurements of 5-HIAA and MHPG excretion for each of the 10 days are shown in figures 1 and 2, respectively. Unipolar depressed subjects had significantly higher MHPG ($p<.05$) and 5-HIAA ($p<.05$) excretion compared with bipolar depressed or control subjects. Carbidopa administration resulted in statistically significant decrements in MHPG ($p<.001$) and 5-HIAA ($p<.001$) excretion in all three groups of subjects. Despite the decrease, the MHPG and 5-HIAA excretion levels remained elevated in unipolar depressed subjects compared with the excretion levels in bipolar depressed and normal subjects.

No differences were found in urinary tryptamine excretion during the precarbidopa baseline period (see table 1). As previously reported (11), administration of carbidopa resulted in a profound decrease in urinary tryptamine excretion in all subjects (more than 85%, $p<.001$). However, the urinary tryptamine excretion in unipolar depressed subjects was found to be significantly lower ($p<.05$) than in bipolar depressed or normal subjects receiving carbidopa.

Free plasma tryptophan concentrations (see table 2) were not different in the three groups during the baseline period. Free plasma tryptophan was significantly elevated in unipolar depressive patients ($p<.05$) and decreased in control subjects ($p<.05$) given carbidopa. These findings were not altered by partitioning out possible correlations in total tryptophan. No significant inter- or intragroup differences in total plasma tryptophan were evident before or during carbidopa administration.

FIGURE 2
Urinary MHPG Excretion in Bipolar (Manic-Depressive) and Unipolar Depressed Patients and Control Subjects

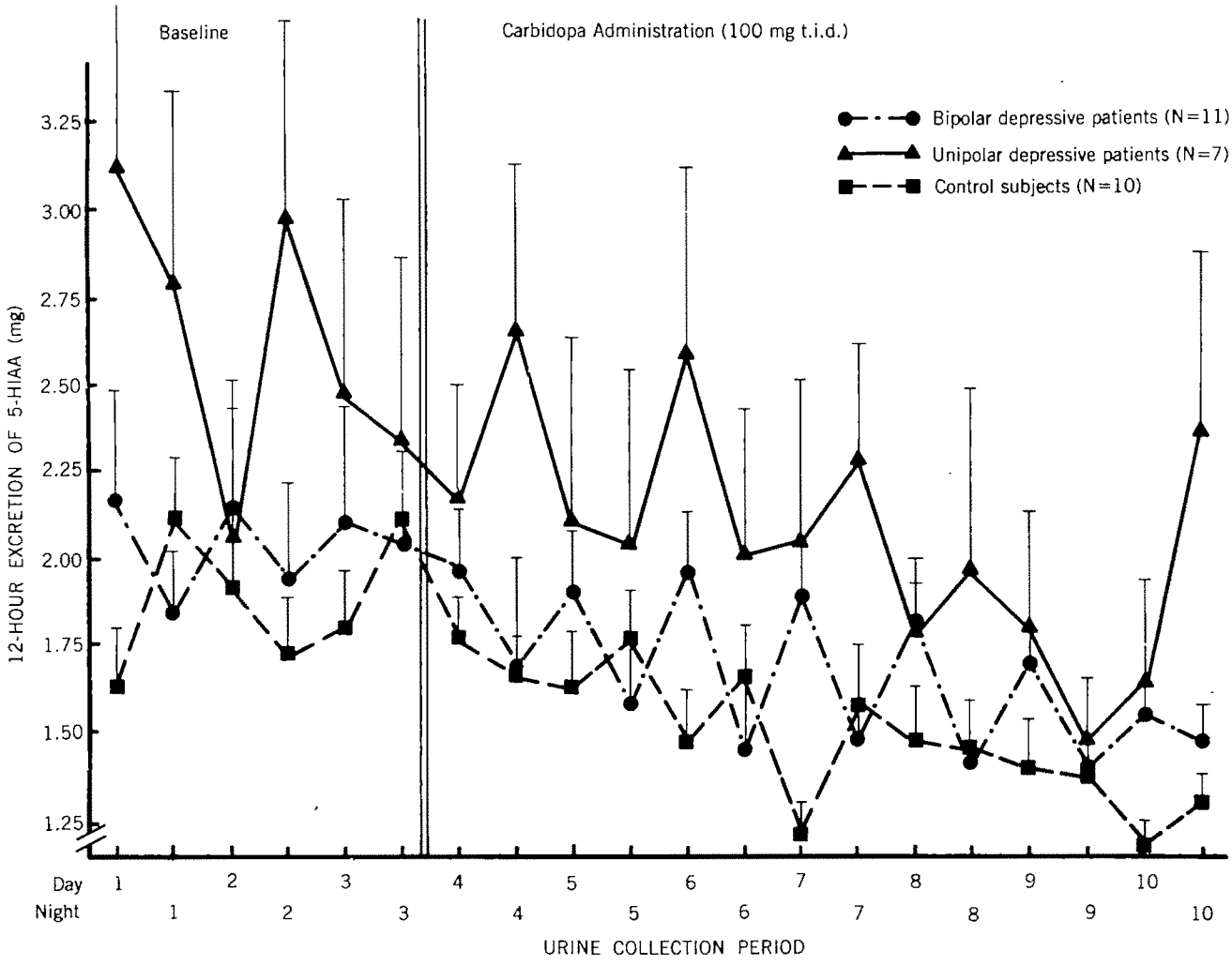


TABLE 1
Twenty-Four-Hour Urinary Tryptamine Excretion (μg/24 hours)

Time of Collection	Control Subjects (N=10)		Unipolar Depressive Patients (N=7)		Bipolar Depressive Patients (N=11)	
	Mean	SE	Mean	SE	Mean	SE
Day 1 (baseline)	103.0	25.7	81.0	28.7	93.8	8.7
Day 2 (baseline)	117.8	38.9	83.8	23.3	116.1	25.7
Day 4	14.3	6.4	1.4[a]	0.5	6.4	1.8
Day 9	4.5	1.5	0.8[a]	0.2	7.2	2.1
Day 10	7.2	3.3	1.1[a]	0.5	7.7	2.5

[a]$p<.05$ compared with bipolar depressed subjects and control subjects.

TABLE 2
Free Plasma Tryptophan Concentrations During Baseline and Carbidopa Administration Periods (μg/ml)

Group	Baseline		Carbidopa Administration	
	Mean	SE	Mean	SE
Control subjects (N=10)	2.05	0.12	1.78[a]	0.12
Unipolar depressive patients (N=7)	2.08	0.15	2.32[b]	0.10
Bipolar depressive patients (N=11)	2.02	0.10	2.11	0.11

[a]$p<.05$ comparing baseline and carbidopa periods; $p<.05$ comparing control subjects with bipolar and unipolar depressive patients receiving carbidopa.
[b]$p<.05$ comparing baseline and carbidopa periods.

DISCUSSION

These results demonstrate a distinct profile of alterations in both indoleamine and catecholamine metabolism in unipolar depressive subjects but little change in bipolar depressed subjects. These findings were brought out only by use of a perturbation technique with a peripheral decarboxylase inhibitor.

In 1976 we reported differences in the disposition of plasma tryptophan in patients with primary depression compared with normal control subjects (12). Carbidopa produces a decrease in free plasma tryptophan in normal subjects, an effect probably mediated through reduced peripheral catecholamine metabolism and subsequent reduced lypolysis and mobilization of

unesterified free fatty acids. This inference is supported by evidence that β-adrenergic-blocking drugs prevent stress-induced increases in plasma unesterified fatty acids and subsequently free plasma tryptophan (15) but adrenaline increases these fatty acids (16). Unipolar depressed subjects display a rise rather than a decrease in free plasma tryptophan in response to carbidopa. As we suggested in 1976 (12), the differential effect of carbidopa in increasing free plasma tryptophan in unipolar depressed subjects but decreasing it in normal subjects may indicate a reduced efflux of tryptophan from the plasma compartment in unipolar depression. This reduced efflux becomes evident only after inhibition of the metabolic clearance of tryptophan through peripheral indoleamine and possibly kynurenine and transaminase pathways that may also be inhibited by carbidopa (17, 18).

The finding of reduced urinary tryptamine excretion in unipolar compared with bipolar or normal subjects supports the concept of a reduced egress of tryptophan from plasma. Tryptamine is a direct product of tryptophan decarboxylation (19). It is likely that CNS tryptamine turnover is dependent on the availability of tryptophan because of the high Km (Michaels-Menten constant) for tryptophan decarboxylation in brain and because brain tryptophan concentrations are below the saturating concentration for decarboxylation (20). Because carbidopa does not penetrate into brain and because it produces a profound inhibition of peripheral tryptamine excretion, it is likely that a large fraction of the tryptamine excreted derives from noninhibited pools of tryptamine formation such as brain. Thus reduced tryptamine excretion may reflect reduced tryptophan availability in the CNS secondary to reduced efflux or uptake from the plasma compartment.

The elevated 5-HIAA excretion in unipolar compared with bipolar depressed or normal subjects would also support this concept. Although urinary 5-HIAA excretion during carbidopa administration was significantly reduced, the gradual and progressive decrement in 5-HIAA excretion suggests that after carbidopa administration some 5-HIAA secreted in urine probably still derives from peripheral pools (13). Elevated 5-HIAA excretion may represent a peripheral compensatory route of metabolic efflux of tryptophan secondary to a reduced clearance of tryptophan from plasma through normally functioning pathways. If, in fact, the significantly reduced urinary tryptamine excretion in unipolar depressed subjects receiving carbidopa is an index of reduced brain tryptophan, this would suggest that a defect in net uptake of tryptophan is present in unipolar depression. Because of the dependence of brain 5-HT on substrate availability, such an impairment of brain tryptophan uptake could lead to reduced brain 5-HT function. Reports of reduced volume of distribution and turnover of plasma tryptophan (21, 22), lowered CSF tryptophan levels (23), and postmortem findings of reduced 5-HT and 5-HIAA in the brains of depressed subjects (24–26) support the latter hypothesis.

The elevated MHPG excretion observed in unipolar depressive patients concurs with earlier findings (5, 7). We found that MHPG excretion in bipolar depressed patients tended to be lower than in normal control subjects, but this difference did not reach statistical significance. The reduction of peripherally derived MHPG following carbidopa administration did not alter our findings. These results persisted after a separate analysis of the male subjects alone. MHPG has been reported to be reduced in bipolar depressed patients (5, 7, 13).

The increased urinary MHPG excretion in unipolar depressed patients may reflect increased CNS noradrenergic function. Furthermore, the changes in indoleamine metabolism reflect the possibility of reduced CNS 5-HT activity. These alterations may be simultaneous and independent. However, there is considerable evidence supporting a functional interaction between brain 5-HT and noradrenergic systems (27), with 5-HT and noradrenergic systems being reciprocally antagonistic (28). If such is the case, reduced CNS 5-HT function might directly or indirectly result in increased noradrenergic turnover and production of MHPG. Since a major fraction of urinary MHPG appears to derive from the CNS (29, 30), the increased urinary MHPG excretion in unipolar patients may reflect such an increased CNS noradrenergic metabolism resulting from reduced 5-HT function. The present data represent the first (to our knowledge) demonstration relating affective illness to a possible imbalance in CNS amine function. This imbalance is demonstrable in unipolar depression.

Alternatively, the MHPG findings could be due to increased adrenal catecholamine synthesis, which appears to be partly under CNS 5-HT inhibitory control (31). Reduced brain 5-HT activity could result in increased peripheral adrenergic metabolism and subsequently in increased MHPG production. It has been shown that a significant fraction of urinary MHPG may arise from peripheral epinephrine as well as norepinephrine metabolism (32).

The possibility of a reduced CNS tryptophan uptake in unipolar depressive patients may explain the inconsistencies surrounding the therapeutic efficacy of tryptophan in depression (33). An abnormality affecting the net uptake of tryptophan into brain might limit the ability of exogenously administered tryptophan to increase brain tryptophan and indoleamine metabolism. It is interesting that lithium increases brain tryptophan uptake and, initially, brain 5-HT synthesis (34). This suggests that lithium combined with tryptophan might improve the CNS uptake of tryptophan in unipolar depressed subjects, thus enhancing its antidepressant effects.

We have demonstrated changes in 5-HT and noradrenergic metabolism in unipolar depressed subjects that distinguish this group from normal and bipolar depressed subjects. These findings suggest a reduced CNS uptake of tryptophan in the pathochemistry of unipolar depression. Primary affective disorders ap-

pear to consist of a number of biochemically distinct subpopulations.

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Physical Activity and Plasma Cyclic Adenosine Monophosphate Levels in Manic-Depressive Patients and Healthy Adults

BY ELEFTHERIOS LYKOURAS, M.D., EVANGELOS GARELIS, M.D., ELEFTHERIA VARSOU, M.D., AND COSTAS N. STEFANIS, M.D.

Intense exercise for one hour induced a significant increase in plasma cyclic adenosine monophosphate (cAMP) in 5 healthy volunteers. In 44 manic-depressive patients, cAMP levels correlated more strongly with mood ratings than with activity scores. The authors conclude that physical activity is one of the factors which contribute to changes of cAMP levels in affective illness.

SEVERAL GROUPS of investigators have reported that cyclic 3,5-adenosine monophosphate (cAMP) concentrations in urine are increased in mania and decreased in depression (1–4). In contrast to these reports, Cramer and associates (5) showed that depressed patients had significantly higher basal levels of cAMP in the cerebrospinal fluid (CSF) than either manic patients or controls. Others found no change in the cAMP content of urine (6, 7) or CSF (8) in affective disorders.

To our knowledge, no studies on the concentration of cAMP in the plasma of manic-depressive patients have been published. This is rather surprising, because plasma offers several advantages over urine for the investigation of the cAMP system in psychiatric patients. In contrast to urine, in which more than 50% of the concentration may be of renal origin, the plasma pool of cAMP derives from many organs, including the central nervous system (9).

Using this approach, we found that in the drug-free state plasma cAMP levels are much higher in mania and much lower in depression than normal (10).

It has been suggested (11), and denied (1, 4, 12), that changes in urinary cAMP in affective illness may simply reflect the increased or decreased psychomotor activity of such patients. The aim of the present study was to investigate the possibility that physical activity may affect plasma concentration of cAMP.

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METHOD

Four blood samples were taken from 5 healthy volunteers (3 men and 2 women) at the end of one-hour intervals, during which subjects successively engaged in usual activity, bed rest, intense exercise, and a second period of normal activity. Exercise consisted of continuously ascending and descending a staircase. The relative effect of disturbed mood and abnormal psychomotor activity on plasma cAMP in affective disorders was studied in a group of 44 unmedicated patients (28 depressed, 9 manic, and 7 normothymic). Rating of activity in these patients was based on a 9-point scale (−4 through +4). A score of zero indicated normal activity; −1 to −4, progressively decreased activity; and +1 to +4, corresponding degrees of increased activity.

A similar scale was used to rate depressed (−1 to −4), normal (0) or elevated (+1 to +4) mood. Both ratings were made at the time of blood sampling by two independent observers. For statistical analysis, the average of the two scores was used. Plasma cAMP was measured by a protein binding assay (13).

All subjects had consented to the study after being informed of its nature. All biochemical determinations were carried out blindly, i.e., on coded samples and without prior knowledge of diagnosis, ratings of activity, or other clinical information.

RESULTS

In the group of volunteers, bed rest did not affect cAMP appreciably. Conversely, exercise induced a highly significant increase ($p<.001$) in plasma concentrations (see figure 1), which returned to nearly baseline levels in 1 hour. Exercise proved to be markedly strenuous for our subjects, as shown by the approximate doubling of heart and breathing rates, which were recorded at the end of each 1-hour period (figure 1).

Results from manic and depressed patients indicate that both psychomotor activity and mood may influence cAMP levels in the plasma, as indicated by the significant correlation of each of these factors with cAMP ($r=.70$ and $r=.95$, respectively). The correlation coefficient, however, is much higher for the mood factor than for exercise (figure 2).

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FIGURE 1
Effect of Exercise on Mean Plasma cAMP, Pulse, and Breath Rate in 5 Healthy Subjects

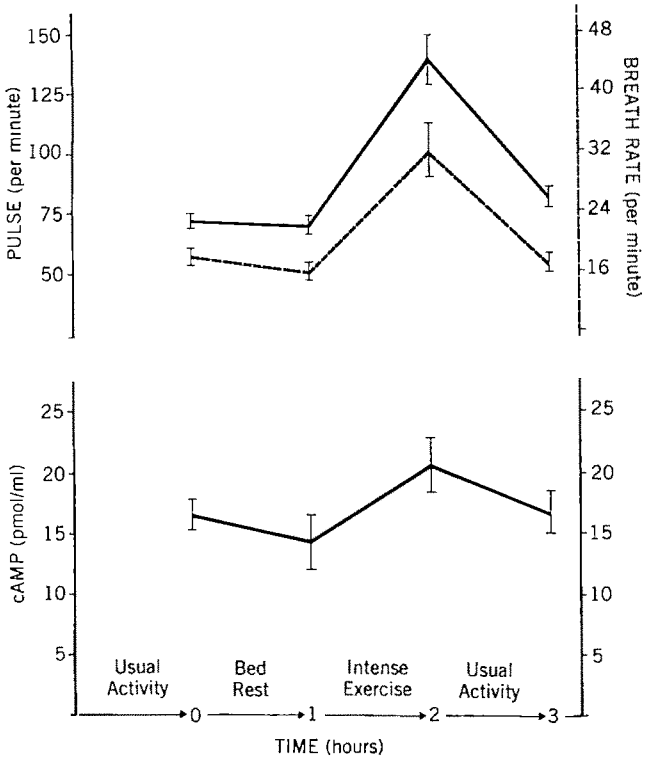
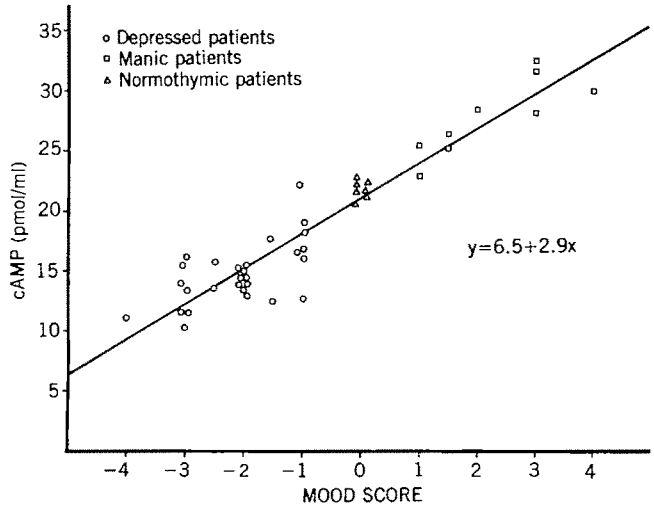


FIGURE 2
Relationship Between Plasma cAMP and Mood in Unmedicated Depressed (N=28), Manic (N=9), and Normothymic (N=7) Patients



When the 28 depressed patients were divided into subgroups on the basis of decreased, normal, or increased activity, no difference in cAMP concentrations among the 3 groups was found (table 1).

The manic (N=9) and normothymic (N=7) groups were too small to be further subdivided.

TABLE 1
Plasma cAMP Levels in Depressed Patients Showing Varying Degrees of Psychomotor Activity

Activity Level	Number of Patients	cAMP Level[a] (pmol/ml) Mean	SD
Normal	5	15.4	4.2
Decreased	17	14.4	2.1
Increased	6	16.1	2.4

[a]None of the differences is significant.

DISCUSSION

Drug-free manic-depressive patients have consistently abnormal levels of cAMP in plasma (10). Affective disturbance seems to be the primary factor associated with these changes, as suggested by the high correlation between mood and cAMP. The similar cAMP concentrations in groups of depressed patients with varying degrees of activity also point to this conclusion.

The assumption that psychomotor activity does not have a decisive influence on plasma cAMP in psychiatric patients is further strengthened by the finding of no correlation between activity and cAMP in a group of 10 drug-free chronic schizophrenics (14). These patients had normal concentrations of plasma cAMP. It is interesting in this context that in 2 patients with periodic catatonia, extreme changes in psychomotor activity (stupor or catatonic excitement) did not alter cAMP excretion in urine (15). Similarly, the high activity of hyperkinetic children does not affect urinary excretion of cAMP (16), and induced exercise did not raise cAMP concentrations in the CSF of depressed patients (5) or in the urine of healthy volunteers (4).

In contrast to the above evidence, other findings indicate that motor activity does have some effect on cAMP levels. Thus, prolonged walking leads to an increase in urinary cAMP (11). Intense exercise significantly raises concentrations of cAMP in the plasma of normal subjects, and there was a significant correlation between ratings of motor activity and cAMP concentrations in the plasma of our patients.

However, psychomotor activity and mood are generally interrelated in manic-depressive illness. Other factors, such as anxiety, may also influence cAMP concentrations in such patients. Therefore, the exact association between cAMP and affective disorders warrants more detailed exploration.

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The 24-Hour Dexamethasone Suppression Test in a Clinical Setting: Relationship to Diagnosis, Symptoms, and Response to Treatment

BY WALTER ARMIN BROWN, M.D., ROBERT JOHNSTON, B.M., B.CH., M.P.H., AND DEMMIE MAYFIELD, M.D.

Of 54 male psychiatric patients undergoing dexamethasone suppression tests in a clinical setting, 40% of those with a major depressive disorder showed escape from suppression over the 24 hours after dexamethasone administration, while all of the patients with schizophrenia, neurosis, alcoholism, and drug abuse showed normal pituitary-adrenal suppression. Only 10% of the depressed patients showed resistance to suppression 8 hours after dexamethasone administration. There was no difference between depressed patients who did and did not show escape from suppression in type of previous episodes, family history, symptoms, or medication. However, those who showed escape tended to respond better to treatment and to be rated as having a more severe depression. The theoretical and clinical implications of these findings are not yet clear.

OVER THE PAST 10 years there has been considerable interest in the relationship between pituitary-adrenal activity and affective disorders. Studies involving urinary excretion of 17-hydroxycorticosteroids, urinary free cortisol, and plasma cortisol concentration show increased cortisol secretion in some depressed patients (1, 2). While it was initially thought that this hypersecretion of cortisol might be related to the turmoil or agitation accompanying depression, careful clinical assessment has shown that the hypersecretion of cortisol cannot be accounted for by a nonspecific stress response (3). A substantial proportion of depressed patients in some studies (2, 4) have shown resistance to pituitary-adrenal suppression with dexamethasone, while other studies (2, 5) have shown normal pituitary-adrenal suppression in depressed patients. These contradictory findings have been based on the overnight dexamethasone suppression test. Carroll and associates (3) have reported that while depressed patients usually show the expected suppression of early morning plasma cortisol after midnight administration of dexamethasone, a substantial subgroup show escape from suppression later in the day, unlike healthy subjects and patients with other psychiatric diagnoses, who maintain suppression for 24 hours.

Studies of urinary steroid excretion, plasma cortisol concentration, and dexamethasone suppression are consistent in showing what appears to be disinhibition of the pituitary-adrenal system in some depressed patients. The theoretical and clinical implications of this phenomenon are not clear. Consistent clinical differences between depressed patients who do and do not show pituitary-adrenal disinhibition have not been identified, and the relationship between pituitary-adrenal disinhibition and response to treatment has not been studied. Some studies of serotonin and norepinephrine metabolism in depression, and particularly the intensive study of urinary 3-methoxy-4-hydroxyphenylglycol (MHPG) excretion, have suggested the existence of multiple "biological" subtypes of "endogenomorphic" depression (6). While no clear-cut clinical differences have been demonstrated between depressed patients with low and normal urinary MHPG, there are data suggesting that MHPG levels can predict response to treatment (6).

The dexamethasone suppression test (DST) may be another marker of a subtype of depressive disorder. This test is of particular interest because its procedural simplicity and lack of risk and discomfort to the patient make it feasible as a diagnostic tool. The present study is an attempt to replicate the work of Carroll and associates (3) with the 24-hour DST in a natural clinical setting. This study assesses the diagnostic sensitivity and specificity of the dexamethasone suppression test, the differences between depressed patients who

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do and do not show abnormal dexamethasone responses, and the role of psychological arousal in responses to dexamethasone.

METHOD

In August 1977 the DST was made available as a laboratory procedure to the four psychiatrists working on an inpatient psychiatric unit of a university-affiliated Veterans Administration hospital. These psychiatrists were aware of the possible usefulness of the DST in identifying patients with a depressive disorder and were encouraged to request it for all patients with clear depressive symptoms and for other patients at their discretion. These clinicians requested the DST for almost all of the patients who presented with a major depressive disorder. Patients taking phenytoin or barbiturates were excluded because of the known effects of these drugs on dexamethasone metabolism (7, 8).

The patients received 2 mg of dexamethasone orally at midnight after a blood sample (10 cc) had been drawn by venipuncture. Subsequent blood samples were drawn at 8:00 a.m., 4:00 p.m., and 11:30 p.m. the next day. In 25 patients ratings of tension-anxiety and depression-withdrawal (on a scale of 1–4) were made at the time of each blood drawing. Serum was separated and frozen until assayed. Cortisol was assayed by a solid phase radioimmunoassay, with all samples from the same subject measured in duplicate in the same assay. The interassay coefficient of variation for this method in our laboratory is 7.5% and the intraassay coefficient of variation, 6%.

A conservative criterion for normal pituitary-adrenal suppression after administration of 2 mg of dexamethasone is a reduction in serum cortisol concentration to less than 6 μg/100 ml over the 24 hours following dexamethasone administration (3, 9). We used this criterion to identify patients who did and did not show normal pituitary-adrenal suppression.

Two Board-certified psychiatrists independently diagnosed each patient who underwent a DST using Research Diagnostic Criteria (10) and assessed response to treatment (none, poor, fair, good) four weeks after admission on the basis of hospital records. These psychiatrists were not involved in the care of the patients and did not know the results of the DST. In cases in which there was disagreement on diagnosis, the final diagnosis was arrived at by consensus after reevaluation and discussion. Patients who received a diagnosis of major depressive disorder underwent further scrutiny to determine the presence or absence of previous episodes of depression, mania, or other psychiatric illness. In addition, patients with a diagnosis of major depressive disorder were independently assigned to a severe or not severe category by the two psychiatrist raters. The admitting clinician noted the presence or absence of specific psychiatric symptoms on a standard form, and these data relating to specific symptoms were obtained from the patients' charts.

RESULTS

Patients and Diagnoses

Between August 1977 and April 1978 DSTs were carried out on 54 (24%) of the patients admitted to the psychiatric unit. They were all men and ranged in ages from 21 to 82, with a mean age of 47. With rare exceptions, patients were taking one or more psychotropic drugs at the time of the DST, including tricyclic antidepressants, benzodiazepines, and neuroleptics. Five patients had one or more major physical illnesses at the time of the DST, including cardiac failure, unstable diabetes, pulmonary disease with fever, and brain tumor. The remaining 49 patients were free of acute or major physical illness and, with rare exceptions, were taking no medication other than psychotropic drugs.

The raters agreed on the diagnosis in 86% of the cases; the remaining diagnoses were arrived at by consensus after discussion. Twenty-two patients were given a diagnosis of a major depressive disorder (endogenomorphic depression); 9, schizophrenia; 10, neuroses (mixed); 11, alcoholism; and 2, drug abuse. None of the depressed patients were bipolar (history of mania or hypomania), but 14 had had previous episodes of depression. In 89% of the cases there was agreement between the raters' diagnosis using Research Diagnostic Criteria and the diagnosis of the treating physician using *DSM-II* guidelines (11).

Dexamethasone Suppression

Forty-one patients showed clear-cut pituitary-adrenal suppression with dexamethasone (table 1). In these patients all of the samples taken after dexamethasone administration had cortisol concentrations of less than 5 μg/100 ml, with the great majority of patients (88%) having serum cortisol concentrations of less than 4 μg/100 ml in all samples. Thirteen patients had serum cortisol concentration of 6 or more μg/100 ml in one or more of the postdexamethasone samples with all but one of these nonsuppressors having at least one cortisol concentration of more than 8.0 μg/100 ml. Thus there was ready differentiation of suppressors and nonsuppressors (see table 1).

Four of the 13 nonsuppressors were excluded from further data analyses because they had major physical illnesses that have been associated with pituitary-adrenal activation and, in some instances, resistance to dexamethasone suppression (3, 12, 13). Of these 4 patients, 1 had a diagnosis of depression and brittle, insulin-dependent diabetes; 2 patients, one with a diagnosis of alcoholism and the other with neurosis, had multiple medical problems including cardiac failure; and 1 had a diagnosis of schizophrenia and multiple medical problems including pulmonary disease with fever. One patient with a diagnosis of depression was found in the course of his hospitalization to have a glioblastoma. He was also excluded from further data analyses because of the possible effects of this lesion on pituitary-adrenal activity.

TABLE 1
Mean Serum Cortisol Concentration in Subjects Showing Suppression and Escape from Suppression with Dexamethasone

	Serum Cortisol Concentration (μg/100 ml)			
	Suppressors (N=41)		Nonsuppressors (N=13)	
Item	Mean	SEM	Mean	SEM
Predexamethasone				
11:30 p.m.	7.4	1.0	12.7	2.1
Postdexamethasone				
8:00 a.m.	2.2	0.1	6.1	1.3
4:00 p.m.	2.1	0.2	9.4	1.6
11:30 p.m.	1.9	0.1	13.7	3.4

TABLE 2
Diagnoses of Suppressors and Nonsuppressors[a]

Diagnosis	Suppressors (N=41)	Nonsuppressors (N=8)
Major depressive disorder	12	8
Schizophrenia	8	0
Neuroses	9	0
Alcoholism	10	0
Drug abuse	2	0

[a]The difference between the groups was significant (χ^2=13.86, p<.005).

TABLE 3
Mean Serum Cortisol Concentration in Depressed and Medically Ill Patients Showing Escape from Suppression

	Serum Cortisol Concentration (μg/100 ml)			
	Depressed (N=8)		Medically Ill (N=5)	
Item	Mean	SEM	Mean	SEM
Predexamethasone				
11:30 p.m.	14.2	2.4	10.4	4.0
Postdexamethasone				
8:00 a.m.	4.0	0.9	9.3	2.5
4:00 p.m.	9.4	2.0	9.4	2.8
11:30 p.m.	16.1	5.3	9.9	2.4

The 8 nonsuppressors without evidence of major physical illness all had a diagnosis of major depressive disorder. Thus 40% of the physically healthy depressed patients in our sample had a relative lack of suppression after dexamethasone administration, while none of those with schizophrenia, neurosis, alcoholism, or drug abuse showed a lack of suppression (see table 2).

The pattern of serum cortisol response to dexamethasone in these depressed patients is particularly noteworthy. Of the 8 depressed patients who were nonsuppressors, only 2 showed resistance to suppression (serum cortisol concentrations of 6 or more μg/100 ml) at 8:00 a.m., 5 at 4:00 p.m., and all 8 by 11:30 p.m. (table 3). This pattern of response suggests initial suppression after dexamethasone administration followed by escape from suppression later in the 24-hour period. Only 10% of the depressed patients showed resistance to suppression in the 8:00 a.m. sample, while 40% of them showed escape from suppression in the 11:30 p.m. sample. Thus the serum cortisol concentration 24 hours after dexamethasone administration was the most sensitive indicator of the presence of a depressive disorder and as specific as the cortisol concentration at any other sampling point or combination of sampling points. This pattern of gradual escape from suppression in the depressed patients is in contrast to the pattern of cortisol response in the 5 patients with major physical illness (table 3). These patients tended to show high cortisol concentrations throughout the 24 hours after dexamethasone administration rather than the gradually increasing cortisol concentrations seen in the depressed patients.

Midnight Cortisol Concentrations

The period of minimal cortisol secretion lies between the late afternoon and early morning hours. While the available normative data indicate considerable variation in midnight serum cortisol concentration, a reasonable upper limit for normal serum cortisol concentration at midnight is 6 μg/100 ml (3). Using this criterion, 23 of the 49 patients (excluding those with major physical illness) had elevated midnight cortisol concentrations (6.3–30 μg/100 ml) before dexamethasone administration. All of the patients who showed escape from suppression with dexamethasone had elevated midnight cortisol concentrations (more than 6 μg/100 ml). Sixty percent of the patients with diagnoses of major depressive disorder had elevated predexamethasone midnight cortisol concentrations, while 38% of the patients with other diagnoses had elevated predexamethasone midnight cortisol concentrations. Thus midnight cortisol concentration appears to be a reasonably sensitive indicator of depressive illness but does not reliably discriminate depressed patients from those with other diagnoses.

Psychological State and Serum Cortisol Concentrations

Since the pituitary-adrenal system is so clearly responsive to transient increases in psychological arousal, we attempted to assess the degree of tension-anxiety and depression-withdrawal at the time of each blood sampling. Ratings were made on each of these variables on a scale of 1 to 4 (1=none, 2=mild, 3=moderate, 4=severe) in 25 patients. The raters included a medical technologist, psychiatric residents, and psychology interns. Psychological state ratings were made at each blood sampling point without knowledge of the previous rating.

There was a high degree of consistency among the ratings for each patient over the 24-hour period, with most patients receiving ratings of none to mild or moderate to severe for each variable over the 24-hour period. Within this sample of patients, cortisol concentration ranged from 1.0 to 18.2 μg/100 ml and psychological state ratings covered the range of the scale. There

was no relationship between predexamethasone cortisol concentration or postdexamethasone cortisol concentration and ratings of psychological state at the time of blood sampling.

Depressed Patients With and Without Escape from Suppression

Since the above analyses indicated that escape from suppression was selectively associated with depressive illness, we addressed the question of whether or not there were clinical differences between the depressed patients who did (N=8) and did not (N=12) show escape from suppression. These 2 groups did not differ significantly in mean age and weight. Six of the nonsuppressors and 8 of the 12 suppressors were taking tricyclic antidepressants at the time of the DST. The rest of the patients in each group were either taking no medication or were on neuroleptics or lithium. None of the depressed patients had a history of mania, but 6 of the nonsuppressors and 8 of the suppressors had had previous episodes of depressive illness. Thus the majority of the patients in each group had unipolar depression. A minority of the patients in each group reported a family history of psychiatric disorder.

In an attempt to evaluate the presence or absence of specific symptoms we used data that the admitting physician entered on a standard form. Most of the patients in each group were noted to have sleep disturbance, fatigue, anorexia, social withdrawal, and feelings of anxiety and depression. About 50% of the patients in each group were noted to have crying spells and suicidal ideation. Two patients who showed normal suppression and 1 who showed escape were noted to have delusions. Thus these 2 groups of depressed patients could not be differentiated on the basis of specific depressive symptoms.

There was agreement on the degree of global severity (severe versus not severe) for 87% of the depressed patients. The few cases in which there was initial disagreement were categorized by consensus after discussion. Fifty percent of the patients who showed escape from suppression were categorized as having a severe depression, compared with only 11% of those who showed normal suppression. All but 2 of the depressed patients were treated with tricyclic antidepressants. Independent global assessments of response to treatment (none, poor, fair, good) showed complete agreement for 50% of the depressed patients and approximate agreement (none-poor versus fair-good) for 88%. Fifty-seven percent of the patients who showed escape from suppression had a fair to good response to treatment compared with 20% of those who showed normal suppression.

DISCUSSION

These data are consistent with those reported by Carroll and associates (3). Like these investigators, we found that 40% of our depressed patients showed escape from dexamethasone suppression, that there was gradual escape over the 24 hours after dexamethasone administration, that the 11:30 p.m. postdexamethasone sample was the single most sensitive and specific indicator of depressive disorder, and that patients with other diagnoses showed normal suppression. We also found, as did Carroll and associates (3), that predexamethasone midnight cortisol concentration was elevated in patients who showed escape from dexamethasone suppression but that this measure of pituitary-adrenal activity did not reliably differentiate depressed from nondepressed patients. The consistency of our findings with those of Carroll and associates is particularly noteworthy in view of the fact that our study was carried out in a different clinical setting with a different patient population and used a different, albeit comparable, methodology for serum cortisol assay.

While our study certainly provides further evidence that escape from suppression after dexamethasone administration in the absence of physical illness is selectively associated with depressive disorder, several cautionary notes must be sounded. While the general classes of psychotropic drugs taken by our patients have been shown to have no effect on circadian patterns of pituitary-adrenal activity (14), these drugs have not been systematically studied for their effects on dexamethasone metabolism and pituitary-adrenal response to dexamethasone. The fact that the depressed patients who showed escape from suppression were taking the same drugs (tricyclic antidepressants) as those who did not show escape would certainly suggest that these drugs in themselves do not account for the abnormal responses to dexamethasone. Carroll and associates' data (2, 3) also indicated that tricyclic antidepressants do not affect the dexamethasone suppression test independently of their effect on clinical state. In addition, approximately half of Carroll and associates' subjects were drug-free, and in these patients escape from suppression was also selectively associated with depressive illness. In spite of these indications that psychotropic drugs are unlikely to account for the selective association between depressive illness and dexamethasone response, systematic study of this issue is important, particularly in light of the potential usefulness of the DST as a diagnostic tool.

Another issue that must be addressed is whether the different responses to dexamethasone could be due to differences in the absorption or metabolism of dexamethasone. While we have no data to definitively rule in or rule out this possibility, the fact that all of the patients who showed escape from suppression also had elevated predexamethasone midnight cortisol suggests that their response to dexamethasone represents a difference in the pituitary-adrenal system rather than aberrant disposition of dexamethasone. Parenteral administration of dexamethasone would help to clarify this issue.

Our data also point to the importance of attending to concurrent physical illness in interpreting responses to

dexamethasone. A number of specific physical illnesses other than Cushing's disease have been shown to be associated with resistance to suppression (3, 13). These data, along with ours, underscore the importance of assessing physical status in using the DST for research or clinical purposes.

The observation that escape from dexamethasone suppression occurs selectively in a subgroup of depressed patients has now been made by two groups of investigators. The clinical and pathophysiological implications of this observation are not yet clear. Using some rather gross and indirect estimates of symptom presentation, we were unable to differentiate the depressed patients who did and did not show escape from suppression with respect to specific depressive symptoms. Although our finding of no differences in symptom pattern between these groups is consistent with the findings of previous studies (4, 15), we feel that the definitive answer on this issue has not yet been reached. Continued detailed clinical assessment with particular attention to sleep patterns, diurnal variation, and other core depressive symptoms is in order.

Our data suggesting that escape from suppression may be related in part to the severity of depression is consistent with the findings of Stokes and associates (4) and Carroll and associates (16), who reported an association between ratings of severity of depression and resistance to suppression using the overnight DST. In a later study, however, Carroll and associates (15) found no relationship between severity of depression and resistance to dexamethasone suppression. These data in the aggregate suggest that global severity of depression alone does not account for the differential responses to dexamethasone among depressed patients.

Our data showed a trend for an association between a good response to tricyclic treatment and escape from suppression. Because several tricyclics were used, we were not able to systematically assess the relationship between response to a specific tricyclic and dexamethasone suppression response. The data suggesting that escape from suppression predicts a good response to treatment must be viewed in light of the tendency for the "escape" patients to have a different depressive baseline (more severe) than those who showed normal suppression. While this difference in baseline severity might account for their apparently greater improvement, our findings point to treatment response as a variable worthy of continued study.

Recent data suggesting the existence of norepinephrine- and serotonin-deficient subtypes of depression have been shown to have important treatment implications (6, 17, 18). Concomitant assessment of pituitary-adrenal activity and neurotransmitter metabolites in depressed patients would provide an important increment in our understanding of the role of these undoubtedly interrelated neurophysiological phenomena in depressive illness.

The possibility that the dexamethasone suppression test is a marker of a clinically important subtype of depression merits continued exploration. Because of its procedural simplicity and the apparent lack of interference by psychotropic drugs, the DST could be uniquely useful as a laboratory aid in the diagnosis and treatment of depression.

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Facial Muscle Electromyography in Depressed and Nondepressed Hospitalized Subjects: A Partial Replication

BY DONALD OLIVEAU, M.D., AND RAGON WILLMUTH, M.D.

Imagining happy events, sad events, and the events of a typical day led to measurable electromyographic (EMG) changes in the corrugator muscle of the face in both depressed and nondepressed subjects. The depressed and nondepressed subjects could not be differentiated on the basis of muscle activity, whether depression was defined by a self-rating scale, an adjective checklist, or research criteria. Previous investigators have observed differences in the facial EMG patterns of depressed and nondepressed subjects who were not hospitalized; the authors suggest that such changes are a consequence of a general psychological stress state rather than of depression specifically.

RECENT REPORTS by Schwartz and associates (1, 2) suggest that a noninvasive electromyographic (EMG) technique may be useful in distinguishing between depressed and nondepressed mood states. These authors found that EMG patterns recorded from facial muscles while subjects imagined a variety of affect-laden events (happiness, sadness, anger, the events of a typical day) could be reliably differentiated and that depressed subjects responded differently than nondepressed subjects. Such a technique would not only provide an objective measure of depression but would be a potential means of assessing clinical progress and the effectiveness of different treatments. Schwartz and associates recruited their subjects from clinic patients considered appropriate for a trial of antidepressant medication and from volunteers who responded to a newspaper ad. We were interested in determining whether their observations could be replicated in hospitalized subjects.

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METHOD

We measured the EMG of the corrugator muscle and aside from minor technical and procedural differences (description available on request), the major difference between our investigation and the investigations of Schwartz and associates was the choice of subjects. Each of our subjects was hospitalized on an inpatient psychiatric unit of a general hospital. Patients were asked to participate in the experiment only if they suffered from neither psychosis nor organic brain syndrome. The experiment was explained to subjects and written informed consent was obtained. All patients who agreed to participate in the experiment were interviewed to determine whether they met research criteria for depression (3). The patients who did not meet research criteria for depression (the nondepressed group) suffered from a variety of psychiatric problems severe enough to require hospitalization. Each participant was asked to complete the Zung Self-Rating Depression Scale (SDS) (4) and a depression adjective checklist (DACL) (5) on the day when the electromyogram was taken. There were 40 subjects (21 women and 19 men) participating in the experiment.

The single pair of electrodes was placed over the right corrugator region just above the medial aspect of the eyebrow. The outline of the procedure was reviewed with each subject and a five-minute baseline recording was made during which subjects were asked to rest and "not think about anything in particular." Subjects were then asked to recall and "picture in their mind" as vividly as they could some sad event, some happy event, or the events of a typical day in their life. After each three-minute period of recollection, the subjects were asked to relax for two minutes. The subject's experience during imagery states was either sad event-typical day-happy event or happy event-typical day-sad event, and each order was used with half of the subjects in both the depressed and nondepressed groups.

The integrated EMG activity during the baseline period was compared to the activity during each of the imagery periods by reducing each to the average number of millimeters per three minutes recorded on the integrator.

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TABLE 1
Median Change in Corrugator EMG from Resting Baseline During Imagery Conditions (millimeters per 3 minutes)

	Grouping						
	Research Criteria		DACL		SDS		
Condition	Depressed	Nondepressed	Depressed	Nondepressed	Depressed	Nondepressed	Total
Happy event	− .2	+ .2	+ .1	− .4	− .3	+ .1	− .2
Sad event	+ .4	+2.4	+9.0	+1.1	+ .7	+2.0	+1.5
Typical day	+1.2	+1.2	+2.2	0	+1.2	+1.2	+1.2

TABLE 2
Significance of Comparisons of EMG Findings Among the Three Imagery Conditions[a]

	Grouping											
	Research Criteria			DACL			SDS			SDS (Swartz)		
Sample	Happy	Sad	Typical	Happy	Sad	Typical	Happy	Sad	Typical	Happy	Sad	Typical
Total sample												
Baseline	n.s.	.001	.02	n.s.	.001	.02	n.s.	.001	.02	.05	.06	n.s.
Sad	.003			.0005			.001			.01		
Typical day	.04	n.s.		.04	n.s.		.04	n.s.		.01	.02	
Depressed subjects												
Baseline	n.s.	.05	.05	n.s.	.01	.01	n.s.	.05	.05	n.s.	.05	.07
Sad	.03			.004			.01			.05		
Typical day	.01	n.s.		.02	n.s.		n.s.	n.s.		.05	n.s.	
Nondepressed subjects												
Baseline	n.s.	.01	n.s.	n.s.	.07	n.s.	n.s.	.01	.09	.02	n.s.	n.s.
Sad	.04			.03			.05			.01		
Typical day	n.s.	n.s.		n.s.	n.s.		n.s.	n.s.		.02	.02	
Depressed versus nondepressed subjects												
Baseline	n.s.	n.s.	n.s.	n.s.	n.s.	.09	n.s.	n.s.	n.s.	.09	n.s.	.08
Sad	n.s.			n.s.			n.s.					
Typical day	n.s.	n.s.		n.s.	n.s.		n.s.	n.s.				

[a]Values (p) are based on either changes from baseline or comparisons between imagery conditions for the total sample (N=40) and for the depressed (N=20) and nondepressed (N=20) groups separately and in comparison.

RESULTS

Table 1 shows the median changes from baseline of corrugator EMG activity during imagery conditions. Table 2 is a summary of the several comparisons between the various baseline and imagery states and between depressed and nondepressed subjects. The p values are derived from Wilcoxon matched-pairs signed-ranks tests for comparisons of the imagery states with baseline measures and from Mann-Whitney U tests for comparisons between groups of subjects (6) (all tests were two-tailed). The corresponding p values from Schwartz and associates (1) are also given.

The SDS mean scores for the depressed group and nondepressed groups were 59.8 and 38.7, respectively, which correspond closely to the means reported by Schwartz and associates (1) (58 and 37, respectively). This correspondence suggests that, at least in terms of depression, our subjects were similar to those tested by Schwartz and associates (1). The DACL mean scores for the depressed and nondepressed groups were 17.4 and 5.5, respectively.

The grouping of subjects in depressed and nondepressed categories was initially done by research criteria for depression (3). Two additional groupings according to scores on the SDS and the DACL were derived by dividing subjects according to whether they scored above or below the median score on each of these scales. The SDS scale was also used by Schwartz and associates. When the subjects were regrouped according to the SDS criteria for depression, a balanced order of presentation remained equally distributed in the two groups; however, when the subjects were classified according to the DACL criteria, a disproportionate number (N=13) of subjects in the depressed group had been given the imagery states in the sad event-typical day-happy event order. Since the amount of EMG activity observed during the typical day imagery state was greater ($p<.05$, Mann-Whitney U test, two-tailed) when the sad imagery state was presented first, the data for the DACL grouping of subjects are less reliable than the research criteria or SDS groupings.

When subjects imagined happy events, we were unable to detect significant changes from baseline EMG activity. Similarly, Schwartz and associates observed no significant changes from baseline activity under happy imagery conditions for their group of depressed subjects; however, they observed significant changes in nondepressed subjects and the total group and

found a significant difference between the depressed and nondepressed groups.

When subjects were asked to imagine sad events, we found significant changes in the total group, the nondepressed groups, and in the depressed groups; Schwartz and associates found significant changes in the total group and in the depressed group but not in the nondepressed group.

With the exceptions of the nondepressed groups (defined by research criteria and the DACL), we found significant changes when subjects imagined the events of a typical day; Schwartz and associates found similar changes only in the depressed group.

We found one significant difference between the depressed and nondepressed groups (for typical day imagery, depressed versus nondepressed as defined by the DACL), but this difference may have been influenced by the lack of a balanced order of presentation of the imagery states.

DISCUSSION

In most of the comparisons our total sample of subjects resembles the group of depressed subjects reported by Schwartz and associates. All of the significant changes from baseline activity that we observed were in the direction of increased EMG activity during imagery states, whereas Schwartz and associates reported significant decreases in activity for their nondepressed group during the happy event and typical day imagery states.

Our results support the work of Schwartz and associates in that differences were found between happy and sad imagery states. However, we were unable to detect differences between the sad imagery state and the typical day imagery state.

There are several factors that may have contributed to the lack of correspondence between our results and those of Schwartz and associates (1, 2). We instructed subjects to imagine the scenes "as vividly as you can," whereas Schwartz and associates (2) asked subjects to "vividly experience," and they noted (2) that different effects are observed when subjects are asked to "feel" than when they are asked to "think." Although we think that our instructions more closely approximate the Schwartz and associates (1) "vividly experience" and "feel" conditions than their "think" condition, the differences in instruction may have subtly influenced the data. The nearly equal mean scores on the SDS between our groups of subjects and theirs suggest that the degree of depression in the two studies was similar in both the nondepressed and depressed groups.

In a study of 5 subjects, Teasdale and Bancroft (7) observed a decrease in corrugator EMG activity from baseline levels during a happy imagery condition in 2 of their subjects but either an increase or virtually no change in the other 3. Since their study was designed for somewhat different purposes than either our study or the study of Schwartz and associates, it is difficult to assess how much their findings might contribute to understanding the differences between our observations and those of Schwartz and associates (1, 2). Nevertheless, their study does suggest that the EMG response of the corrugator muscle is variable under happy imagery conditions.

While our observations parallel those of Schwartz and associates (1) quite closely when our subjects were divided according to DACL scores, these observations cannot be considered as reliable as either the research criteria or SDS grouping observations. The "spillover" effect of presenting the sad imagery condition first to a disproportionate number in one group may have influenced the typical day observations in the DACL groupings.

It is also important to note that we made EMG recordings on only one muscle, while Schwartz and associates (1, 2) made recordings from several muscles. We chose the corrugator primarily because of the bidirectional differences between sad and happy imagery conditions they reported, which suggested to us that this muscle would be the most useful one as a clinical tool. Schwartz and associates (2) suggest that the zygomatic muscle may be more responsive to the happy imagery condition and the corrugator to several imagery conditions in addition to sadness. Thus it is possible that the general stress of the conditions for which our subjects were hospitalized tended to wash out the differences attributable to depression alone.

The results of our experiment support the observations that when subjects are asked to imagine affect-laden events reliable changes in facial muscle EMG can be observed, and that different affects evoke different patterns of change. Our results do not corroborate previous reports that depressed and nondepressed subjects respond differently; this may suggest that subjects undergoing psychological stress respond similarly to one another, i.e., differences between depressed and nondepressed subjects may be a function of psychological stress rather than a specific function of depression.

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Diagnosis and Lithium Treatment of Affective Disorder in the Retarded: Five Case Studies

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The authors report on five institutionalized retarded patients who had symptoms strongly suggestive of manic-depressive illness and who showed significant symptom reduction in a single-blind placebo-controlled three-year trial of lithium. The number of illness episodes was reduced, and symptoms decreased in all major illness dimensions. These findings indicate that major affective disorder can be both diagnosed and treated in individuals with primary mental retardation.

SYMPTOMS OF manic-depressive disorder in the retarded were observed as early as 1883 (1). Others have continued to note the coexistence of symptoms of major affective disorder and mental retardation (2–10).

Reid (11) found that the incidence of manic-depressive psychosis in a hospitalized population of mentally retarded persons in Scotland was 44 per 1,000 (as compared to an incidence of 33.2 per 1,000 for schizophrenic psychosis). In that study, of the 21 patients with manic-depressive psychosis 11 were depressed, 7 were bipolar, and 3 were manic. The suggested treatment approaches were tricyclic antidepressants, major tranquilizers, and ECT. Naylor and associates (12) used lithium to treat 14 retarded patients in a double-blind crossover study. Six of these patients had bipolar illness, 3 had unipolar mania, 3 had unipolar depression, and 2 were difficult to classify. The number of weeks of illness was significantly reduced on lithium therapy compared to placebo. The number of illness episodes was fewer but not significantly so.

The present study involved prolonged and repeated observations of 5 retarded patients who had symptoms of manic-depressive illness. Diagnosis was established by the most recent criteria (13). Lithium carbonate was compared with placebo over a 3-year period of symptom observation and recording. Factors that might have worked against a successful demonstration of drug efficacy included the difficulty of establishing a diagnosis in the retarded patient and the complexities involved when symptoms are rated and recorded by different personnel, at different times, and over a long period. However, we thought that any drug effect would be apparent if the diagnosis were correct and the treatment specific.

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METHOD

From a population of 1,300 retarded residents of a state school, one of us (T.M.R.) consulted on 457 residents referred for behavior disorder. Of these, 5 had symptoms suggestive of a major affective disorder of the unipolar or bipolar type (13). A systematic observation and recording of diagnostic symptoms was then begun. Ninety days after symptom observation was initiated, patients were started on a 1-year therapeutic trial of lithium carbonate. At the end of the year, lithium was stopped and placebo substituted during a trial period of at least 90 days. Placebo was stopped and lithium restarted when obvious manic symptoms reappeared. The second lithium treatment period was 1.5 years. Symptoms were systematically observed and recorded throughout the 3 years of the study. The experimental design was known only by one of us (T.M.R.).

Behavioral change was assessed by a list of symptoms or behaviors in five dimensions that correspond to major areas of disturbance as described in proposed *DSM-III* criteria for manic-depressive illness (13). The dimensions associated with mania were increased vocalization, motor activity, aggressiveness and sleeplessness, and decreased appetite lasting over a week. Those associated with depression were mutism, decreased motor activity, withdrawal, sleeplessness or excessive sleep, and decreased appetite lasting over a week (13).

The specific symptom patterns for each patient were selected by a consensus of direct-care staff raters, with the criterion that the symptoms could be reasonably and easily observed and recorded during an 8-hour shift yet still be concordant with the diagnostic criteria for manic-depressive illness. For example, a sign of increased motor activity in 1 patient was translated into an abnormal amount of pacing and flailing of arms on a particular 8-hour shift. The recorders were blind

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TABLE 1
Descriptive Data for Five Retarded Patients

Case	Sex	Age	Degree of Mental Retardation	Family History of Affective Disorder	Affective Disorder Diagnosis
1	F	24	Moderate	Positive	Bipolar
2	F	30	Mild	Possible	Unipolar mania
3	F	29	Moderate	Negative	Bipolar
4	M	28	Severe	Possible	Bipolar
5	F	33	Moderate	Positive	Bipolar

TABLE 2
Ninety-Day Target Symptom Averages Recorded During Four Conditions

	Condition[a]			
Patient	Baseline	Lithium	Placebo	Lithium Retreatment
1	464	76	573	45
2	248	22	196	34
3	324	33	311	27
4	175	12	111	24
5	284	42	201	25
Mean	299.0	37.0	278.4	31.0
SD	107.3	24.6	179.3	8.7

[a]Analysis of variance yielded $F(3, 12)=16.8$, $p<.0001$. Hotelling's $T^2=611.5$, $p<.01$. Newman-Keuls comparisons indicated significant differences ($p<.01$) for all combinations of nontreatment (baseline and placebo) versus treatment (lithium and lithium treatment).

to drug changes from lithium to placebo and back to lithium again. For each treatment and placebo period, averages of each patient's symptoms were computed. All symptoms or behaviors were rated equally in the total symptom count.

If, after 90 days of placebo, symptoms had reappeared with increased frequency or severity, clinicians could discontinue placebo. Predrug (baseline), treatment, drug-free, and retreatment periods were compared statistically for the group.

Signed informed consent was obtained for the use of lithium carbonate from parents or legal guardians in all cases and, when possible, from the patients themselves. All medical safeguards recommended for the use of lithium carbonate, including screening for thyroid, cardiac, and renal disease, and regular serum lithium level monitoring, were observed (14). EEGs were done on all patients before and after lithium was started.

RESULTS

All 5 patients had mental retardation (full-scale IQs of 70 or less) of cultural-familial or unknown etiology. Two patients had positive family history for major affective disorder, and 2 had family members whose history was suggestive of major affective disorder. Table 1 summarizes the descriptive data.

There were significant differences in target symptoms between drug-free and lithium-treatment periods (see table 2). Each patient showed a significant decrease in symptoms when lithium was started, returned to a pretreatment level of symptoms when given placebo, and again improved significantly when lithium was reintroduced. The large standard deviations in the untreated periods reflect substantial intersubject differences in the clinical occurrence of symptoms. Standard deviations during treatment periods were small, indicating clinical similarity in regard to observed symptoms among the patients when their conditions improved. Symptoms in all observed dimensions (motor activity, degree of vocalization, degree of irritability, sleep, and appetite) improved to equal degrees in both lithium-treatment periods and relapsed to equal degrees on withdrawal of the drug. No one symptom or subgroup of symptoms appeared to respond better to the drug.

All patients had a significant recurrence of manic symptoms within 5.5 months of the placebo trial but none showed mania or depression during the lithium treatment (1 year) or retreatment (1.5 year) periods. This suggests a substantial reduction in illness episodes.

There were no complications or significant side-effects attributable to lithium therapy. EEGs were normal or nonfocally abnormal before lithium administration. Subsequent EEGs taken in patients 3, 4, and 5 at 1 week, 1 month, and 6 months after the administration of lithium were not significantly altered, and no observable seizures occurred. Two case vignettes are presented below to illustrate features of diagnosis and methods of clinical investigation of the group described.

CLINICAL VIGNETTES

Case 1. Ms. A, a 30-year-old woman, was placed in the state school when she was 11 years old. Her father was unknown, and no family history could be obtained from the paternal side. Her mother was known for promiscuity and antisocial behavior. One of her maternal uncles was an alcoholic and another had spent extended periods in a state mental institution.

Ms. A was the product of a normal gestation, labor, and delivery. Her developmental milestones were slowed, and she became a behavior problem when she started school. She had a short attention span and was disruptive. At the age of 11, when she was admitted to the state school, she had a full-scale IQ of 68 (performance=66, verbal=70).

She was always described as very active and talkative but was able to attend school and obtain employment. However, at age 25, she began to have episodes in which she became so disruptive, confused, and hyperactive that she would lose her job. During these episodes she intruded into the affairs of others and became threatening and assaultive. Seclusion appeared to calm her. A major tranquilizer in a moderate dosage was also helpful. The episodes subsided in 3–4 months and she was then able to return to her job. She had one episode a year from ages 25 to 28.

When she was 28, Ms. A was seen by a psychiatrist, who reviewed her history with the staff. A diagnosis of recurrent mania was considered. She was started on lithium in adequate doses and the major tranquilizer, which she had received for 4 years, was stopped. She had no episodes or outbursts during the year of lithium treatment. Then 16 weeks after placebo was started, she had an episode wherein she again became assaultive, hyperactive, sleepless, and overtalkative. Lithium was restarted and in 14 days she was much improved.

The patient has remained on lithium for an additional year and a half and has had no further manic episodes. She has remained steadily employed and was recently placed in a half-way house.

Case 2. Mr. B, a 28-year-old man, was placed in the state school by his parents when he was 11. His father and two paternal uncles were known to drink and gamble. There was a possibility that his mother had battered several of her children, three of whom (in addition to Mr. B) were said to be retarded. A maternal grandmother was reported to have committed suicide in a state hospital.

Mr. B was the fourth of 8 children. He was the product of a normal gestation, labor, and delivery. He had late developmental milestones and was said to be a withdrawn, fearful child. Social workers involved with the family suspected that he had been abused on a number of occasions, and there was some evidence that he had spent long periods locked in a closet during his early years. At the age of 11 he was placed in a state school for the mentally retarded by court order because he lacked schooling and adequate medical care.

On admission to the state school, Mr. B was fearful and withdrawn and uttered only stereotyped phrases and words. He threatened the examiner who tested him. Results of testing indicated he was severely impaired, with a full-scale IQ of 28. Organic impairment was not evident on psychological tests or neurologic examinations.

He soon became one of the school's severest behavior problems. He usually remained by himself but occasionally assaulted staff members and other residents. When Mr. B was 20, a staff member who had known him for several years observed that he had "good periods," lasting at times for 6 months to a year, during which he was easily manageable and generally withdrawn. However, every 6 months to a year he would, in the argot of the institution, "go on a bat." The staff noticed that he would pace more, sleep only 3–4 hours a night, mutter more, and assault other residents and staff. He also hit walls and attempted to break windows. Various doses of various major tranquilizers did not abort these episodes.

Mr. B had recently broken the rib of a staff member and three of his own fingers. He had also broken bones of his wrist and forearm, which necessitated his admission to a local general hospital. In the hospital, he kicked a wall and broke several bones in his foot and despite a heavy cast, was so agitated and violent that it was thought necessary to restrain all of his limbs. At the suggestion of the psychiatrist, he was started on haloperidol and restricted to a single room. He soon became manageable in the general hospital on this regimen, and after his cast was removed he was discharged.

After an uneventful period of five months during which he was generally withdrawn and not agitated, he began to pace and shout more and assaulted staff members and other residents. Despite increased doses of haloperidol, he smashed a window, severing two tendons at the wrist and breaking several small bones in his fingers. He was rehospitalized. The psychiatrist considered using the lithium protocol for apparent recurrent manic episodes. He was started on lithium, and haloperidol was stopped. His behavior improved, and within 2 weeks he was relatively calm and easily manageable. He was discharged and maintained on lithium for 1 year. During that year, he had no episodes of assault or self-injury. He occasionally threatened staff and other members but did not "escalate" in the way he had before.

After 1 year, lithium was stopped and placebo was started. Four months later Mr. B began to escalate in characteristic ways. Haloperidol was started immediately, but despite this he smashed a wall with his fist and again broke multiple bones in his fingers and wrist. Within 2 weeks of hospitalization on a combination of lithium and haloperidol, he became manageable. Haloperidol was tapered and stopped within a month. He has continued on lithium for a year and a half and has not injured himself or the staff in this time.

DISCUSSION

Institutionalization per se can produce signs and symptoms that resemble major psychosis or major affective disorder. In addition, mental impairment clouds or confuses attempts at secondary psychiatric diagnosis. However, major psychiatric diagnosis can and should be made in some institutionalized mentally retarded individuals because the treatment can be useful and perhaps central to their habilitation.

The diagnosis of mania in the retarded can best be made by comparing the patient's normal behavior to that he or she exhibits during periods of unstable mood. When vocal function is present, even if speech is incomprehensible, pressure of vocalization can be observed. Increased motor activity appears in the manic phase. When compared to a previous baseline, intrusiveness, assaultiveness, destructiveness, and self-abuse also suggest mania. The duration of symptoms helps differentiate mania from panic states. Confusion, cognitive impairment, and hallucinations can occur during mania but are more easily detected in patients with higher IQs (11). Direct-care staff commonly note changes in eating and sleeping patterns, usually decreased appetite and sleep in mania and either increased or decreased appetite and sleep in depression.

Psychomotor retardation is difficult to differentiate from mental retardation per se, organic deterioration, or psychosis not related to affective disorder in the mentally retarded if the patient's normal function is not known. However, if a retarded patient's normal behavior is well-known to the family and staff, the diagnosis of depression can be made without great difficulty. Cyclically depressed mood and confusion were present in all of the depressed patients we studied. Periodic psychomotor slowing, social withdrawal, loss of appetite, and disturbed sleep patterns were other symptoms of depression.

A controlled study by Naylor and associates (12) indicated that lithium was superior to placebo in the treatment of periodic disorders of mood and behavior

in a group of 14 retarded patients. Our study corroborates this finding by documenting amelioration of symptoms by lithium and by demonstrating symptom relapse when lithium was stopped and placebo substituted. Naylor and associates (12) found a suggestive but not significant difference in the number of episodes of mania or depression when patients were given lithium. The fact that we found illness episodes significantly less with lithium treatment may reflect the relatively high number of bipolar (four) and unipolar manic (one) patients and absence of any unipolar depressed patients in our group. The young age of our patients may also have predisposed to a successful result with lithium (15).

The experimental procedure used in this study allowed staff to select symptoms they could easily observe and record that were in accord with major criteria of manic-depressive disorder. Such a flexible recording system is inexpensive and practical given the cooperation of invested direct-care staff, even if they are not trained in research procedures. The large number of different staff recorders (fifty) and recording periods in this study might have obscured a drug effect. The significant and positive drug effect demonstrated is, therefore, all the more compelling and suggestive of the important contribution of lithium carbonate as a treatment for the subjects in the study.

No seizures occurred in the patients, three of whom had nonfocally abnormal EEGs before lithium treatment. These focal abnormalities improved in two patients and were unchanged in the third. Our experience suggests that lithium can be used without the risk of seizure in retarded patients in the same way as it has been used in some epileptics (16–18). Lithium-related status epilepticus, as described by others (19, 20), was not encountered.

The results suggest that among an institutionalized mentally retarded population, unipolar and bipolar affective disorder, as first outlined by Kraepelin (3) and refined by recent diagnostic criteria (13), can be diagnosed and effectively treated with lithium carbonate. We feel that the correct criteria for diagnosis of major affective illness should not exclude those individuals with primary mental retardation, and we would not agree with those who feel that manic-depressive illness cannot be diagnosed or does not exist in those with mental retardation (21–23). The presence of manic-depressive disorder as a potentially treatable condition deserves wider attention in texts and reviews of the emotional disorders of the retarded, where it is currently largely unmentioned.

Although they require further replication, our results suggest the importance of the evolving medical model (24) as it relates to major affective disorder. Symptoms that might otherwise have been considered random or peculiar to a particular individual were identified by using diagnostic criteria and the help of families and direct-care staff as corresponding to a specific and well-described illness for which there is a specific treatment. Once an adequate rating system for these symptoms is devised, an additional effective treatment modality could be documented and proved to be an important contribution to the multidisciplinary efforts to habilitate these patients.

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The Efficacy of Drugs and Psychotherapy in the Treatment of Acute Depressive Episodes

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The efficacy of tricyclic antidepressants and various psychotherapies, in comparison with one another or in combination, has not been fully established in randomized clinical trials. The authors present a randomized controlled trial comparing the combination of amitriptyline and short-term interpersonal psychotherapy, either treatment alone, and nonscheduled supportive psychotherapy in ambulatory patients with acute depression. They found that pharmacotherapy and psychotherapy alone were equally efficacious and better than nonscheduled treatment. The combination treatment of psychotherapy and pharmacotherapy was more effective than either treatment alone and delayed the onset of symptomatic failure.

IN CLINICAL PRACTICE the major treatments for acute depression are tricyclic antidepressants and various psychotherapies. Their efficacy, either in comparison with one another or in combination, has not been fully established in randomized clinical trials.

Ideally, before proceeding to studies including both drugs and psychotherapy, there should be evidence for the efficacy of each treatment alone, in comparison with one another, and, finally, in combination (1). For depression, such information is only partially available. There are many studies showing the efficacy of pharmacotherapy alone (2). We are aware of no published studies of psychotherapy alone in a homogeneous sample of more than 10 acutely depressive patients (3–6), although several are now under way (7–9). The one comparison study of imipramine versus cognitive therapy in acute depression (10) showed a positive effect for cognitive therapy over imipramine. There have been three studies comparing maintenance tricyclic antidepressants and various psychotherapies in recovering depressed patients (11–14). In general, these studies support the value of combining drugs and psychotherapy.

This paper will report on a 16-week controlled clinical trial conducted collaboratively by two clinics comparing the efficacy of tricyclic antidepressants and short-term interpersonal psychotherapy each alone and in combination in acutely depressed patients. Other publications will describe the study in detail (15).

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METHOD

Patients

Acutely depressed male and female ambulatory patients aged 18–65 were considered eligible for inclusion in the study. Inclusion criteria also required a nonbipolar, nonpsychotic, acute primary major depression of sufficient intensity to reach a score of at least 7 on the Raskin Three Area Depression Scale (range, 3–15) (16). The diagnosis of major depression was made by a psychiatrist using the Schedule for Affective Disorders and Schizophrenia interview (17) and based on the Research Diagnostic Criteria (18).

Excluded were patients with another predominant disorder, such as organic brain syndrome, schizophrenia, or mania. Patients were also excluded who failed to respond to a previous course of weekly psychotherapy of at least 3 months' duration, who were currently in psychotherapy, who had had an adequate trial of amitriptyline in the past 3 months, or who had a history of medical disorder precluding the use of tricyclic antidepressants.

Design

Patients who met the initial inclusion criteria were assessed over 1 week to exclude patients who remitted

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rapidly without specific treatment. Rapid remission was defined as symptomatic improvement sufficient to rate a 6 or less on the Raskin scale during the no-treatment evaluation week. After the evaluation week eligible patients were randomized into one of four treatment cells, as follows:

Psychotherapy alone consisted of at least one weekly 50-minute session with the treating psychiatrist. No psychotropic drugs were allowed. The nature of the psychotherapy, as described in an unpublished manual of the Boston-New Haven Collaborative Study, was short-term, interpersonal, and focused on the social context of the depression.

Pharmacotherapy consisted of amitriptyline in a flexible divided dose, 100–200 mg, which was raised to 125 mg within a week, stabilized over the next 3 weeks, and maintained for the next 12 weeks.

Psychotherapy plus pharmacotherapy consisted of both treatments as described above.

Nonscheduled treatment involved assigning patients a psychiatrist whom they were told to contact whenever they felt a need for treatment. No active treatment was scheduled, but the patient could telephone and, if his or her needs were of sufficient intensity, a 50-minute session (a maximum of one a month) was scheduled. This procedure allowed a patient to receive periodic supportive psychotherapy "on demand."

Independent Clinical Evaluation

Participation in the study was only by the informed consent of the patients. Procedures were explained verbally and in writing before patients entered the study. All of the patients were seen for clinical assessment after 1, 4, 8, 12, and 16 weeks, or at the termination of treatment, by a clinical evaluator (a psychiatrist or a psychologist) who was independent of and blind to the patient's treatment. Patients were instructed not to discuss with the evaluator the type of treatment they were receiving. The treating psychiatrist also evaluated the patient. Based on a weighted Kappa analysis, agreement between clinical evaluator and psychiatrist, using the Raskin Three Area Depression Scale (16), was excellent and significant ($p<.001$) in both clinics.

Symptom Failure As an Outcome Criterion

Multiple outcome measures were used (15). The main outcome criterion for this presentation was symptomatic failure, defined as a Raskin depression score of 9 or more, as rated by the clinical evaluator after 8 weeks of randomized treatment. Patients who had previously responded and then worsened as well as patients who had never shown substantial clinical improvement were considered symptomatic failures, as were those in whom the clinical evaluator or the treating psychiatrist noted risk of suicide or rapid worsening of symptoms requiring alternate active intervention, hospitalization, or ECT at any time during the study. All symptomatic failures were withdrawn from the study and treated as clinically indicated.

Other Criteria for Withdrawal

Patients who made substantial deviations from the randomized treatment condition (e.g., refusal of medication or failure to attend psychotherapy) were withdrawn at the point of deviation. However, if the symptoms of these patients did not get worse they were not considered symptomatic failures.

Statistical Analysis

A life table approach (19, 20) was used to calculate symptomatic failure rates. Patients withdrawn from the study for reasons other than symptomatic failure, such as noncooperation, were treated in the analysis as censored observations, i.e., as if they were lost to follow-up at the time of their withdrawal.

RESULTS

Characteristics of the Patients

Ninety-seven patients entered the study. One of these was a "rapid remitter." Therefore, 96 patients were randomized into treatment (51 in Boston, 45 in New Haven). However, 15 of the randomized 96 patients refused the treatment assigned and did not complete 1 week of treatment. They have been excluded from the efficacy analysis, leaving 81 patients for analysis. There was no difference in sociodemographic or clinical characteristics between patients who completed 1 week of treatment and those who did not or between treatment groups.

Reasons for Termination

Table 1 shows the completion rates and the reasons for early termination. Sixty-seven percent of the patients in combined treatment completed the full 16 weeks, compared with 48% in psychotherapy alone, 33% in pharmacotherapy alone, and 30% in the nonscheduled group.

Relative Probability of Symptomatic Failure

Figure 1 presents the probability of symptomatic failure over the 16 weeks in the various treatment groups. The lowest rate of failure was in the combination treatment group and the highest was in the nonscheduled treatment group.

Table 2 shows the significance of the difference in the survival curves presented in figure 1. Patients in the three active treatment groups showed a significantly lower failure rate than those in the nonscheduled treatment group. Although there were fewer relapses in the combined treatment group than in either psychotherapy or pharmacotherapy alone, this difference was significant at only the $p<.10$ level. There were no statistically significant differences in failure rates between the two single treatments of psychotherapy and pharmacotherapy.

In the nonscheduled group there was a steady increase in failure that began early in the first week and

TABLE 1
Reasons for Termination from Randomized Treatment, by Treatment Groups

Item	Treatment Group							
	Psychotherapy and Pharmacotherapy		Psychotherapy		Pharmacotherapy		Unscheduled	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Less than 1 week of randomized treatment (excluded from efficacy data) (N=15)	1	4	8	32	4	17	2	9
Terminated at or after one week								
Symptomatic failure (N=20)	1	4	3	12	5	21	11	48
Other reasons								
Adverse side effects (N=2)	1	4	0		1	4	0	
Refused treatment (N=12)	3	13	0		6	25	3	13
Received insufficient medication (N=1)	1	4	0		0		0	
Moved (N=3)	1	4	2	8	0		0	
Total (N=18)	6	25	2	8	7	29	3	13
Completed 16 weeks in randomized treatment (N=43)	16	67	12	48	8	33	7	30
Total (N=96)	24		25		24		23	

FIGURE 1
Life Table Calculations of the Product-Limit Estimate of Symptomatic Failure

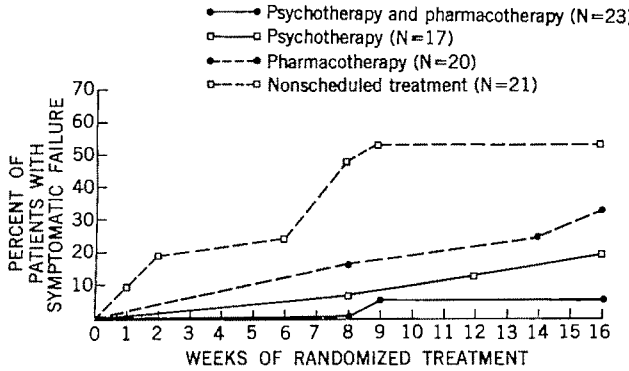


TABLE 2
Significance of the Differences in Survival Curves Among Treatment Groups

Comparison	W	Z Value	Significance
Psychotherapy and pharmacotherapy versus			
Pharmacotherapy	79	1.7	$p<.10$
Psychotherapy	37	1.0	n.s.
Nonscheduled treatment	222	3.5	$p<.001$
Pharmacotherapy versus			
Psychotherapy	35	0.8	n.s.
Nonscheduled treatment	138	2.2	$p<.05$
Psychotherapy versus nonscheduled treatment	147	2.6	$p<.01$

[a]According to the Gehan modification of the Wilcoxon test (20).

increased until week 12, when it reached a plateau. There were no symptomatic failures in the combination group before week 12. The independent clinical evaluator, who was blind to the treatment conditions, was required by the protocol to withdraw at 8 weeks patients who were symptomatic at the Raskin level of 9 or higher. No patients in the combined treatment group were withdrawn at this point. In the pharmacotherapy alone and psychotherapy alone groups there were no failures before week 8.

DISCUSSION

These data suggest that the combination of tricyclic antidepressants and short-term individual psychotherapy is more efficacious than either treatment alone or than nonscheduled treatment for acute depressive episodes. Either treatment alone, whether drug or psychotherapy, was about equal and more efficacious than nonscheduled treatment. Moreover, the combination treatment delayed the onset of symptomatic failure if it did occur.

Research on the psychotherapy of depressive disorders has been developing rapidly, and a number of studies are currently in process. Although we are aware of no directly comparable studies completed as yet, the study of Rush and associates (10) is similar. These authors compared cognitive therapy with imipramine and found cognitive therapy to be more efficacious in reducing symptoms and preventing attrition. We did not find that psychotherapy was more efficacious than pharmacotherapy. The individual treatments were equally efficacious and better than nonscheduled treatment. The study of Rush and associates did not have a combined treatment group or a control group, so we do not know if they would have found the combination treatment superior or if their active treatments were superior to a control treatment.

Our results also showed that most of the depressive patients did not recover spontaneously in the short run. Although there was mild symptomatic reduction during the evaluation week, the patients were still symptomatic and still required treatment. Only 1 patient out of 97 was considered a rapid remitter without specific treatment. Before concluding that rapid remission did not occur, we must acknowledge possible selection due to help-seeking and referral patterns.

The nonscheduled treatment group, which was not offered regularly scheduled treatment, had the worse overall outcome. Despite their generally poorer outcome, a small proportion of patients in this group were not symptomatic failures and reported satisfaction with this treatment. Our own clinical experience with depressed outpatients has been that some of them report comfort in knowing help is available but prefer to contact the therapist as they feel it is indicated rather than on a prescribed basis. It should be emphasized that this group was not a no-treatment or a no-contact group. These patients had the security of knowing that they could contact the clinic, and they knew they were to be seen for regular research assessments. Although the majority of the patients required more active intervention, it would be important to be able to predict who are the substantial minority of patients who responded to this infrequent, nonscheduled treatment.

There were major differences in the patients' acceptance of different treatments. Thirty-two percent of the patients assigned randomly to psychotherapy dropped out before the first week. Initial nonacceptance of treatment was lower in the groups offered pharmacotherapy: only 17% dropped out before the first week. However, during the course of treatment, 25% of the patients in the pharmacotherapy group discontinued their medication. Since the efficacy of drugs alone and psychotherapy alone was about equal and both treatments were better than nonscheduled treatment, the patient who does not accept psychotherapy may be a good candidate for drugs alone, and vice versa.

We should comment on the generalizability of these data. Our sample of patients were all ambulatory and had a diagnosis of neurotic depression according to *DSM-II* and major depression according to *DSM-III*. There is controversy as to whether they would be considered unipolar because of the differing requirements of number of previous episodes. They definitely were not bipolar or psychotic depressive patients.

CONCLUSIONS

Combined tricyclic antidepressant treatment and weekly short-term interpersonal psychotherapy reduces overall symptoms in ambulatory patients suffering from a nonpsychotic, nonbipolar major depression better than either treatment alone or nonscheduled and irregular contacts with a psychiatrist. For the patient who will not accept the combination of treatments, either pharmacotherapy or psychotherapy alone is preferable to nonscheduled contact. Future publications will describe in detail the rationale for our study design, particularly the exclusion of a placebo group, the specifics of the psychotherapy, and the differential outcomes of drugs and psychotherapy on clinical symptoms (15).

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Depression Treated with Imipramine and ECT: The DeCarolis Study Reconsidered

BY DAVID AVERY, M.D., AND ALDO LUBRANO, M.D.

The authors reevaluate a large prospective Italian study (437 patients) that compared high-dose imipramine with ECT treatment in the treatment of depression. The superiority of ECT was evident among patients with endogenous depression, and especially evident among those with delusional depression (83% improved with ECT versus 40% with imipramine) and depressions defined as severe (83% versus 35%).

IN 1964 in the Italian journal *Sistema Nervosa*, DeCarolis and associates published one of the largest studies (437 patients) comparing ECT and imipramine in the treatment of depression (1). This study is one of the few to compare ECT with high-dose imipramine therapy—at least 200 mg/day to a maximum of 350 mg/day for 25 days. The study is unique in that all 437 patients were given the trial of imipramine, and the ECT group was composed only of the imipramine failures. Unfortunately, the English readers' only exposure to the article has been a short *Excerpta Medica* summary in which the authors presented their data in such a way that the efficacy of imipramine was emphasized (2). A look at the original data is crucial to an adequate understanding of the study.

METHOD

The study included 183 men and 254 women who were hospitalized for depression at the University of Genoa Hospital. The mean age was 47.3 years. The depressions were classified into eight types: 1) manic-depressive psychosis—bipolar affective disorder, depressed phase; 2) endogenous monopolar depressive psychosis—unipolar depression, without manic episodes, with clear periodicity (these were "frankly endogenous"); 3) cycloid psychosis—as described by Kleist (3) and Leonard (4), psychosis associated with a clearly phasic course but atypical, i.e., schizophrenic symptoms such as catatonia, delusions, and feelings of influence; 4) atypical depressive psychosis—psychosis associated with a strong delusional component, dissociative symptoms, a clinical picture dominated by depressive symptoms, and no evidence of periodicity; 5) symptomatic depression—secondary depression, e.g., depressive symptoms secondary to schizophrenia; 6) psychoorganic depression—depression associated with deterioration or organic lesions clearly evident clinically or radiographically; 7) neurotic depression—depression associated with a structural conflict and clearly abnormal personality traits, e.g., phobic-obsessive; 8) reactive depression—depression with a very clear relationship between the precipitating event and the psychopathology; patients who had had a previous episode of an endogenous type were not considered in this group.

The depressed patients were also examined for the presence of specific symptoms, presence of precipitating events, and severity (mild, moderate, or severe). A severe depression was defined by the presence of suicidal ideation and alteration of psychomotor activity, either retardation or agitation.

All 437 patients were started on imipramine at a minimum dose of 200 mg/day to a maximum of 350 mg/day for at least 25 days. In some cases the imipramine had to be given parenterally. In some cases a barbiturate or phenothiazine was added. All patients whose trial of imipramine was unsuccessful after a period of 30 days were started on a course of ECT (a minimum of 8 treatments, a maximum of 10).

Evaluation by at least two of the authors took place at least one month after the end of treatment. A patient was considered improved if he recovered with rebound euphoria; returned to his premorbid personality, *resitutio ad integrum*; or showed remarkable improvement and was essentially reintegrated socially. A patient was considered unimproved if there was worsening of symptoms; no change in symptoms; or improvement was insufficient for return to social functioning.

DeCarolis used 437 as the denominator to express the percentage of patients who improved with ECT, even though the total number of those who received ECT was 437 minus the number who responded to imipramine. Even when a subgroup of the total sample

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TABLE 1
Response of Different Diagnostic Groups to Imipramine and ECT

Diagnosis	After Imipramine N	Improved	%	After ECT N	Improved	%
Manic-depressive psychosis	15	7	47	8	8	100
Endogenous monopolar depression[a]	249	153	61	96	80	83
Cycloid psychosis	18	13	72	5	4	80
Atypical depressive psychosis[a]	64	21	33	43	31	72
"Symptomatic" depression	4	0	0	4	4	100
"Psychoorganic" depression	9	6	67	3	1	33
Neurotic depression[b]	57	35	61	22	6	27
Reactive depression	21	12	57	9	2	22

[a]p<.001.
[b]p<.05.

TABLE 2
Response of Various Subgroups to Imipramine and ECT

Sample	After Imipramine N	Improved	%	After ECT N	Improved	%
Total group	437	244	56	190	137	72
Endogenous depressions[a]	282	172	61	109	93	85
Severe depressions[a]	170	60	35	110	91	83
Delusional depressions[a]	181	72	40	109	91	83
Neurotic-reactive depressions[b]	78	47	60	31	8	26
Precipitating event	263	152	58	111	69	62
No precipitating event[a]	174	95	55	79	67	84

[a]p<.001.
[b]p<.005.

TABLE 3
Comparison of Delusional and Nondelusional Depression Treated with Antidepressants

Study	Delusional Patients N	Improved	%	Nondelusional Patients N	Improved	%
Simpson and associates (5)[a]	15	8	53	36	31	86
Hordern and associates (9)[a]	27	4	15	110	89	81
DeCarolis and associates (1–4)[b]	181	72	40	256	174	68
Glassman and associates (23)[c]	13	3	29	21	14	67

[a]p<.01.
[b]p<.001.
[c]p<.05.

(e.g., delusional depression) was statistically analyzed, the percentage who improved with ECT was calculated using the total number of delusional patients as the denominator, whether or not they received a course of ECT.

We recalculated these data simply by dividing the number improved with ECT by the total number who received ECT. Statistical analysis was done using chi-square analysis, two-tailed without Yates' correction. In addition, 196 subjects were followed up for an average of 18 months (range, 8 months to 4 years) for recurrence of symptoms (5, 6).

RESULTS

The results of the recalculation of the DeCarolis data are summarized in tables 1–3. Table 1 shows the percentage response for the diagnostic groups. The clear superiority of ECT is seen among the manic-depressive psychoses, the endogenous monopolar depressions, and the atypical psychoses. Among the neurotic and reactive depressions, a greater percentage responded to imipramine initially compared with the percentage of imipramine failures who responded to ECT.

Table 2 summarizes the percentage response of the different subgroups to the two treatments. The endogenous groups include the manic-depressive psychosis, endogenous monopolar depressions, and the cycloid psychosis. Of the delusional patients 97 had typical depressive delusions (delusions of guilt, self-accusation, low self-esteem, and ruin), and 84 had atypical depressive delusions (hypochondriacal, persecutory, etc.). The superiority of ECT was essentially the same for both types of delusional patients. Table 3 shows that in

the DeCarolis study delusional patients were significantly less likely to respond to imipramine than were nondelusional patients (40% versus 68%, $p<.001$).

In the follow-up study, persisting recovery after imipramine therapy (37%) was found no more frequently than after ECT (38%). However, the duration of the remission with ECT was significantly longer than with imipramine (14 months versus 10 months, $p<.01$).

COMMENTS

There is increasing evidence that the dosage of antidepressant medication may be a crucial factor in antidepressant response. For example, Simpson (7) found that depressed patients receiving imipramine, 300 mg/day, did significantly better than those on a 150-mg dose. In the psychiatric literature written in English, seven studies involving over 2,000 patients conclude that ECT is superior to antidepressant medication (8-14). Three studies involving 200 patients show no difference (15-17). None show antidepressants superior to ECT. In general, these studies used doses of 150-200 mg/day with two exceptions. Kristensen (15) rapidly increased imipramine to 300 mg/day by the sixth treatment day and found a 57% recovery rate (evaluated prospectively), which was comparable to the recovery rate (evaluated retrospectively) seen in the ECT group. Wilson and associates (18) found that imipramine at a 150-mg dose was inferior to ECT, but when 240 mg/day was given to 9 patients, there was no significant difference compared with 10 patients receiving ECT. Because of the question of whether high-dose tricyclics can approach the efficacy of ECT, the large DeCarolis study and its use of imipramine, 200-350 mg/day for 25 days, takes on special significance.

Even though the study is not without obvious flaws in design, it adds much valuable data relevant to common clinical decisions. The study was not blind. The samples of the treatment groups were not independent—the ECT group consisted only of imipramine failures. Yet it would seem that this inequality would bias the data against ECT, since the imipramine failures contained a greater percentage of severely depressed (58%) and delusional depressed patients (57%) than the original sample (39% and 41%, respectively). Furthermore, antidepressant failure in itself may predict a relatively poor outcome (19, 20) and therefore may be a source of bias against a good ECT response. Another weakness of the study is that criteria for depression were not specified; however, it is clear that those with severe depression had suicidal ideation and either psychomotor retardation or agitation. The signs and symptoms of depression of the nonsevere groups were not made explicit.

Despite these methodologic problems, the study is a close approximation to a common clinical practice in the treatment of depression. In many hospitals, the DeCarolis experimental design is the routine way of treating clinical depression regardless of severity. In such a situation there frequently arises an important clinical question that the study addresses: if a depressed patient has not responded to an adequate course of antidepressants, what are his chances of responding to ECT? The data indicate that if an agitated or retarded, suicidal depressed patient has failed to respond to an adequate course of antidepressants, he still has an excellent chance of responding to ECT. The other studies that have shown this to be true used lower doses of antidepressants (9-11).

The neurotic and reactive depressions responded poorly to ECT (see table 1). It could be argued that these patients, being imipramine failures, were the most treatment-resistant and that a greater percentage would have responded had they been given ECT first. Furthermore, because of the lack of criteria for depression in this study, a number of these imipramine failures may not have had signs and symptoms of depression and may have been experiencing chronic characterologic unhappiness, a problem that is unlikely to improve with somatic therapy. Although ECT's efficacy in neurotic-reactive depression relative to imipramine could be questioned, it seems clear that neurotic-reactive depressions respond less well to ECT than do endogenous depressions. This finding is in accord with at least three other investigations (21-23). Although only 2 of 9 reactive depressions responded to ECT the presence of a precipitating event in itself was not associated with a poor ECT response (62% response rate). Thus, a history of a precipitating event in itself should not preclude the possible use of ECT.

The excellent ECT response seen in patients with atypical depressive psychoses suggests that the indications for ECT may not be limited to clear-cut unipolar and bipolar affective disorder. In these patients, although depressive symptomatology dominated the clinical picture, there was no evidence of any episodic nature of the illness, and strong delusional and dissociative symptoms were common.

The data are also relevant to a more common question: what should be the *initial* treatment of a severely depressed person? Although this study cannot offer strict guidelines for this decision, the data can give the clinician a better idea of the probability of response. The DeCarolis data indicate that when the practice of routinely giving a trial of antidepressants first is rigorously followed, the majority of the severely depressed patients will not have an adequate response to the medication (table 2).

A study by McLeod (24) gives indirect support for this finding. The severity of depression has been associated with high plasma cortisol levels and abnormal dexamethasone suppression (25). McLeod found that those depressed patients with a high cortisol level at 8:00 a.m. and those with an abnormal dexamethasone suppression test had a very poor response to antidepressants; only 26% (9 of 35 patients) improved with either imipramine or amitriptyline, 150-225 mg/day,

for a period of at least four weeks. A significantly greater percentage of improvement, 63% (22 of 35 patients), was seen among those depressed persons who had normal cortisol levels and a normal dexamethasone suppression test ($p<.005$) (24). The implications of these data are broadened by the finding that psychiatric patients who commit suicide have significantly higher cortisol levels than those patients who do not ($p<.01$) (26).

The DeCarolis data also show that the majority of the delusional depressed patients will not have an adequate response to antidepressants. Three other prospective studies led to a similar conclusion (7, 11, 27) (see table 3). On the other hand, delusional depressions respond well to ECT (21, 22, 27). The high probability of antidepressant failure and the resultant prolongation of patient discomfort and increased expense should be factors in determining the initial treatment of delusional as well as severe depressions.

At least two investigations have shown a positive relationship between clinical improvement and imipramine plasma levels, but they also reported that the delusional depressed patient has a poor response to imipramine even at high plasma levels (28, 29). Further studies should be done to resolve whether the establishment of therapeutic plasma levels can increase the efficacy of antidepressants to the quality of response seen with ECT.

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Lithium Maintenance: Factors Affecting Outcome

BY JAMES H. KOCSIS, M.D., AND PETER E. STOKES, M.D.

The authors reviewed the charts of 61 manic-depressive outpatients maintained on lithium for 6–75 months for relationships between clinical outcome and various demographic, clinical, and pharmacological variables. Good outcome (50% reduction in episode frequency) did not correlate with any demographic or most natural history variables. A history of frequent episodes was correlated with decreased episode frequency but not with a lower percentage of prophylaxis failure during follow-up. A nonsignificant trend toward decreasing episode occurrence with increasing duration of lithium maintenance was present. Depressions occurred considerably more frequently than manic episodes at adequate maintenance plasma lithium levels. More medication noncompliance was found among patients who experienced both manic and depressive relapses.

THE EFFICACY of lithium carbonate for maintenance therapy of bipolar affective disorder has been established in a number of double-blind, controlled studies (1–8). Results from these studies, however, have shown rates of occurrence of affective episodes in bipolar patients on lithium maintenance as high as 50%. Factors cited as relating to unfavorable lithium maintenance outcome include inaccurate diagnosis (8), previous diagnosis of schizophrenia (9), diagnosis of schizo-affective psychosis (10), rapid cycling (1, 2), lack of insight (11), side effects (12), and inadequate dosage (8, 13, 14). Single reports of more favorable outcome with advancing age (11) and family history of bipolar illness (13) have not been confirmed by others (1, 2, 10).

To gather further evidence relating to clinical and pharmacologic factors in lithium maintenance outcome, we reviewed the records of 61 bipolar patients treated on an open basis with lithium carbonate for 6–75 months (mean= 50.6 months). In this report, we will give the results of this chart review.

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METHOD

Eighty-eight patients with bipolar affective disorder were admitted to our outpatient lithium program during 1970 and 1971. Of these, 24 patients were seen for less than six months, so lithium maintenance outcome could not be evaluated, and 3 patients were later felt not to have bipolar disorders. The remaining 61 patients were maintained on lithium for 6–75 months (mean= 50.6).

Patients were diagnosed as having bipolar affective illness (at least one episode of depression and mania or hypomania requiring treatment within the past two years) by two psychiatrists, including one of us (P.E.S). Criteria for diagnosis included a history of clear-cut episodes of both depression and mania (hypomania) with typical symptoms (15) and the absence of current Schneiderian first-rank symptoms of schizophrenia. Almost without exception these patients fulfilled Research Diagnostic Criteria (16) for bipolar depression with mania or hypomania, primary (N=51) or secondary (N=10) type. (These criteria had not been published at the time of these patients' admission.) Demographic data for these patients are shown in table 1.

Patients were treated with lithium carbonate on an open basis. Plasma lithium levels were obtained monthly on Monday mornings 12–14 hours after the last lithium ingestion. Zero levels indicate medication noncompliance. Clinic visits were scheduled for every one to four months, and patients were seen more often if this was clinically indicated. At each visit a symptom checklist was filled out and a Payne Whitney Clinic Global (PWCG) rating of affective state (18, 19) was made by a physician. An episode was defined as an occurrence of affective symptoms requiring hospitalization or any additional psychopharmacologic intervention including an increase in lithium dose. Appropriate concomitant medications (phenothiazines or antidepressants) were used or lithium dose was increased as clinically indicated. Although patients were urged to take their medication as directed, no attempt was made to strictly monitor compliance, and the usual problems of outpatient psychopharmacologic treatment occurred.

At the time of admission to the clinic, patients were interviewed by at least two psychiatrists and a thorough history was taken with particular attention to the number and type of episodes and hospitalization during the five years before admission. Good outcome was defined as a decrease of at least 50% in the annual

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TABLE 1
Demographic and History Variables of Patients on Lithium Maintenance

Variable	Outcome Group	
	Good (N=31)	Poor (N=30)
Mean age	45.6	47.1
Sex		
Male	13	15
Female	18	15
Mean social engagement[a]	2.41	2.20
Type of affective illness		
Primary	26	25
Secondary	5	5
Mean age at onset	29.6	28.7
Mean duration of illness (years)	16.4	18.6
Mean number of episodes per year (last 5 years)	1.14	0.86[b]
Episodes per year (last 5 years)		
Fewer than 0.5	6	12[c]
0.5–2.0	17	13
More than 2.0	9	4
Lithium treatment at entry		
Begin	15	10
Continue	16	20
Affective state at entry		
Manic or hypomanic	9	11
Depressed	7	9
Normothymic	15	10

[a]Three points possible, one each for marriage, employment, and assumption of appropriate family responsibility (17).
[b]Difference between means is significant (p=.025, Student's t test).
[c]Difference between high and low frequency groups is significant (p<.05, chi-square analysis).

TABLE 2
Annual Rate of Affective Episodes and Hospitalization Before and During Lithium Maintenance Follow-Up

Episodes	Five Years Before Lithium	During Lithium	p[a]
Manic	.49	.21	.005
Depressive	.51	.27	.02
Total	1.01[b]	.48	.005
Hospitalizations	.33	.07	.005

[a]Paired comparison t test, one-tailed.
[b]35 index episodes were eliminated for purpose of statistical comparison.

Rate of episode occurrence during the lithium maintenance follow-up period.

Data were collected by review of charts, PWCG ratings, and records of plasma lithium levels for each subject. Two factors assessed in addition to demographic and natural history variables were social engagement and condition at entry to the lithium clinic. Social engagement (17) is a factor composed of three points—one for marriage, one for employment, and one for assumption of appropriate family responsibility, e.g., support of children. Three is the maximum score. Condition at entry was defined as whether the patient was continuing or beginning lithium treatment at the time of admission and his or her clinical mood state (manic, normothymic, or depressed).

RESULTS

Table 2 shows the annual rate of episode occurrence before lithium clinic admission and during the lithium maintenance period. The frequency of both types of affective episodes and the frequency of hospitalization decreased significantly in the follow-up period.

The relationships between good outcome and demographic and natural history variables are shown in table 1. There was no significant correlation between outcome and most of the variables examined. Patients with a previous history of more frequent episodes had a more favorable outcome. However, because improvement was defined as a percentage reduction in episode frequency this difference may be a statistical artifact.

To determine if this was the case we examined outcome in terms of the percentage of patients who experienced lithium prophylaxis failure, i.e., had at least one affective episode during follow-up, and found that the frequency of previous episodes did not significantly predict differential prophylaxis failure rates in our subjects. However, it was noteworthy that 4 patients with prior histories of frequent episodes (more than 2 per year) had no episodes during follow-ups ranging from 60–75 months.

We found that nine of the ten (90%) secondary bipolar patients had episodes during lithium maintenance, compared to 34 of the 51 (67%) primary bipolar patients. This trend was not statistically significant (the number of secondary patients was small). Preexisting diagnoses in secondary patients included schizophrenia (5), alcoholism (3), and epilepsy (2).

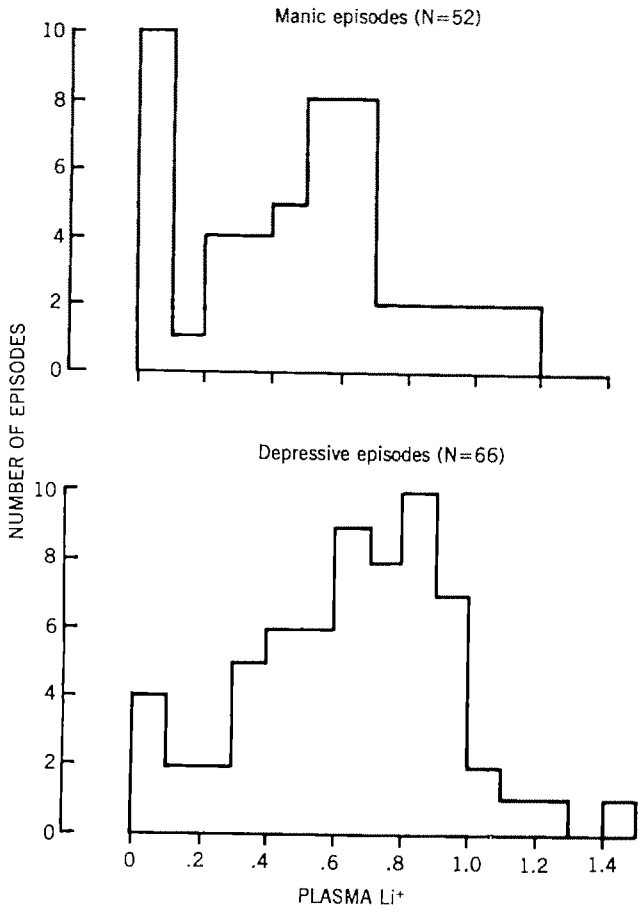
The relationship between episode occurrence and duration of lithium maintenance treatment was examined (in the patients we assigned to lithium at admission). There was a slight trend toward decreasing episode occurrence with longer duration of lithium maintenance treatment, but this did not reach statistical significance. Plasma lithium levels were significantly lower during the three-month periods preceding manic episodes (mean= .47 mEq/liter) than periods preceding depressed episodes (mean= .64 mEq/liter) ($t=2.85$; $p<.01$). Also, more zero levels were recorded prior to manic episodes (10 of 52 episodes) than depressed episodes (3 of 66 episodes), indicating more noncompliance with medication prior to manic episodes. Mean yearly lithium levels taken at anniversary months following admission to the clinic for the patients who experienced no episodes during maintenance treatment was .65 mEq/liter. This was higher than mean levels recorded during the three months prior to manic episodes, depressed episodes, and total episodes (mean= .57 mEq/liter).

The distribution of mean plasma lithium levels before all episodes is shown in figure 1. Seven patients experienced 10 episodes of mania or hypomania at plasma levels consistently higher than .7 mEq/liter. Of these, however, 4 occurred while patients were on concomitant tricyclic antidepressants. Fifteen patients

TABLE 3
Plasma Lithium Levels During Three Months Preceding Affective Episodes

Episode Type(s)	Number of Episodes	Plasma Lithium Levels During 3 Months Before Episode		
		Average Mean (mEq/liter)	Number with Mean of 0	Number with Mean >0.7
Manic only (N=13)	28	.52	3	5
Depressed only (N=15)	26	.62	0	10
Manic and depressed (N=15)				
Manic	24	.41	7	5
Depressed	40	.64	3	20
Total (N=43)	118	.57	13	40

FIGURE 1
Distribution of Mean Plasma Lithium Levels (mEq/liter) During the Three Months Preceding Affective Episodes



experienced 30 depressions while plasma lithium levels were consistently higher than .7 mEq/liter. With the exception of three hospitalizations and one suicide, these depressions were managed psychopharmacologically on an outpatient basis.

Further relationships between types of episodes experienced by all patients and plasma lithium levels three months prior to episode occurrence are shown in table 3. Patients who experienced both manic and depressed episodes had lower lithium levels prior to their manic episodes and more noncompliance than did patients who experienced only manic breakthrough.

DISCUSSION

In evaluating the annual rate of occurrence of affective episodes and hospitalizations for patients in our lithium maintenance program, it is important to be aware that the treatment was open medication, compliance was not strictly monitored, and that other factors such as the use of concomitant medications and supportive psychotherapy effects were present. However, the marked reduction in the rate of episode occurrence and hospitalization during follow-up further supports the previously reported efficacy of this treatment (1–8).

Good outcome (a greater than 50% decrease in episode frequency) was not correlated with most demographic or natural history variables. Patients with a prior high frequency of episodes (two or more per year) improved at a higher rate than did those with prior low frequency of episode ($p < .05$). However, when outcome was examined in terms of the incidence of prophylaxis failure, the prior high frequency group did somewhat worse than the prior low frequency group. Nonetheless, it was noteworthy that 4 patients with prior high frequency experienced no episodes during lithium maintenance periods ranging from 60 to 75 months. These findings suggest that at least some patients with histories of frequent episodes can benefit from lithium maintenance, although amelioration rather than prophylaxis may be the outcome. There may be some overlap between our prior high-frequency group and the so-called rapid cyclers described by Dunner and Fieve (1).

The trend toward a higher incidence of prophylaxis failure among bipolar patients with a history of preexisting psychiatric or medical conditions compared to those with primary bipolar disorder supports the validity of the primary-secondary distinction originally proposed by Robins and Guze (20). We agree with other authors (8–10) who have emphasized the importance of accurate diagnosis in predicting lithium maintenance response. More research is needed on the place of lithium ion for the treatment of secondary affective disorders.

We observed a slight decrease in the annual rate of episode occurrence with longer duration of lithium maintenance, particularly for depression. This may have related to our increasing awareness in recent

years of the importance of maintaining higher plasma levels. Our data are consistent with the possibility that lithium helps to stabilize some underlying biochemical or humoral disruption in bipolar disorders.

Manic episodes arising with plasma lithium levels consistently above 0.7 mEq/liter, in the absence of concomitant antidepressant medication, were infrequent (2 of 61 patients). Depressive episodes occurring at usually clinically adequate plasma levels were considerably more frequent. However, the depressive occurrences were more frequent early in maintenance and were generally mild and 28 of 30 such episodes responded to outpatient pharmacologic intervention. The finding of more medication noncompliance and lower mean plasma lithium levels prior to episodes in patients who experienced both rather than only one type of recurrence may reflect a practice of some patients to decrease lithium when depression occurs, thus creating a "vicious cycle" with rebound mania in patients in the former group. Our findings point out the importance of adequate medication compliance and adequate plasma lithium levels.

It is important to the patients who develop affective episodes while on adequate lithium maintenance as well as the rapid cyclers who stay well during prolonged maintenance on lithium. If they represent biologically homogeneous groups of responders and "nonresponders," it would be interesting to characterize them clinically and biologically. We recently reported erythrocyte lithium data on a population of these important subtypes (21).

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The Delineation of an MMPI Symptom Pattern Unique to Lithium Responders

BY FRED A. STEINBERG, PH.D.

The author attempted to determine whether the Minnesota Multiphasic Personality Inventory (MMPI) would be sensitive to a symptom pattern unique to lithium-responsive patients regardless of diagnosis. Statistical analyses of the MMPI scores of lithium responders, lithium nonresponders, and a group of patients receiving other psychotropic drugs revealed that the depression, psychasthenia, mania, and social introversion scale scores significantly differentiated the lithium response and lithium nonresponse groups. No strong indications of significant difference were found between the MMPI scale scores of the lithium nonresponse and psychotropic groups. The results suggest that there is a lithium-responsive syndrome that exhibits a distinctive symptom pattern regardless of its varying phenomenological features in different individuals.

WITHIN THE PAST 25 years lithium has been acknowledged to have a pronounced therapeutic effect in the treatment of some manic and manic-depressive disorders. A few studies (1–4) have also indicated that a positive response to lithium was observed in patients diagnosed as having recurrent depression, schizo-affective disorders, or acute schizophrenia. The literature (5–6) suggests that recurrent episodes of hyperactivity, excitement, or agitation are common features of individuals with lithium-responsive disorders. However, the regular recurrence of these symptoms does not necessarily indicate which patients will positively respond to lithium therapy. For this reason, the objectification of a basic symptom pattern that is both common among and unique to lithium-responsive patients could have great clinical importance.

Two studies (7–8) attempted to determine whether the Minnesota Multiphasic Personality Inventory (MMPI) could delineate the characteristics of lithium-responsive patients. However, these studies were inconclusive due to methodological deficiencies. I attempted to determine the value of the MMPI for delineating a symptom pattern unique to lithium responders. The data from my investigation were analyzed with respect to the following two hypotheses:

1. At least two MMPI scales will significantly differentiate those who do and those who do not respond to lithium.
2. No MMPI scale will significantly differentiate between patients who do not respond to lithium and patients who are receiving other psychotropic drugs.

METHOD

I used the treatment records and chart documentations of 30 male and 15 female psychiatric outpatients and short-term inpatients who were between the ages of 19 and 63. All patients previously diagnosed as having organic brain dysfunction were excluded from the study due to the possible confounding effects of such impairment on ratings of improvement.

Treatment records were used to assign patients to a lithium response group, a lithium nonresponse group, and a comparison group receiving psychotropic drugs other than lithium. Each group contained 15 patients. The assignment of patients to the lithium response and lithium nonresponse groups was done retrospectively by inclusion of consecutively admitted relevant cases. Each patient's dated medication history was used to determine response or nonresponse to lithium. Patient records used for the psychotropic drug comparison group were chosen randomly.

The drug treatment mode and dosages for patients in all groups were determined by the treating physician prior to this investigation. Most patients initially selected for lithium treatment had episodically recurring disorders marked by agitation, hyperactivity, or excitement. Lithium was prescribed for patients with these pathological features without regard to formal diagnostic categories (see table 1). Subjects were diagnosed as having an episodically recurring disorder if they had had at least one documented episode during the 18 months prior to their latest admission.

The lithium response group consisted of patients whose psychopathology had been stabilized by treatment with lithium alone for a period of at least 4 weeks. It was essential that none of the patients in the lithium response group showed a need for adjunctive

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TABLE 1
Diagnostic Composition of Patient Groups

Diagnosis	Lithium Response Group (N=15)	Lithium Nonresponse Group (N=15)	Psychotropic Group (N=15)
Manic-depressive disorder	10	7	0
Schizophrenia	1	2	5
Unipolar depression	1	2	0
Schizo-affective disorder	1	0	0
Cyclothymic personality	1	0	0
Anxiety reaction	0	0	1
Involutional depression	0	1	0
Depressive reaction	0	2	7
Conversion reaction/ depression	1	1	0
Psychotic depression	0	0	1
Hysterical personality	0	0	1

medications once they were stabilized on lithium. Patients who did need other medications during this time period were placed in the lithium nonresponse group. In some cases it was necessary to administer varying doses of adjunctive medications during the initial phases of drug therapy before lithium produced a therapeutic effect. In such cases, lithium responders were defined as patients whose psychopathology was adequately controlled by lithium alone for a period of 4 months after the withdrawal of adjunctive medications. Patients who warranted a represcription of adjunctive medications during withdrawal from these medications were considered lithium nonresponders.

The third group consisted of patients for whom various psychotropic medications were prescribed. Such medications included phenothiazines, dibenoxepins, butyrophenones, tricyclic antidepressants, and monoamine oxidase inhibitors. Patients in this group had a variety of symptoms other than those used as criteria for prescription of lithium.

All patients had been given the MMPI before any therapeutic drug trials to objectively measure symptoms or symptom patterns characteristic of their pathological states. Any patients whose MMPI profiles had an F scale score greater than 80 were excluded from the study. Essentially, only patients with clinically interpretable MMPI profiles were studied.

A one-factor between-groups analysis of variance was done for each MMPI scale to determine scales that differed significantly among the three patient groups. These analyses were conducted for the following MMPI scales: L, F, K, Hs, D, Hy, Pd, M-F, Pa, Pt, Sc, Ma, and Si. Post-hoc Newman-Keuls tests were then employed to ascertain which patient group had contributed most to the significant variance of the MMPI scales previously delineated.

RESULTS

Figure 1 illustrates the mean MMPI profile for each group of patients. A one-way analysis of variance was calculated to determine the relative degree to which each scale differentiated between groups. Significant differences among the scores for the three groups were found on the following scales: Ma ($F(2, 42)=4.4243$, $p<.01$); Si ($F(2, 42)=6.742$, $p\leq.01$); D ($F(2, 42)=3.6956$, $p\leq.05$); Pt ($F(2, 42)=3.0583$, $p\leq.05$); and L ($F(2, 42)=3.3280$, $p\leq.05$).

DISCUSSION

The first hypothesis of this investigation was that at least two MMPI scales would differentiate significantly between the lithium response and the lithium nonresponse group. Newman-Keuls post-hoc tests revealed that the Ma scale scores were significantly higher in the lithium response group than the lithium nonresponse group ($W_2=8.77$, $p\leq.05$). Three other MMPI scales were significantly lower for the lithium response group compared to the lithium nonresponse group. These scales include the Si scale ($W_2=8.15$, $p\leq.05$), the D scale ($W_2=13.37$, $p\leq.05$), and the Pt scale ($W_2=14.56$, $p\leq.05$).

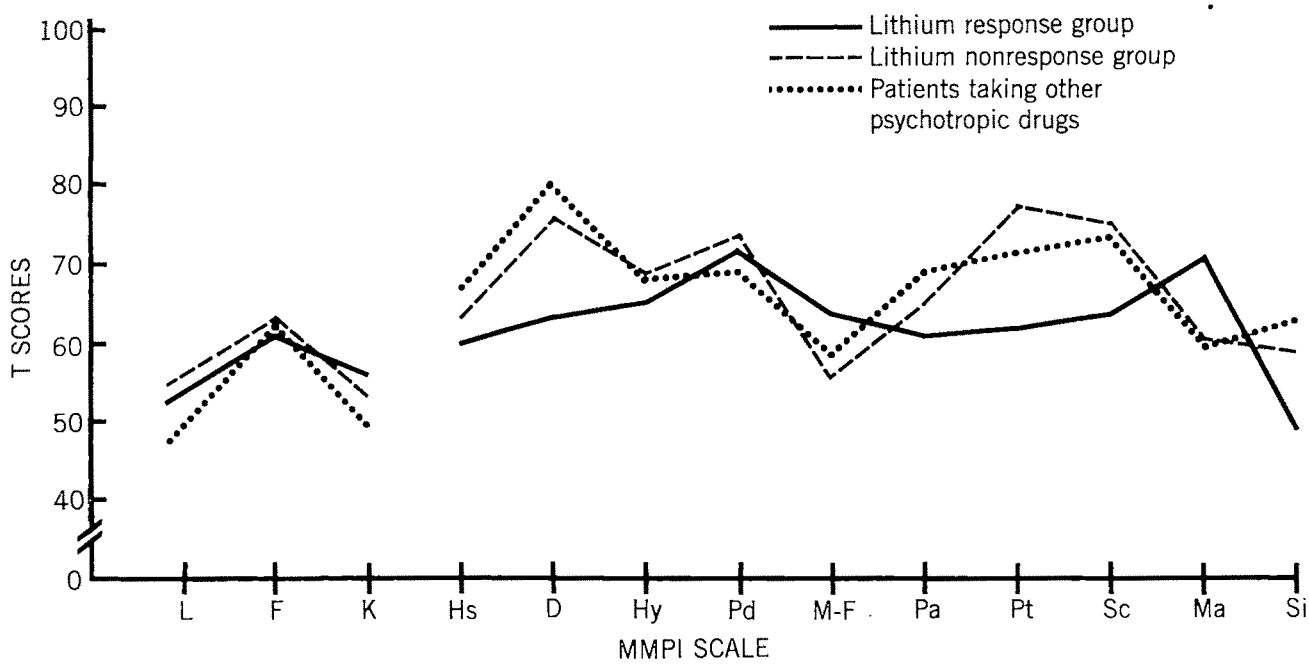
The significantly higher Ma scale scores of lithium responders indicate a higher activation level, increased flight of ideas, and emotional excitement when compared to lithium nonresponders (9). Although this finding would be expected, it is interesting that the lithium responders' Ma scale scores are significantly different from those of other patients whose psychopathology also indicated a lithium trial. The lithium response group also had significantly lower D scale scores. This indicates that lithium responders experienced less subjective depression, psychomotor retardation, physical malfunctioning, mental dullness, and brooding (9). The significantly lower Pt scale scores of lithium responders suggest less anxiety, obsessional rumination, and phobic behavior. Finally, the significantly lower Si scale scores of lithium responders show their tendency toward versatility and sociability (9) when compared to the more socially constricted lithium nonresponders.

The second hypothesis stated that there would be no significant differences between the scale scores of the lithium nonresponse group and the group receiving other psychotropic drugs. It was found that the lithium nonresponse group had significantly higher L scale scores than the psychotropic group ($W_3=6.27$, $p\leq.05$). However, since this scale was the only one that suggested a significant difference between these groups and since it also has few items, it is highly possible that this finding was due to chance.

COMMENT

The focus of this study was on the delineation of a symptom pattern unique to lithium responders. Although not conclusive, the results of this investigation

FIGURE 1
Mean MMPI Scale Scores for Lithium Responders, Lithium Nonresponders, and Comparison Group



may be viewed as a description of symptoms that are unique to lithium responders. The predictive capability of these findings remains untested. In addition, because this study included so few female lithium responders and nonresponders, caution should be used when applying these results to female psychiatric patients.

The diagnostic composition of the lithium response group was not as heterogeneous as had been anticipated. Future research in this area should be directed at replicating the results of this study using patient groups that include more diverse diagnostic categories.

Finally, the mean profile of the lithium response group (figure 1) represents an MMPI actuarial code type that is typically termed sociopathic or antisocial personality (10). The results of this investigation suggest that the construct validity inherent in some MMPI actuarial code types may not be keeping pace with our expanding knowledge of various clinical syndromes. Further investigations of the psychological test characteristics of a "lithium responsive syndrome" are encouraged.

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Treatment of Tardive Dyskinesia and Mania with RBC Lithium Determinations

BY LARRY ERESHEFSKY, PHARM.D., TERRY N. RUBIN, PHARM.D., AND STEPHEN FRIEDMAN, M.D.

The authors report the usefulness of RBC lithium determinations in managing the treatment of tardive dyskinesia. They present a case report in which manic manifestations and tardive dyskinesia symptoms paralleled the rise and fall of RBC lithium and plasma lithium levels were constant during the period of symptom improvement. They conclude that RBC lithium levels appeared to reflect toxicity, control of mania, and symptom suppression of tardive dyskinesia better than plasma lithium levels alone.

LITHIUM HAS been shown to be useful in the treatment of manic-depressive illness (1–6); several studies on the use of lithium in the treatment of tardive dyskinesia (TD) have demonstrated variable outcomes (7–12). All of these studies used lithium plasma (or serum) levels to monitor therapy; however, recent literature disputes the validity and reliability of following the treatment of affective disorders with Li^+ plasma levels alone (13, 14). Red blood cell (RBC) Li^+ determinations (Hisayasu method) are currently being used as a method of assessing the extent of distribution and uptake into central nervous system tissue (15). This measurement is a more reliable prognosticator of impending toxicity or efficacy (16–22). The development of an organic brain syndrome with ordinarily therapeutic doses of Li^+ has been reported. Plasma levels in the low therapeutic range (Li^+, 0.75 mEq/liter) have been associated with both confusion and hyperactivity. Many reports hypothesize that central nervous tissue concentrations and not plasma levels correlate better with the observed behavioral and neurotoxic effects (23–26). A therapeutic window for the treatment of mania with RBC lithium levels between 0.20 and 0.60 mEq/liter has been proposed (13). Other investigators have found a significantly increased RBC lithium level and RBC/plasma lithium ratio in responders than in nonresponders (14, 16). However, the clinical value of RBC lithium determinations remains a controversy (27, 28). In the case report that follows, RBC Li^+ levels correlated well with suppression of TD, neurotoxicity, and changes in manic symptomatology.

No previous studies have correlated treatment of TD with RBC Li^+ determinations. During the hospital course of our patient, an oculofacial TD characterized by blepharospasms, upward deviation of both eyes, tongue protrusion, and jaw movements was observed.

To more easily interpret the following case report and discussion, a subjective rating scale for mania, organicity, and tardive dyskinesia symptomatology was devised (see figure 1). The staff rated the patient based on severity of the target symptom as outlined in the clinical course. A rating of 1 indicated minimum impairment and a rating of 5 represented maximum severity. Figure 2 illustrates plasma Li^+, RBC Li^+, and the RBC plasma Li^+ ratio versus time during this patient's hospitalization.

CASE REPORT

Ms. A, a 52-year-old black woman with a 25-year history of manic-depressive psychosis was admitted to Los Angeles County/University of Southern California (LAC/USC) Medical Center Psychiatric Hospital. Her medical history included three episodes of Li^+ toxicity, one of which resulted in coma. One week prior to this admission, Ms. A was admitted to another hospital for 5 days because of increasing agitation. Ms. A was discharged from this first hospitalization on 1800 mg/day of lithium carbonate, 1 g of chloral hydrate h.s., and 1 tablet of spironolactone with hydrochlorothiazide (Aldactazide) a day. On admission to LAC/USC Psychiatric Hospital, she exhibited polydypsia, polyuria, abdominal pain, anorexia, drowsiness, ataxia, dysarthria, and confusion. Her pertinent laboratory data taken on admission included plasma Li^+, 2.44 mEq/liter; Na^+, 133 mEq/liter; K^+, 3.5 mEq/liter; and serum creatinine, 1.3 mg/100 ml. Her drug history disclosed the use of moderate to high doses of phenothiazines from 1965 to 1971, including chlorpromazine, trifluoperazine, fluphenazine, and thioridazine together with prophylactic trihexyphenidyl. The daily dose was approximately 600–1000 mg of chlorpromazine or its equivalent. In 1972 thioridazine was discontinued, Li^+ therapy was begun, and Ms. A began to exhibit the TD symptoms. Concomitant neuroleptic therapy for periods of agitation and sleeplessness were employed when necessary.

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FIGURE 1
Clinical Rating Scale for Mania, Organicity, and Tardive Dyskinesia

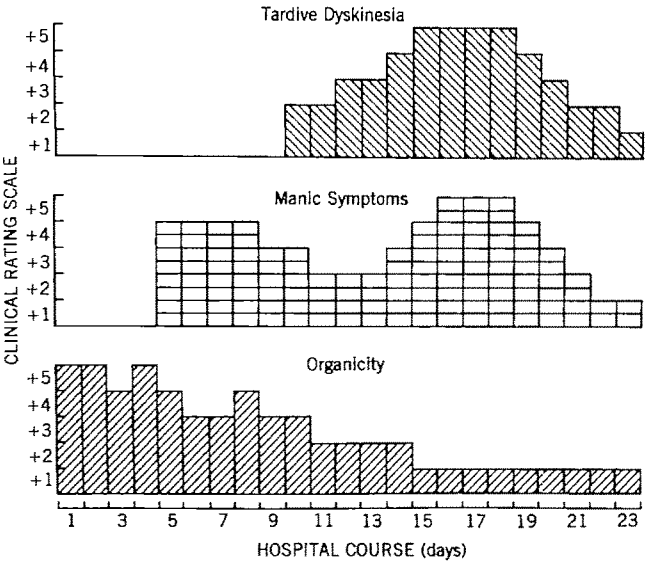
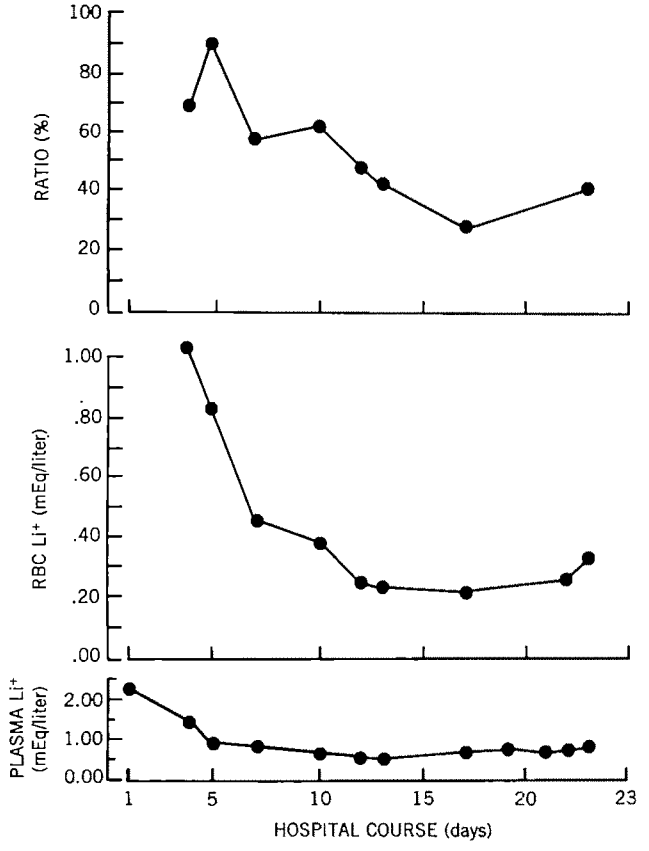


FIGURE 2
Plasma Li^+ Levels, RBC Li^+ Levels, and RBC/Plasma Lithium Ratio



Hospital Course

Days 1-4. On day 1, Ms. A was lithium toxic (plasma Li^+, 2.24 mEq/liter). At this time no medications were administered, but oral fluids (200 ml/hour) were given and no movement disorder was noted. See figure 2 for other lithium values.

Days 5-9. On day 5, Ms. A's lithium carbonate therapy, 300 mg t.i.d., was begun, based on her falling plasma levels and her increasing irritability and euphoria. She was oriented to time, place, and person and was less confused; her speech, although still slurred, was more coherent. During this period her short-term memory slowly improved and the movement disorder remained undetectable. On day 5, her plasma Li^+ was 0.96 mEq/liter, and her RBC Li^+ was 0.84 mEq/liter.

Days 10-13. An improvement in Ms. A's cooperativeness, memory, attention span, and orientation all were noted as evidence of a resolving acute organic brain syndrome. On day 10 writhing movements of the fingers and hands, frequent blinking of both upper eyelids, and masticatory movements of the mouth were independently observed by the nurse, the psychiatrist, and the two pharmacy residents. Daily interviews were conducted by either the psychiatrist or pharmacy residents, and no dyskinesia was observed prior to day 10. On days 12 and 13 finger and jaw movements remained about the same, but upward deviation of both eyes along with increased blepharospasms and involuntary tongue movements were observed. On day 10 her plasma Li^+ was 0.60 mEq/liter, and her RBC Li^+ was 0.37 mEq/liter.

Days 14-15. On day 14, Ms. A became more easily upset, irritable, and loud and displayed a labile affect. Her TD symptomatology worsened; she could not hold out her tongue for more than 10 seconds unless she curled it down resting it on her chin and lip. However, Ms. A remained oriented, had good short-term memory, and displayed minimum residual from her acute organic brain syndrome. The lithium carbonate dose was increased to 1200 mg/day on day 15.

Days 16-20. On days 16 and 17 Ms. A had increasing difficulty falling asleep. Her speech became increasingly pressured as she became agitated. The movement disorder remained about the same as described on days 14 and 15. On day 17 her plasma Li^+ was 0.67 mEq/liter and her RBC Li^+ was 0.21 mEq/liter.

Days 21-23. A noticeable improvement was observed in Ms. A's blepharospasms, and tongue, jaw, and finger movements over this time period. She could hold her tongue out for 25-30 seconds without any apparent discomfort. Her behavior also improved. Ms. A was discharged on day 23 on 600 mg of lithium carbonate b.i.d. On day 23 her plasma Li^+ was 0.75 mEq/liter, and her RBC Li^+ was 0.31 mEq/liter.

DISCUSSION

Lithium's efficacy in the treatment of tardive dyskinesia with the sole use of plasma levels has recently been demonstrated. Reda and associates (9) in a noncontrolled study of six patients demonstrated lithium's usefulness in alleviating but not eradicating symptoms of TD, particularly oral and facial types. Prange and associates (10) demonstrated a dose-related improvement of TD symptoms in two patients who relapsed when placed on placebo. In another study by Reda and associates (8), lithium carbonate in doses of 600-1200 mg/day resulted in a statistically significant improvement ($p<.05$) in bucculolingual masticatory and choreoathetoid movements. Other investigators have

found that dyskinetic movement disorders caused by L-dopa and Huntington's chorea are alleviated by the use of Li^+ (11, 29, 30). Based on the studies by Ashcroft (31), Colburn (32), and Friedman (33), the mechanism of action for Li^+'s beneficial effects are 1) a decrease in the sensitivity of the postsynaptic receptor to dopamine; 2) an increase in the reuptake of dopamine and norepinephrine, and 3) a decrease in dopamine synthesis in the corpus striatum secondary to feedback inhibition. Additional evidence for lithium's antagonistic action on dopamine can be seen in reports of extrapyramidal symptoms induced by lithium therapy (34). These limited clinical trials demonstrated that at least two weeks of treatment was necessary for TD symptom alleviation, even though plasma levels ranged between 0.70 and 1.70 mEq/liter throughout their study period.

Ms. A's toxic picture manifested itself as an organic brain syndrome without any movement disorder. These symptoms as outlined in the clinical course correlated better with the RBC Li^+ levels than with the plasma levels (see figure 1 and figure 2). Lithium plasma levels of 0.53–1.47 mEq/liter were observed from hospital day 4 and thereafter. However, based on criteria published by the University of Southern California pharmacokinetics laboratory (13), Ms. A's RBC Li^+ levels were still considered toxic on day 6 (RBC $Li^+ > 0.60$ mEq/liter) (13). As Ms. A's RBC Li^+ level fell, her organicity improved. It took an additional 5 days for the organicity to abate. Many case reports document this lag period in improvement and explain the phenomenon based on intracellular stores reequilibrating slowly into extracellular fluid (23, 35).

Ms. A exhibited manic symptoms that responded to Li^+ RBC levels within the usual therapeutic range of 0.30–0.40 mEq/liter (16) (figures 1 and 2). Due to the complexity of distinguishing mania from organicity, it was difficult to rate Ms. A or to make generalizations about her early hospital course. Li^+ RBC levels greater than 0.50–0.60 mEq/liter have also been associated with a recrudescence of manic behavior (13, 17). As Ms. A's toxic RBC Li^+ levels decreased, her manic symptoms seemed to decrease. However, as her RBC levels continued to fall, she became increasingly more hyperactive and irritable. Then, as the RBC Li^+ levels increased by day 23, she became more calm and cooperative. It appears that our case has a very narrow "therapeutic window." RBC Li^+ levels greater than 0.24 mEq/liter but less than 0.40 mEq/liter brought her manic behavior under control.

The current literature reports the limited usefulness of lithium in the treatment of neuroleptic-induced permanent dyskinesias. To date no attempt has been made to use RBC Li^+ levels, which correlate better with central nervous tissue lithium levels, as a predictive tool in the treatment of TD. The lag period of 5–10 days that has been cited in the literature as the length of time needed before TD symptom suppression could be observed is the same interval necessary to control manic behavior (7); therefore, RBC Li^+ levels may prove to be a useful tool in the proper assessment of what constitutes an adequate trial of lithium in the treatment of TD as well as mania.

During our contact with Ms. A, TD was first manifested on day 10 of her hospitalization (see figures 1 and 2). Prior to this, her RBC Li^+ levels were appreciably higher and no signs of TD were apparent. In addition, as her RBC Li^+ levels continued to decline the staff noticed marked movement disturbances. These low RBC Li^+ levels coresponded to the period in which the movement disorder was most severe (days 13–21). On day 15, Ms. A's lithium carbonate dose was increased to 1200 mg/day. Her plasma Li^+ levels increased by day 19, but no improvement in TD was observed, in contrast to the absence of any movement disturbance on day 7 when her plasma level was comparable but her RBC level was appreciably higher. Then as her RBC Li^+ level increased (days 21–23) and her plasma Li^+ remained fairly constant, her TD symptoms decreased. In this case, TD symptom suppression required Li^+ RBC levels approximately equal to 0.31 mEq/liter or greater. The presence of TD symptomatology on day 10 (see figure 1), compared to the greatly reduced TD symptomatology on day 23, can be explained based on Ms. A's increased manic behavior and organic symptoms. Crane and associates (36) report that TD symptoms may be intensified by hyperactivity and agitated emotional states; one would then expect that both the manic behavior and the residual organic symptoms on day 10 enhanced the severity of the TD, which necessitated higher RBC Li^+ levels to obtain a therapeutic effect.

In conclusion, Ms. A's TD symptom suppression occurred while her Li^+ plasma levels remained fairly constant (approximately 0.70–0.75 mEq/liter) and her RBC Li^+ levels increased from 0.21 to 0.31 mEq/liter. Also, the RBC/plasma Li^+ ratio increased correspondingly from 30% to 40%. In this patient the RBC Li^+ levels showed a better correlation than plasma levels in predicting the symptomatic relief of TD. In view of our favorable results, we recommend more research with Li^+ RBC determinations in the management of tardive dyskinesia to further evaluate its clinical usefulness.

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Exacerbation of Psychosis by Tricyclic Antidepressants in Delusional Depression

BY J. CRAIG NELSON, M.D., MALCOLM B. BOWERS, JR., M.D., AND DONALD R. SWEENEY, M.D.

The authors describe three patients with delusional unipolar depression whose delusional thinking worsened markedly following administration of tricyclic antidepressant drugs. The patients had met Research Diagnostic Criteria for major depressive episode and had no evidence of schizophrenia or mania. Since tricyclic antidepressants are known to exacerbate psychosis in schizophrenic patients, it is sometimes suggested that the exacerbation of psychotic thinking in depressed patients indicates schizophrenia. The authors suggest that such an exacerbation does not in itself indicate schizophrenia but may occur in patients with an affective disorder who are prone to depressive delusions. The authors discuss the use of antipsychotic medication in this patient group and present a neurochemical hypothesis to explain the interaction of the drug with the illness, which results in exacerbation of psychotic thinking.

TRICYCLIC ANTIDEPRESSANT drugs are known to produce three types of psychotic reactions (1, 2): they can exacerbate psychosis in schizophrenic patients, precipitate mania, or produce a confusional state or toxic psychosis. We will report another clinical syndrome, delusional unipolar depression, in which treatment with tricyclic antidepressant drugs can exacerbate the psychosis.

Delusional unipolar depression appears to be a distinct subtype of unipolar depression. Glassman and associates (3) suggested this because of their observation that delusional depressives have a lower rate of response to tricyclic antidepressants than nondelusional patients. Their review of the literature indicated that previous investigators had also noted a poor response to tricyclic antidepressants among delusional depressives. In a more recent report Quitkin and associates (4) failed to find evidence of a difference in response to imipramine treatment between psychotic and nonpsychotic depressives in a review of data from a study conducted in the early 1960s. The authors suggested that the lack of drug response in previous studies may have resulted from inadequate dosage or duration of drug treatment. It is possible that the difference in findings reported by Glassman and associates and Quitkin and associates is related to the method of selection of patients. In the latter study psychotic patients were selected on the basis of a scale rating of perceptual distortion. It is not clear from the report whether these patients met the criteria for delusional thinking used by Kantor and Glassman (5) or whether some of the patients only had exaggerated depressive concerns.

The question of inadequate dosage has been addressed at least in part in other recent studies (6–8), which have noted lower rates of response in the delusional unipolar patient group even when delusional patients were given a higher dosage of tricyclic (imipramine, 300 mg) (6) or when drug plasma levels were obtained to ensure an adequate therapeutic drug level (7, 8). On the basis of this difference in pharmacologic response it has been argued that delusional unipolar depression may represent a distinct subtype of depression. The three delusional depressives we will describe not only failed to respond to tricyclic antidepressants but suffered an exacerbation of their psychosis after receiving these drugs.

The three patients met the Research Diagnostic Criteria (RDC) for major depressive episode (9). None of the patients had a history of mania. Although they did show evidence of delusional thinking, this appeared to be part of the depressive syndrome. The patients did not show RDC evidence of schizophrenia. The patients were hospitalized on a research unit where daily behavioral ratings (10) were available. All patients were kept drug-free during the first two weeks of hospitalization.

CASE REPORTS

Case 1. Ms. A, a 47-year-old unmarried secretary, was admitted for her first psychiatric hospitalization with the complaint that she could not think and was losing her mind. The patient's difficulties had started 2 years previously after the death of her mother. At first Ms. A experienced withdrawal, self-consciousness; and anxiety. In the 2 months before admission she lost her job, her appetite decreased, she lost weight, felt weak, and slept poorly, her social withdrawal increased, and she felt that her mind was blank and that she

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was stupid. Ms. A had had no previous psychiatric treatment. However, at age 35 she had been sufficiently depressed that her ability to work was impaired and her social functioning decreased. On admission she appeared depressed, tense, and mildly suspicious. She thought people had special ideas about her and were going to hurt her. However, after she adjusted to the hospital she was less frightened by these ideas. During the first 2 weeks, which were drug-free, the patient continued to look moderately depressed. At this point she was started on amitriptyline in dosages that were increased to 150 mg over several days. During the first 2 weeks of amitriptyline treatment her depressed mood, irritability, and withdrawal continued. During the third week she noted slight improvement in mood, but her paranoid thinking worsened and was rated as extremely severe. She thought that other patients knew that she was "guilty" and that they were going to dissect her or cut her up. She thought her sister might be hurt and wrote her a note telling her not to visit. There was no change in her orientation, concentration, or memory. This increased paranoid thinking persisted over the next 5 days but responded quickly to trifluoperazine, 8 mg. Treatment continued with trifluoperazine and amitriptyline, 200 mg. Ms. A's mood improved considerably and after 5 weeks on this combination of medications she was considered asymptomatic.

Case 2. Mr. B, a 51-year-old married truck driver, was admitted for his first episode of serious psychiatric illness. His symptoms had started 3 years previously and appeared related to his being placed on part-time status at work. He began experiencing anxiety, apathy, loss of energy, and feelings of worthlessness. He had a brief period of thinking he might be harmed at work but continued to work part-time. In the 6 months before his admission he began to believe that he had cancer and was going to die. In the 2 months before admission he lost 25 pounds, had frequent crying spells and early morning awakening, experienced no pleasure in his activities, and felt helpless and hopeless. He took an overdose of analgesic medication and was hospitalized. On admission Mr. B was markedly depressed and lethargic. There was no evidence of delusional thinking at this point. However, at the end of a 3-week drug-free period evidence of mild paranoid thinking was noted. Amitriptyline was started in dosages increased by 50 mg/day to a maximum of 250 mg. By the fourth day of amitriptyline administration Mr. B's agitation became extremely severe. His paranoid thinking had increased considerably, and he thought he and his family would be killed or harmed. Perphenazine, 24 mg/day, was begun. The patient's level of agitation and delusional thinking diminished rapidly over the next several days. Three weeks after he received the combination of perphenazine, 24 mg, and amitriptyline, 250 mg, Mr. B was still moderately depressed. As a result, the amitriptyline was discontinued and he was started on imipramine, which resulted in noticeable improvement in his depressive symptoms.

Case 3. Ms. C, a 64-year-old woman, had a history of depression beginning at age 55. She had had three hospitalizations early in the course of her illness. Two months before admission her sleep decreased and she experienced early morning awakening. She began to ruminate about her mother and her past. One week before admission she began to experience delusional thoughts that her food was poisoned. She stopped eating and working, stopped caring for her apartment, and withdrew from friends. On admission Ms. C described feelings of guilt, self-reproach, and hopelessness. She appeared agitated but had little spontaneous speech. After an initial drug-free period she was treated with nortriptyline, 150 mg, for 26 days. No improvement was noted, and she continued to appear severely depressed. Nortriptyline was discontinued and imipramine was started in dosages that were gradually increased to 200 mg. Eight days after imipramine was initiated Ms. C became markedly paranoid and extremely agitated. The imipramine was discontinued and she was given 9 ECT, with good response.

DISCUSSION

Previous reports have described a low rate of response among delusional unipolar depressives to tricyclic antidepressants, but exacerbation of psychosis as a result of such treatment has been described rarely. An exception is the observation by Sandifer and associates (11), who reported that tricyclic antidepressant drugs aggravate delusional depression. The three cases we have described demonstrate that tricyclic antidepressant drugs appear to aggravate delusional thinking. Although this may have been merely a worsening of the clinical course of the illness, the temporal relationship suggests that the increase in delusional thinking is a response of the underlying illness to treatment with tricyclic medication. The delusional thoughts that are pharmacologically provoked are consistent with the existing depressive syndrome. Typically, the delusional thinking consists of thoughts of guilt coupled with a paranoid fear that others, knowing of this guilt, will harm the patient. There is no evidence of a toxic delirium or clouding of consciousness. Even during the heightened period of delusional thinking the patient appears neither manic nor schizophrenic. According to Research Diagnostic Criteria, the patient would be designated as having a major affective disorder, psychotic.

The question of whether the development of psychosis during tricyclic treatment in itself suggests schizophrenia is an interesting one. Certainly, the exacerbation of psychosis in schizophrenic patients given tricyclic antidepressants has been well documented in the literature (12–18). Two reports describe the development of psychosis in depressed patients treated with tricyclic drugs. Newman and Fischer (19) described three depressed patients who became overtly psychotic after receiving tricyclic drugs, and they concluded that these medications can push "schizophrenic" patients into psychosis. Two of the patients, however, were 45-year-old men with a depressive syndrome. Apparently they were considered schizophrenic on the basis of the psychosis that developed after tricyclics were administered. In another report Baldessarini and Willmuth referred to the "common clinical impression that latent psychosis may be uncovered during amitriptyline treatment for depression" (20). Their discussion of two cases was interesting in that it described the more florid delusional symptoms as being depressive in nature and consistent with the patient's depressive syndrome. The authors did not sug-

gest these patients were schizophrenic but noted that they might be considered "prepsychotic." Certainly, it is well recognized that severely depressed patients may have depressive delusions, typically with themes of guilt or somatic concern and in some cases associated with paranoid thinking. Exacerbation of these depressive delusions by tricyclic drugs does not in itself indicate schizophrenia but rather a worsening of the affective disorder.

Recognition that tricyclic antidepressants can exacerbate such delusional thinking in depressed patients may help to avoid an inappropriate diagnosis of schizophrenia and also has important treatment implications. We described previously the successful treatment of 12 of 13 patients who had delusional unipolar depression with a combination of tricyclic antidepressants and antipsychotic drugs (21). Exacerbation of delusional thinking was not noted when patients received a sufficient dosage of antipsychotic drug. Implementation of such treatment first requires the identification of patients at risk. It has been our experience that patients who have developed florid delusional thinking following tricyclic treatment have usually presented some evidence of delusional thinking prior to treatment, although it may not have been prominent. Cases one and two in this paper illustrate our experience. A history of episodes of delusional depression or of tricyclic-induced psychosis might also suggest treatment with an antipsychotic-antidepressant combination.

Although a neurochemical understanding of the aggravation of delusional thinking in this diagnostic group is speculative, there is suggestive evidence that patients with this syndrome may have alterations of both dopamine and norepinephrine systems (22), that tricyclic antidepressants may facilitate dopamine systems through an intermediate effect on norepinephrine (23, 24), and that psychotic depressive patients receiving tricyclics may show an increase in CSF homovanillic acid, the major metabolite of dopamine (25). Thus, it is possible that tricyclic antidepressants may aggravate delusional thinking in this patient group through a facilitative effect on dopamine systems.

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A Possible Cardiovascular Effect of Lithium

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Heart rate rises associated with performance of mental arithmetic tasks were compared in 22 euthymic manic-depressive patients receiving lithium carbonate and 17 drug-free normal controls. The lithium-treated subjects showed a markedly lower heart rate response to the mental arithmetic stimulus. No effect was observed on basal heart rate. The results are consistent with lithium's reported inhibition of noradrenaline-induced rises in adenylate cyclase activity but lack of inhibition of basal adenylate cyclase activity. The biochemical and psychophysiological findings may parallel lithium's unique ability to calm excited behavior without sedating normal behavior.

BIOCHEMICAL THEORIES of lithium's mode of action include the possibility that lithium inhibits noradrenergic adenylate cyclase in the brain (1), thus reducing the functional effect of the hypothesized excess of noradrenaline in mania (2). Supporting this possibility is the marked inhibitory effect of lithium on peripheral β-adrenergic adenylate cyclase in man (3). Since lithium has such a marked effect on the peripheral noradrenergic adenylate cyclase, one would expect therapeutic doses to have cardiovascular effects. Fann and associates (4) found the pressor response to noradrenaline reduced by 22% in subjects taking lithium, but a review of the literature did not reveal reports of cardiac slowing or hypotension resulting from lithium use (5). In contrast, hypotensive effects are regularly seen with some phenothiazines and are ascribed to their α-adrenergic blocking effect (6). Since Ebstein and associates (3) reported inhibition of the β-adrenergic adenylate cyclase by lithium, we decided to test its effect on a specifically β-adrenergic cardiovascular function.

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The "mental arithmetic test" is a method of causing consistent, temporary heart rate rises in humans (7–9). The adrenergic receptor involved in heart rate is known to be of the beta type (10), and changes in human heart rate have adrenergic as well as vagal determinants (9, 11). These β-adrenergic receptors are thought to function via adenylate cyclase (3). We therefore chose the mental arithmetic test as a method of inducing heart rate rises in order to compare such rises in lithium-treated and untreated subjects. We predicted that lithium-treated subjects would show a much smaller heart rate rise in response to mental arithmetic tasks than normal subjects. Such a difference would support the concept that lithium in vivo is a physiological inhibitor of noradrenergic function, perhaps in the brain as well as in the heart.

METHOD

The experimental subjects were manic-depressive patients at the lithium clinic of the Jerusalem Mental Health Center. Subjects who had been treated with other drugs in the week prior to examination or whose lithium levels were at toxic or below therapeutic levels were excluded. The mean plasma lithium level of the patients at the determination closest to the time of study (usually later on the same day) was .79 mEq/liter (SEM=±.40). Patients with physical disease, especially cardiovascular, diabetic, hypertensive, or organic brain disease, were excluded. All patients were free of psychiatric symptoms or close to this criterion at the time of study, that is, they were in a euthymic state. Controls were employees of the Jerusalem Mental Health Center who had taken no drugs in the preceding week and who suffered from no physical or mental illnesses. The lithium-treated group included 10 men and 12 women, with a mean age of 42.4 (range, 21 to 63). Controls included 7 men and 10 women, with a mean age of 42.3 (range, 26 to 66). Seven patients had not finished high school, 7 had finished high school only, and 8 had had some education beyond high school. None of the control subjects had not finished high school, 5 had finished high school only, and 12 had had post-high school education.

The mental arithmetic tasks were similar to those of Lacey and associates (7) and Tursky and associates (8). Each of the four tasks demanded mental effort and concentration; to avoid introducing anxiety as a possible factor, no time limits were set.

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Patients and control subjects were introduced to the experimenter in a quiet room by the clinic director (R.H.B.). They were told in advance that they were to be given a test of heart rate. After 3–10 minutes to acquaint themselves with the procedure, subjects were attached to a middle finger photoelectric transducer. The transducer was connected to an analog chart recorder that continuously recorded heart rate at each beat. The subjects were asked not to talk during the experimental trials; all responses were written with the unattached hand.

The first task was to count backward mentally from 100 to 50 and to signal "finish" with the free hand when done. The second task consisted of 23 simple addition problems, in which the subject was asked to add 4 to one number and subtract 3 from the next number in an alternating fashion. The third task comprised 15 multiplication problems at two levels of difficulty. The "hard" set was given to high school graduates and the "easy" set to nongraduates. The fourth task was a modification for use in Hebrew of Lacey and associates' "reverse spelling task," during which the experimenter read aloud 17 common words and the subject was asked to reverse the letters of the word and write down the result.

Each task was presented on a sheet of paper that the subject held on his knees. The answers were written on the same paper. Between each task the next task was explained; this interval was about 50 seconds. After the last task the subject was asked to sit quietly for 30 to 60 seconds, and a "baseline heart rate" was recorded when heart rate ceased to decline more than one beat per minute over a 15-second period. The total procedure took from 10 to 20 minutes for each subject.

Heart rates for each task period were determined by counting 15 seconds from the onset of the task (on the recorded tape) and then averaging the following three instantaneous heart rates at 5-second intervals. Each subject thus had an average heart rate for each of the four tasks. Heart rate in the final rest period was calculated as the mean of the three instantaneous heart rates at 5-second intervals after heart rate ceased to decline more than one beat per minute over a 15-second period. Data were analyzed by using the final rest period as baseline and calculating increases by subtracting the subject's "resting" heart rate from the heart rate associated with each task.

RESULTS

Table 1 shows the rise in heart rate with the various mental arithmetic tasks in the lithium-treated and untreated subjects. There is no significant difference between the two groups in "rest period" heart rate. The lithium-treated subjects show a consistently smaller increase in heart rate with each task than the control subjects.

Analysis of the results by sex show no differences in the rise between sexes. Splitting the lithium-treated group into 15 patients with high school education or greater and 8 patients with less than high school education revealed no effect of education on the heart rate response to any of the tasks. There was no difference in heart rate response between those who received the "hard" and those who received the "easy" versions of task three. There was no difference in the percentage of correct answers between the lithium-treated and untreated groups; all subjects gave over 85% correct responses in every task.

TABLE 1
Heart Rate Increases[a] During Mental Arithmetic Tasks In 22 Lithium-Treated and 17 Untreated Subjects

Task	Lithium Group		Untreated Group		Significance[b]
	Mean	SEM	Mean	SEM	
Counting backward	3.5	1.7	7.4	1.7	p=.07
Addition problems	6.8	1.0	12.9	1.7	p<.05
Multiplication problems	7.3	1.2	12.8	1.8	p<.05
Reverse spelling	4.4	.7	11.2	1.8	p<.05

[a]In beats per minute over mean resting heart rate (lithium group=78.4±2.4 SEM; untreated group=80.7±2.9 SEM; difference is not significant).
[b]One-tailed Student's t test.

DISCUSSION

These results suggest that treatment with lithium inhibits the cardioacceleration induced by various mental arithmetic tasks. This result is consistent with the interpretation that lithium inhibits noradrenergic adenylate cyclase and thus inhibits the function of the sympathetic nerves to the heart. Misu and associates (12), in studying isolated rabbit sympathetic nerve-heart preparations, found that lithium perfusion blocked the heart rate response to electrical stimulation of the nerve. It is of interest that in our experiment the effect of lithium is seen only on the cardioacceleratory response and that lithium-treated subjects do not show a reduction in baseline heart rate. That is, lithium seemed to block activation without affecting baseline activity. This may explain why lithium can block mental arithmetic-induced increases in heart rate and the pressor response to infused noradrenaline (4), yet does not cause cardiovascular side effects (5) due to interference with "basal" sympathetic activity. A similar phenomenon may also be apparent on the biochemical level, where lithium inhibits the noradrenaline-induced activation of adenylate cyclase but does not reduce basal activity levels of the enzyme (1, 3, 13). If such mechanisms occur similarly in brain noradrenergic systems that affect mood and behavior, the paradox of lithium's calming effect in mania but absence of a sedating effect in normal or euthymic individuals could perhaps be understood (14).

The results of this study might have significance for understanding lithium effects beyond the use of the

cardiac sympathetic nerve as a peripheral model for what might occur in brain noradrenergic systems. The James-Lange theory of emotion (15) suggests that peripheral arousal contributes importantly to the brain's perception of emotion. If the pressure of thinking and speech in mania causes cardiac acceleration, lithium treatment would tend to block this effect. A vicious cycle of central activation, peripheral arousal, and feedback central activation might thereby be interrupted.

One possible weakness of our study is the fact that all of the lithium-treated subjects were manic-depressive patients, whereas all untreated subjects were psychiatrically normal. Attempts to increase the comparability of the two groups included use of only those patients who were in a euthymic or nearly normal state; choice of an untimed mental arithmetic test, which is probably less affected than timed tests by anxiety, personality, or other extraneous factors (7, 8); statistical analysis to show that educational level had no effect on heart rate response; and demonstration that both groups had equivalent degrees of attentiveness, as measured by proportion of correct responses. The present results are therefore consistent with the theory that lithium is an inhibitor of noradrenergic neurotransmission, and especially of noradrenergic activation beyond basal levels.

Effects of lithium on heart function in patients with cardiac disease have recently been reviewed (16). Whereas some cardiac arrhythmias may be exacerbated by lithium, other arrhythmias, including paroxysmal atrial tachycardia, may be ameliorated (17). The present results suggest the value of controlled study of the prophylactic use of lithium for paroxysmal atrial tachycardia, especially in those cases in which the heart is otherwise healthy and the cardiac nerves (due to psychosomatic factors) may initiate the attacks of elevated heart rate.

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The Quantitative Measurement of Depression and Anxiety in Male Alcoholics

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Depression and anxiety have been reported to be commonly associated with alcoholism. Most attempts to clarify this relationship have suffered from a patient selection bias in that only those alcoholics who sought treatment were studied. The authors performed quantitative measurements of depression and anxiety in a group of 48 men referred for treatment solely on the basis of excessive drinking. The prevalence of depression and anxiety as measured by both the Zung and Hamilton scales was lower than that shown in previous studies. The results of this study indicate that clinical depression and anxiety are not common problems in young, healthy male alcoholics.

APPROXIMATELY NINE MILLION Americans suffer from alcoholism, a progressive illness with early, middle, and late stages (1) characterized by psychological, social, and physical symptoms. Both depression and anxiety are commonly seen among alcoholics (2–4), but their interrelationships are unclear. Menninger (5) conceptualized alcohol addiction as a chronic progressive suicide. Alcoholics, like depressives, have a higher incidence of suicide than the normal population (1, 6, 7). Some reports note that severe drinking leads the alcoholic into a precarious life position beset with many real object losses that result in the emergence of depressive affects (6–8). Other studies stress the antidepressant psychopharmacologic effects of acute alcohol ingestion (9–11). Investigators who study neuropharmacologic effects of alcohol suggest that chronic alcohol ingestion leads to depletion of neurotransmitters, thereby causing depression (12–15). A genetic basis for alcoholism and a genetic relationship between alcoholism and affective disorder have also been proposed (4, 16, 17).

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Alcoholics who seek treatment and suffer from alcohol-related physical illnesses and/or psychosocial problems usually comprise the study groups reported in the literature. Our sample was referred solely for problems of excessive drinking without the complications of associated medical and/or significant psychiatric problems. Studying such samples may help to clarify the relationship between alcoholism, depression, and anxiety.

METHOD

The study was conducted on the Alcohol Rehabilitation Unit of the National Naval Medical Center, Bethesda, Md. All patients admitted to this 15-bed unit completed a six-week structured program, which included mandatory participation in Alcoholics Anonymous. These patients, all active duty male military personnel, were referred by supervisors, physicians, or alcohol counselors. Inclusion criteria were as follows: 1) age between 20 and 45 years; 2) no significant medical or psychiatric problems; 3) a score of more than 6 on the Michigan Alcoholism Screening Test, a widely used 25-item yes-no questionnaire (18, 19); and 4) no borderline or psychotic condition or organic brain syndrome, as determined by the Research Diagnostic Criteria (RDC) (20, 21) and by psychiatric, psychological, and medical assessments, when indicated.

The 48 subjects had a mean age of 32 (SD=7.9). There was a family history of alcoholism in 70.8% of the sample. Lineal family history was positive for major affective disturbance in 13.8% and for suicidal behavior in 16.2%. The duration of excessive use of alcohol (more than 2,000 ounces of absolute alcohol per year) ranged from less than 1 year to 17 years (mean=5±4.5). Eight patients experienced mild withdrawal symptoms. Within 48 hours of admission, the patients completed the Zung Self-Rating Depression Scale (SDS) and Self-Rating Anxiety Scale (SAS) (22–24). Raw scores were converted to index ratings based on 100. Within 72 hours of admission, the patients were rated on the Hamilton Depression Scale (HDS) and the Hamilton Anxiety Scale (HAS) (25–27). All rating scales were completed again at 3 weeks and at 6 weeks after admission. Some patients did not complete all of the self-rating scales at the designated rating times because the treatment program schedule made them unavailable. Likewise, some patients were

TABLE 1
Depression and Anxiety Scores at Three Periods for Alcoholics in Treatment

Scales	Baseline			3 Weeks			6 Weeks[a]		
	N	Mean	SEM	N	Mean	SEM	N	Mean	SEM
Hamilton Depression Scale	48	10.0	0.9	41	7.7	0.8	33	6.3	0.7
Hamilton Anxiety Scale	48	5.8	0.6	41	4.9	0.5	33	4.4	0.8
Zung Self-Rating Depression Scale	46	44.7	2.0	46	40.0	1.8	44	36.7	1.4
Zung Self-Rating Anxiety Scale	46	43.8	1.6	45	37.7	1.4	44	36.5	1.2

not available for interview at all of their designated interview points. This is reflected in the varying Ns as seen in table 1.

RESULTS

None of the 48 patients scored above 25 (the cutoff point for severe depression) on the baseline HDS, and only 3 scored above 22 (6.3% of the group). Scores of 22–25 are indicative of depression in inpatients (25, 28). There are currently no standardized values for HAS. Mean HDS and HAS baseline, 3-week, and 6-week scores are summarized in table 1. The mean values for the SDS and SAS are also summarized in table 1. At baseline 34.8% of the group scored above 50. Zung cited a score of 50 as indicating depressive affect and noted the following means: 64 for inpatients, 33.8 for normal controls. By 6 weeks only one individual had a score above 50 and the group mean SDS was 36.5.

As all raw data were approximate values obtained from ratings, Spearman rank-order correlation analyses were applied to the data. There were significant HDS-SDS and HAS-SAS correlations at baseline and 3 weeks, but neither correlation was significant at 6 weeks. The HDS and HAS and the SDS and SAS were significantly correlated ($p<.01$) at all three rating points. No significant correlations were found between any rating scale scores and age, duration of excessive drinking, or amount of alcohol consumed.

DISCUSSION

The prevalence of depression and anxiety in this study is lower than that found in previous studies (2, 29–33). Except for 3 patients, the initial HDS study group scores were all lower than the cutoff range used to identify depressives. More than a third (34.8%) of the group scored above 50 on the baseline SDS, but scores similar to these were reported by Zung and associates (34) for personality disorder, anxiety reaction, and transient situational disturbance. The results of this study indicate that depression and anxiety in the clinical range are not common among young, healthy alcoholics. Thus, our findings are not consistent with the hypothesis that alcoholics are basically depressed individuals who drink to alleviate their dysphoria. A possible explanation for the low incidence of depression we found is that few of these patients had suffered alcoholism long enough to result in major life losses or that they simply had a lower "loss risk" because of their age. Holmes and Rahe (35) have shown that major life changes are associated with an increased incidence of physical and psychiatric problems. Depression is more common in older people in general (36) and may be more common in older or later stage alcoholics who have suffered more medical and/or psychosocial consequences of alcoholism and/or age. Similarly, if depression secondary to alcoholism were related to chronic depletion of neurotransmitter stores, this study group would be at less risk than an older sample. No correlation between depression and length of drinking was noted; however, the age distribution within this study group would bias against such a finding.

The present study raises the possibility that previous studies reporting a high incidence of depression in alcoholics (2, 29–33) used biased samples of alcoholics, e.g., those in need of medical or psychiatric hospitalization or those with coincident conditions that would lead to spurious positive correlations. Partial confirmation for these possibilities comes from a study in which Woodruff and associates (37) compared alcoholics who seek psychiatric treatment with those who do not. The nature of alcoholism in the two groups was similar. Depressive symptoms and the presence of secondary affective disorder clinically distinguished the two groups. The alcoholic who does not seek treatment is probably more similar to the group we studied and, perhaps, to the majority of alcoholics in the general population.

The positive correlation between the HDS and SDS is similar to the finding of Brown and Zung (38) for these two rating instruments. The lack of correlation at the 6-week comparison probably has no clinical meaning; values on both scales were quite low and the mathematical corrections for tied values in the Spearman rank-order correlations were much more extensive than would be the case with broader ranges of values. The entire range of HDS-SDS values for this population was somewhat narrow and could not have been used to reassess the earlier finding (38) that HDS scores increase at a slower rate than SDS scores among severely depressed patients. In the present

study, a higher rating for depression was seen on the SDS than the HDS. The cutoff point of 50 for the SDS was derived from an outpatient population with mixed psychopathology. The HDS was standardized using an inpatient population of depressed patients.

CONCLUSIONS

Previous studies of alcoholics have found a high incidence of co-existing depression, and this finding had led to a number of hypotheses. Relatively little attention has been paid to possible patient selection bias. Often, such studies relied on patients who were in urgent need of medical and/or psychiatric care, although such patients may not be representative of alcoholics in the general population. The patients in our study were selected by others as being suitable for rehabilitation from alcohol abuse and did not have significant medical or psychosocial problems. Depression and anxiety were not common problems for this group. Further studies, with close attention to methods of patient selection, are necessary to clarify the relationship between alcoholism, depression, and anxiety.

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Bipolar Illness: Factors in Drinking Behavior

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Alcohol use and abuse was investigated in 73 patients with bipolar I manic-depressive illness who were attending a lithium clinic. Alcohol-related problems were identified in 7 of the male and none of the female patients. Family data revealed a higher morbid risk for alcoholism among relatives of patients with drinking problems than among relatives of patients without drinking problems. Morbid risks for affective disorder for these two groups of relatives were similar, suggesting that alcoholism among relatives of patients with bipolar illness is not solely "genotypically" related to the affective disorder.

THE PURPOSE of the study described in this paper was to investigate alcohol use among a well-defined group of manic-depressive (bipolar I) patients and to determine if alcohol-related problems in these patients were associated with alcoholism in their relatives. Increased alcohol intake during manic and depressive states is often observed. This subject was reviewed by Freed (1), who cited several reports indicating increased alcohol intake during depression. Morrison (2) studied the drinking behavior of patients admitted to a Veterans Administration hospital for mania and found that a high percentage of such patients were heavy drinkers, although a secondary diagnosis of alcoholism among these patients was no more frequent than among patients with other psychiatric disorders admitted to the same hospital.

Evidence for a genetic factor in the etiology of alcoholism was recently reviewed by Goodwin (3). Goodwin described a subgroup of alcoholics whose illness had an early onset and who had strikingly positive family histories of alcoholism. Although an increased risk for alcoholism is frequently found among relatives of bipolar patients (4, 5), the relationship of alcoholism in such families to drinking problems among the bipolar patients themselves has not been studied.

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METHOD

The subjects studied were attending the lithium clinic of the New York State Psychiatric Institute. All of the patients met diagnostic criteria for primary affective disorder (6), and all of the patients were subclassified as having bipolar I affective disorder because they had been previously hospitalized for mania (5). Patients voluntarily consented to participate in the study, which was conducted over a four-month period. All bipolar I patients who attended the clinic during the study interval participated. The selection criteria for treatment of patients in the clinic excluded from the study patients who had severe drinking or drug problems. For example, patients with bipolar illness who had histories of alcohol addiction resulting in treatment for alcoholism were screened out at the initial evaluation.

The patients were asked to complete a questionnaire regarding their current and past drinking behavior. This form included all items pertinent to the diagnosis of alcoholism in a structured interview (7) based on the criteria of Feighner and associates (6). Responses to the questionnaire were reviewed with the patient by one of us (B.M.H.) and were also confirmed by review of responses to similar questions recorded during the structured interview.

A subject was considered to have (or to have had) drinking problems if he had three or more alcohol-related symptoms, which included social (job, marriage, legal), psychological, and medical problems that a physician and/or the patient attributed to alcohol. The specific symptoms examined were derived from the criteria of Feighner and associates (6) for alcoholism. None of the patients met these criteria for alcoholism.

Family histories were obtained through systematic interviews of the patients and, in many cases, through structured interviews of relatives. Only data regarding parents and siblings were included. The total number of relatives studied was 371. Of these, 310 were still living; 154 were interviewed directly.

Diagnosis of alcoholism in a relative was made through blind review of the structured interview records using the criteria of Feighner and associates (6) or by information from the proband or other relatives.

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The problems related to drinking in these latter cases also had to be severe enough to result in social, medical, or psychological complications. A relative was diagnosed as alcoholic through family history only if the history was confirmed by at least two family members. Family history data were determined independently of the alcohol status of the patient. Morbid risk data for alcoholism were calculated using the ages at risk from Amark (8).

Family history of affective disorder was determined from blind review of the structured interviews or family history data, and affectively ill relatives were diagnosed according to the criteria of Fieve and Dunner (9). Morbid risks for bipolar and unipolar illness were calculated by considering relatives who had not been treated for primary affective disorder as not psychiatrically ill and by using the ages at risk for bipolar II illness and unipolar illness previously determined by a review of age-of-onset data from our patients (D.L. Dunner, unpublished data).

RESULTS

There was good agreement between the reports of drinking behavior from the questionnaire and the structured interview. Of the 73 patients studied, 36 were women and 37 men; 27 of the women and 28 of the men drank socially. Alcohol-related problems were identified in 7 of the men and none of the women. This difference was statistically significant ($\chi^2=5.48$, $p<.02$). The differences in mean ages of the groups (women, 43.9±16 years; men without drinking problems, 48.8±15.3 years; men with drinking problems, 44.6±14.8 years) were not statistically significant.

Among the 7 patients with drinking problems, the alcohol-related symptoms identified most frequently were missed work, difficulty with family or friends, driving problems, problems with the police, blackouts, and physical violence while drinking. Each of these symptoms except physical violence was reported by 4 patients; physical violence was noted by 3 patients.

Morbid risk for alcoholism was significantly higher in relatives of patients who had drinking problems than in relatives of patients without drinking problems (see table 1). Furthermore, relatives of men without drinking problems showed a significantly lower morbid risk for alcoholism than relatives of women patients. There was no significant difference when relatives of all male and all female patients were compared for alcoholism. Thirteen of the relatives of women were identified as alcoholic. Six of these relatives were male and 7 were female. Two of the 3 alcoholic relatives of males without drinking problems were male, as were all 5 alcoholics related to male patients with drinking problems.

Morbid risks for affective disorder among relatives were comparable in the patient groups (see table 2). Overall, 17 of 164 male relatives of patients without drinking problems had affective disorder as compared to 2 of 22 male relatives of patients with drinking problems. Similarly, 39 of 174 female relatives of patients without drinking problems had affective disorder as compared to 2 of 11 female relatives of patients with drinking problems.

TABLE 1
Family History of Alcoholism of 73 Bipolar I Patients

Patient Group	Relatives: Number Studied	Relatives: Number at Risk	Relatives: Morbid Risk %
Men with drinking problems (N=7)	33	20.2	24.9[a]
Men without drinking problems (N=30)	151	101.8	2.8
Women (N=36)	187	122.5	10.2

[a]Significantly higher than relatives of men without drinking problems ($\chi^2=9.87$, $p<.01$).

DISCUSSION

We found that 7 of our 37 male patients (approximately 19%) had histories of problems related to alcohol. Other studies of alcoholism and/or alcohol-related problems among manic-depressive patients reveal incidences of alcoholism of 20%–40% (1, 2). Many of these studies were done on patients who were diagnosed as having affective illness by criteria less rigorous than those applied to our sample. Since our clinic sample was prescreened to exclude alcoholics, our data suggest that there would be considerable overlap of alcohol-related problems in a bipolar I population not prescreened to exclude such problems.

The morbid risk for alcoholism is increased among relatives of patients with alcohol-related problems as compared to relatives of patients without such problems. Although environmental factors are implicated in the etiology of both alcoholism and affective disorder, recent data suggest the importance of genetic factors in the development of these illnesses (3, 4, 10). Thus it is likely that the alcoholism found among relatives of patients with drinking problems is occurring on a genetic basis or, conversely, that patients who have family histories of alcoholism are likely to have symptoms of alcoholism.

There has been some controversy regarding whether alcoholics found among relatives of bipolar I subjects should be considered to have the affective "genotype." Our data suggest that most of the alcoholism seen in relatives of male bipolar I patients may be genetically unrelated to the affective disorder, since the morbid risks for affective disorder were similar among relatives of patients with and without drinking problems. Morrison (2) also interpreted his data as indicating that alcoholism and primary bipolar affective disorder may be independently caused. His population was 90% male. Further support of the hypothesis that alcoholism and primary affective disorder may be genetically unrelated could be shown by studying the rel-

TABLE 2
Primary Affective Disorder Among Relatives of Bipolar I Patients With and Without Alcohol Problems

Patient Group	Male Relatives			Female Relatives[a]		
	Number	Bipolar Illness Morbid Risk %[b]	Unipolar Illness Morbid Risk %[b]	Number	Bipolar Illness Morbid Risk %[b]	Unipolar Illness Morbid Risk %[b]
Men with drinking problems	22	9.6 (1/10.4)	8.6 (1/11.6)	11	15.7 (1/6.4)	14.4 (1/7.0)
Men without drinking problems	74	9.1 (4/44.0)	8.2 (4/48.7)	77	11.3 (6/53.0)	17.2 (10/58.2)
Women	90	12.2 (6/49.3)	5.4 (3/55.3)	97	14.5 (8/55.0)	21.8 (15/68.8)

[a]There were no significant differences among male or female relatives for the different patient groups.
[b]Numbers in parentheses refer to the ratio of number ill to number at risk.

atives of a large population of women with bipolar illness who also have drinking problems. Unfortunately, no such women were found in this sample. The hypothesis would predict that relatives of such women should have elevated morbid risks for alcoholism but similar risks for primary affective disorder as compared to the results obtained from the women patients in this study. Data from other centers regarding this problem would be of interest.

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Are All Recently Detoxified Alcoholics Depressed?

BY MARTIN H. KEELER, M.D., C. INGA TAYLOR, M.D., AND WILLIAM C. MILLER, M.D.

Reports of the prevalence of depression among alcoholics vary from 3% to 98%; the authors attribute this variation to the use of different diagnostic criteria. They used clinical diagnosis, the Hamilton Depression Rating Scale, the Zung Self-Rating Depression Scale, and the MMPI to diagnose 35 men recently withdrawn from alcohol. The percentages of depression diagnosed were 8.6%, 28%, 66%, and 43%, respectively. The authors point out that the Hamilton, Zung, and MMPI scales are not diagnostic of depression in themselves and that accurate diagnosis of depression in alcoholics will reduce inappropriate treatment of nondepressed alcoholics and ensure careful treatment of the truly depressed.

WE STUDIED the prevalence of depression in 35 men recently withdrawn from alcohol, using the following criteria: clinical diagnosis, the Hamilton Depression Rating Scale (1), the Zung Self-Rating Depression Scale (2), and the Minnesota Multiphasic Personality Inventory (MMPI). The study was designed to compare the effects of using different criteria to diagnose depression among alcoholics rather than to determine the absolute prevalence of depression among alcoholics, which would require a sample stratified so as to include different settings and populations.

The results of using different criteria are important for two reasons: 1) reports of the prevalence of depression among alcoholics vary from 3% to 98%, which obviates any understanding of the relationship between them, and 2) reports of high rates of depression among alcoholics have in the past led to the use of antidepressant drugs as treatment for alcoholism independent of any specific evidence of depression. The diagnosis of depression among alcoholics has been accepted as a reason to use tricyclic antidepressants and is of immediate clinical, in addition to theoretical, relevance. This has been only slightly modified by using criteria for depression that would apply to virtually the entire population of alcoholics.

These trends have run their course; however, the availability of lithium as a treatment for mania and perhaps for depression, as a prophylaxis against manic and depressive episodes in patients who have bipolar illness, and as a possible prophylaxis against depressive episodes in unipolar depressive illness could readily lead to the administration of lithium to almost all alcoholics. This could occur if alcoholism were equated with depression or if overinclusive diagnostic criteria were used.

PREVIOUS ESTIMATES OF THE PREVALENCE OF DEPRESSION IN ALCOHOLICS

Winokur and associates (3), using data from structured interviews as the criteria, reported a figure of 3% primary affective disorder among males treated for alcoholism. Shaw and associates (4), accepting as criteria elevation in the Zung Self-Rating Depression Scale (2), the Beck Depression Inventory (5), or the depression scale of the MMPI reported a prevalence of depression of 98% among patients requesting treatment at a halfway house. Weingold and associates (6) reported that 72% of those admitted to a state hospital alcoholism program could be diagnosed as depressed by the Zung scale. Rosen (7) reported the group mean of the depression scale of the MMPI to be significantly elevated for a group of male patients admitted to the UCLA Alcoholism Clinic and for a group of skid-row alcoholics.

Three studies (4, 8, 9) have evaluated the use of tricyclic antidepressants alone or with benzodiazepines as treatment for alcoholism, although in only one of the studies (4) were changes in depression systematically ascertained.

METHOD

Our subjects were male alcoholics consecutively admitted to the alcoholism treatment service of a Veterans Administration hospital. Of 39 patients admitted, 35 completed the protocol. All were withdrawn from alcohol by the use of decreasing doses of chlordiazepoxide or diazepam. All of the diagnostic methods were applied between the fifth and seventh days after admission. No other psychotropic medication was administered before the completion of evaluation.

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Clinical diagnoses based on *DSM-II* criteria were made at case conferences in which the patients were interviewed in the presence of the attending staff. The Hamilton Depression Rating Scale (1) was administered by a psychiatrist. This test is widely used in studies of depression, but Hamilton stated that the scale should not be used to diagnose depression, although it can measure changes in depression.

Likewise, Zung did not comment directly as to whether there is a cutoff point on the Zung Self-Rating Depression Scale (2) at which depression may be diagnosed but emphasized the ability of the scale to compare populations and to measure change. The developers of the MMPI (10) stated that elevations in any scale are not in themselves diagnostic of that entity. We included the MMPI in this study because other studies have reported MMPI means for alcoholics.

RESULTS

Table 1 summarizes the results of our study. If clinical diagnosis is used as the criterion for depression, only 8.6% of the 35 alcoholics we studied were depressed. The Hamilton Depression Rating Scale has no cutoff point for depression and has not been used by others as a test for depression among alcoholics. We selected a score of 20 as a cutoff point on the basis of a study of mean values in groups of depressed patients (11) that used the Hamilton and Zung scales. By this criterion, 28% of the group in our study were depressed. Weingold and associates (6) used a score of 44 on the Zung Scale in their study of the prevalence of depression among alcoholics. By this criterion, 66% of the patients in our study were depressed. The group mean of the depression scale of the MMPI was 71.3, and 43% of the patients had scores exceeding 70. The Spearman correlation coefficient (.56) between the MMPI and Zung results was significant ($p<.0005$). The correlations between the Hamilton and Zung (.25) and the Hamilton and the MMPI (.27) were not significant.

TABLE 1
The Prevalence of Depression Among Recently Detoxified Alcoholics (N=35)

Diagnostic Method	Depressed Patients (%)	Score	
		Mean	SD
Clinical diagnosis	8.6		
Hamilton Depression Rating Scale (20+)	28	13.2	7.4
Zung Self-Rating Depression Scale (44+)	66	49.8	12.2
MMPI depression scale (70+)	43	71.3	17.1
MMPI or Zung	69		

DISCUSSION

This study of the prevalence of depression among recently detoxified alcoholics replicates the disparate results reported by others. The difference is that in our study it is obvious that the varying results depend on the criteria used to diagnose depression. The 8.6% prevalence based on clinical diagnosis is similar to the 3% prevalence based on data derived from structured interviews in the study of Winokur and associates (3), although the bases for diagnoses differ. It is possible for a structured interview to detect evidence of recurrent affective disorder when it is in remission. It is also possible for clear-cut evidence of manic or depressive episodes to be present without the patient's history suggesting such disorder. The similarity between this study and that of Winokur and associates is that each reports a low prevalence of depression among detoxified alcoholics.

The mean for the depression scale of the MMPI is above 70 in our study and in Rosen's study (7). The Zung scale diagnosed 66% as depressed in our study and 72% as depressed in the study of Weingold and associates (6). Comparison with the study of Shaw and associates (4) is difficult because we did not use the Beck scale. Nevertheless, the 71% diagnosed as depressed by either the depression scale of the MMPI or the Zung scale is impressive.

There is no shortage of factors that could account for the disparate results. The depression scale of the MMPI is in itself not diagnostic of depression. The results of the MMPI are expressed in patterns rather than in single-scale elevations. The F scale of the MMPI, which measures confusion and/or random answering of questions, was as significantly elevated (70.6) as the depression scale. Some confusion in a population would be expected immediately after alcohol withdrawal that in turn followed a period of excessive drinking. Many of the MMPIs are invalid due to this confusion. There is no validation scale for the Zung, but manifest inability to produce a valid MMPI gives reason to question the validity of the Zung.

Carroll and associates (11) reported a critical comparison of the ability of the Zung and Hamilton scales to distinguish between populations of depressed patients who were hospitalized on a psychiatric ward, cared for in a day hospital, and treated in a general practice clinic. The Hamilton but not the Zung separated the groups. This result raises a question about whether the Zung scale can distinguish differences in the severity of depression among patients, although it does not impugn the ability of the device to measure changes within an individual.

Hamilton stated specifically that his scale does not diagnose depression because diagnosis depends on the presence of signs and symptoms of a condition and the absence of evidence of other conditions. The results of chronic alcoholism, a recent period of excessive drinking, and recent alcohol withdrawal can produce responses to items of the Hamilton scale that do not indicate depression. Zung made no such reservations about his scale, but we believe that the same principle applies.

The mean for the most elevated items on the Zung scale was above three on a four-point scale, with one defined as absent or minimal. These items were 1) not enjoying the company of attractive women, 2) diminished eating, 3) less hope for the future, 4) difficulty in making decisions, and 5) not feeling useful and needed. The mean for the least elevated Zung items was below two on a four-point scale. These were 1) crying spells or feeling like crying, 2) weight loss, 3) constipation, 4) tachycardia, 5) irritability, and 6) thinking "others would be better off if I were dead." The mean of the five high items and complete denial of all of the other items in the Zung scale would give a value diagnostic of depression. Not feeling useful and needed, eating less, and lacking hope for the future are consistent with the reality situations of the populations studied. The high Hamilton items are those for which the mean is above one on a five-point scale with zero defined as absent. We noted only those items rated on a five-point scale and reports of sleep disturbance adjusted to such a scale. Depression, guilt, somatic evidences of anxiety, report of sleep disturbance, and hypochondriasis are the high items. The low Hamilton items, those for which the mean was less than one-half with zero as absent and one as mild, are suicidal tendencies and agitation. This pattern is more suggestive of marked depression than is the Zung pattern, but the means are much lower.

The psychiatric diagnoses given to our subjects were all of psychotic rather than neurotic depression. All of the patients so diagnosed were treated with tricyclic antidepressants; no other patients in the study were so treated. It is likely that in other situations some other patients would have been considered to have neurotic depression.

CONCLUSIONS AND SUMMARY

A rather troubling conclusion is that the diagnosis of depression in an alcoholic population by rating scales alone is almost as inclusive as considering all alcoholics to be depressed. This can have two effects, the first and more obvious of which is the widespread use of inappropriate treatment. The second and more subtle effect is the failure to detect the considerable number of individuals who are truly both alcoholic and depressed and require careful treatment with appropriate medication.

The difference between 3% and 98% of affective disorder in alcoholics in other studies is matched by the range from 8.6% to 71% in our study. The figure depends on which diagnostic criteria are used. Many obvious factors account for this result. These include diagnosis by devices either not claimed to be diagnostic (the MMPI and Hamilton scale) or whose use without clinical verification has been questioned (the Zung scale). A significant factor may be that the sequelae of chronic alcoholism and/or a recent prolonged spell of excessive drinking and/or recent alcohol withdrawal can produce signs and symptoms similar to those of depression and invalidate self-administered tests. This result is of critical importance if the diagnosis of depression leads to treatment with tricyclic antidepressants or lithium. However, there are indeed many alcoholics who have the types of affective disorders that require such treatment.

It must be emphasized that studies of the relationship between affective disorder and alcoholism which are based on a sample biased by demographic factors such as ethnicity, social standing, region, and the method of subject location do not give accurate measures. Nevertheless, within any one study the differences in rate that depend on the criteria for diagnosis are valid.

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Family Rituals and the Recurrence of Alcoholism Over Generations

BY STEVEN J. WOLIN, M.D., LINDA A. BENNETT, PH.D., AND DENISE L. NOONAN

There have been relatively few attempts to identify environmental factors in the transmission of alcoholism across generations. Using the framework of family systems theory, the authors examined the extent of change in family rituals in 25 families in which at least 1 parent was or had been an alcoholic. They found that families whose rituals were altered during the period of heaviest parental drinking were more likely to evidence transmission of an alcohol problem to the children's generation than families whose rituals remained intact.

OUR OBJECTIVE in this paper is to report the results of a family study assessing the impact of the maintenance of disruption of family ritual on the recurrence of alcoholism over generations. Jellinek (1) and Winokur and associates (2), among others, have documented the frequent incidence of alcoholism across generations of families. However, to date there is little evidence based on research to explain why alcoholism transmission occurs in some families but not in others. The studies of Goodwin and associates of adoptees of alcoholic biological parents in Denmark (3, 4) suggested that genetic factors may play a role in the development of chronic and severe alcohol problems over generations of families. In contrast, few attempts have been made to identify environmental factors—those associated with family life, for example—that are clearly related to alcoholism transmission.

In this research we looked to the family's psychosocial environment to explain the continuity of alcoholism over generations. Our work is grounded in family systems theory, which views the family unit as the locus of pathology in the sense that the pathology is so intertwined with ongoing family functioning that the problem cannot be isolated from the rest of family interaction and behavior.

Applying the systems concept to alcoholic families, Ewing and Fox (5) described the alcoholic marriage as a homeostatic mechanism that strongly resists change. Ablon (6) suggested,

> If the family is considered as a social system with interdependent parts consisting of interacting personalities with each having his own expected role functions, it follows that the behavior of each part . . . affects the functioning of the others. (p. 209)

Steinglass (7) proposed that alcoholism not only becomes enmeshed in family functioning but can also develop into an adaptive organizing mechanism for the family:

> Alcohol, by dint of its profound behavioral, cultural, societal, and physical consequences, might assume such a central position in the life of some families as to become an organizing principle for interactional life. . . . In such a system the presence or absence of alcohol becomes the single most important variable determining the interactional behavior. (p. 106)

According to the theoretical framework of our research, the family is powerfully motivated to maintain itself, even over generations. It strives to preserve the roles its members play, the values and beliefs they have in common, and the rules on which they agree. The family's shared perception of itself, or family identity, is conveyed by way of family rituals and myths.

Family rituals are patterns of behavior that have meaning beyond their practical outcome or functional purpose. Family myths are stories or legends that incorporate the crucial events, the important people, and the major themes of the family's history.

The underlying hypothesis of our research is that families which persist in maintaining their family rituals throughout the children's growing up period, even in the face of a serious parental alcohol problem, will evidence less transmission of alcoholism into the children's generation. Conversely, families whose rituals are substantially changed are much more likely to have alcohol problems in both generations. The former category of families we have called "distinctive," i.e.,

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the alcohol abuse behavior remained distinct from the ritual life of the family. The latter group of families we have termed "subsumptive," indicating that the alcohol abuse behavior subsumed the ritual life of the family.

RESEARCH DESIGN

Our study group consisted of 25 families, primarily of middle- and upper-middle-class background and European origin, living in the greater Washington, D.C., area. All 25 families included at least 1 parent who met the criteria of Goodwin and associates for alcoholic or problem drinker (3). In 18 of the families it was the father who was the alcoholic; in 5, the mother; and in 2, both parents. Twelve families evidenced no alcohol problems in children's generation (nontransmitter); 7 families had at least 1 child who was a heavy drinker (intermediate transmitter); and 6 families included at least 1 child who was an alcoholic or problem drinker or who was married to a problem drinker (transmitter). According to our theoretical perspective, marriage to someone with an alcohol problem represents a perpetuation of the alcoholic family system and therefore constitutes transmission.

Chi-square tests verified independence between the transmission of alcohol problems and parents' ethnic background, religion, education, and occupation. T tests revealed no significant differences between families in the three transmission categories in respect to mean age of parents, length of time since alcoholic parent last drank heavily, and current drinking status of the alcoholic parent (active or inactive). The mean age of interviewed children in the transmitter families was higher than the mean age of interviewed children in the nontransmitter families, significant at the .05 level. However, no significant difference was found between the mean age of children with alcohol problems (26.9) and the mean age of children without alcohol problems (23.9).

Structured individual interviews were conducted with both parents in the 25 families and with as many grown children as were willing to participate in the study (N=63, an average of 2.5 children per family). The first interview covered the personal history of the interviewee, including an alcohol history and a history of his or her relationship with the family of origin and the present nuclear family. The second interview addressed the continuity of family heritage from the grandparents' generation into the current nuclear family and provided detailed information about 7 areas of family life while the children were growing up: dinnertime, holidays, evenings, weekends, vacations, visitors in the home, and discipline.

To avoid bias on the part of the research staff, a clinical psychologist who had no previous involvement with the study was employed to code the interview data. A coding manual was devised to direct the coder in this process, and all references to children's drinking habits were deleted from the interview transcripts.

Two issues were addressed in the coding for each family: 1) which family life areas were rituals and 2) what happened to those ritual areas during the period of the alcoholic parent's heaviest drinking. The coder was asked to answer 12 questions from the point of view of each family member interviewed in respect to the 7 areas of family life.

The first 5 questions assessed whether or not a given area of family life was characterized by patterned behavior and whether or not that area had ritual aspects. Patterned behavior is behavior that is repetitive, stable with respect to roles, and continued over time.

Our definition of ritual builds on the description of patterned behavior but also encompasses the elements of acceptance by family members of the continuation of this behavior in the same way time after time and symbolism, or attribution of meaning or purpose to the continuation of this behavior beyond any practical considerations.

The first 5 ritual-related questions focused on the family life before the period of heaviest drinking, and the last 7 alcohol-related questions referred to the time when the drinking was heaviest. We examined these two different periods to obtain a picture of the family and its rituals before the drinking was most severe and to determine what impact the alcohol-abusing behavior had on the family's already established rituals. In other words, we were looking for evidence of change in whatever rituals the family had developed.

Questions 6–12 addressed the following alcohol-related issues in respect to each area of family life: presence of the alcoholic parent, alcohol use by the alcoholic parent, intoxication of the alcoholic parent, response of the family to the intoxication, change in level of participation of the alcoholic parent when intoxicated, response of the family to that change, and overall change during the period of heaviest drinking.

We reduced the resulting codes in order to determine which areas of family life were rituals in each of the 25 families. Looking at those identified rituals, we determined the degree of change, if any, during the period of heaviest drinking. Each family was categorized as being distinctive (no overall change in the rituals), intermediate subsumptive (approximately half the rituals altered), and subsumptive (all rituals changed). Eight of the families were considered distinctive, 10 were considered intermediate subsumptive, and 7 were considered subsumptive. Table 1 shows the distribution of distinctive and subsumptive ritual life areas in each category.

To check interrater reliability, a staff member who had not previously worked with the data and had no information on the results was given 6 randomly selected family files to code. The original codes and the new codes were then compared for agreement on designation of each family life area within a family as patterned behavior and/or ritual (based on questions 1–5). For these 7 possible areas across the 6 families there was 88% agreement on the final decision of patterned

TABLE 1
Identification of Distinctive (D) and Subsumptive (S) Family Ritual Life Areas by Overall Ritual Change Type

	Possible Ritual Life Areas					
Family	Dinner	Holidays	Evenings	Weekends	Vacations	Visitors
Distinctive						
1		D				
2		D		D		
3	D	D				
4	D	D				
5	D	D		D		
6	S	D			D	
7	D	D		D	D	
8		D				
Intermediate subsumptive						
9	D	S	D	S		D
10	D			S		
11	S	D				
12	S	D				
13	S	D				
14	D	D			S	S
15	D	D	S	S		
16	S	D			D	S
17	S	S		D	S	
18	S	S	D			
Subsumptive						
19	S	S		S	S	S
20	S	S	S		S	S
21	S	S		S		
22		S				
23		S				
24	S	S		S	S	
25		S		S		

TABLE 2
Ritual Change Type for 25 Families by Alcoholism Transmission Category[a, b]

	Alcoholism Transmission Category		
Ritual Change Type	Transmitter (N=6)	Intermediate Transmitter (N=7)	Nontransmitter (N=12)
Subsumptive (N=7)	4	2	1
Intermediate subsumptive (N=10)	2	2	6
Distinctive (N=8)	0	3	5

[a] $p<.10$ by chi square when all three ritual change types are compared with all three alcoholism transmission categories.
[b] $p<.025$ by Fisher's exact test when only the extreme groupings of families (subsumptive and distinctive versus transmitter and nontransmitter) are considered.

behavior and 83% agreement on the decision of ritual. Reliability was also checked for the data obtained from alcohol-related questions 6–12. Comparison of the codes derived by summarizing across all identified ritual areas within each family yielded results of 67% agreement on questions 9 and 11; 83% agreement on questions 7, 8, and 10; and 100% agreement on questions 6 and 12. A second measurement for question 12, comparing final classification of families into ritual change types, resulted in an agreement score of 83%.

DATA ANALYSIS

To test our original hypothesis—the greater the change in family rituals, the more frequent the transmission of alcoholism to the children's generation—we examined the distribution of the 25 families studied according to transmission category and ritual change type. Table 2 displays that distribution. Using chi-square analysis, we arrived at results providing evidence for a relationship between change in ritual life and transmission when all 25 families were considered. All 6 transmitter families were in the subsumptive or intermediate subsumptive categories; the 7 intermediate transmitter families were evenly spread throughout the three ritual change types; and the 12 nontransmitter families clustered in the intermediate subsumptive and distinctive categories.

When we disregard all the intermediate categories for both transmission and ritual change and examine only the extreme groupings of families, we arrive at convincing results (see table 1), indicating that there is a relationship between substantial change in ritual life and transmission of problem drinking or alcoholism into the children's generation.

The coded data provided considerable additional alcohol-related information for the 25 families. This material allowed us to compare overall characteristics found in the three transmitter categories according to particular results from questions 6–12. Families without dinnertime or holiday rituals were excluded from this comparison. The 6 transmitter families were clearly more similar to each other than to either the intermediate transmitter or nontransmitter groups of families. At least two-thirds of the transmitter families conformed to the following characteristics: the alcoholic parent was usually present in the home during the time family rituals occurred, he or she was usually drinking at those times, he or she was usually intoxicated, the family accepted that intoxication by demonstrating no active or negative response, there was a change in the level of participation of the alcoholic parent, the family accepted that change in level of participation, and there was overall change in identified family rituals.

In comparison, at least two-thirds of the intermediate transmitter families shared only the following characteristics: the alcoholic parent was usually drinking, he or she was usually intoxicated, and the family accepted whatever change in level of participation occurred. At least two-thirds of the nontransmitter families, on the other hand, had only one outstanding quality in common: a rejection of the intoxication of the alcoholic parent through such means as confronting the alcoholic parent openly or privately or talking about his or her behavior disapprovingly.

When we specifically scrutinized change versus no change in dinner and holiday rituals for each family transmission category, we found that only the intermediate transmitter families had their dinnertimes altered and only the transmitter families had their holidays changed. Conversely, both the intermediate

transmitter and nontransmitter families kept their holiday rituals intact. This is noted because we have observed that holidays are ordinarily the family rituals most likely to persist, even in the face of a serious alcohol problem.

Certain answers to particular alcohol-related questions indicated protection of family rituals. For all families there were 5 possible protective characteristics: 1) that the alcoholic parent was not usually intoxicated, 2) that the family rejected the alcoholic parent's intoxication, 3) that there was no change in the alcoholic parent's level of participation in ritual areas, 4) that the family rejected any change in the alcoholic parent's level of participation in ritual areas, and 5) that there was no overall change in ritual areas during the alcoholic parent's period of heaviest drinking.

Taking into account all of the answers for all families in each transmission category, we found a relationship between percentage of protective characteristics and transmission category at below the .001 level of significance using chi-square analysis. As a group, nontransmitter families had the highest percentage of protective characteristics (53%); the transmitter families had the lowest (13%); and the intermediate transmitter families fell in the middle (34%).

DISCUSSION

In developing our concept of subsumptiveness we have attempted to describe a family-level process important in the transmission of alcoholism over generations of families. We found that some families maintained their ritual behaviors in spite of stress under the impact of alcoholism, while others were less able to maintain their rituals under similar stressful conditions. In the subsumptive families, alcoholism behavior had invaded normal family ritual behavior, thereby substantially changing family life. For these families, alcoholism became a central organizing force, ritual continuity was disrupted, and there was a higher risk of transmission.

In this research our objective was to distinguish these two family types. However, an examination of ritual change versus no change during the period of heaviest drinking demonstrated three distinct family styles of handling an alcohol problem. Therefore, our categorization of 25 families includes an intermediate grouping.

Distinctive Families

As a group, the 8 distinctive families had the following characteristics in common: whenever intoxication occurred at ritual times (a relatively rare event), the families tended to accept that behavior by ignoring it; there was very little change in the level of participation on the part of the alcoholic parent; and there was considerably less transmission of the alcoholism into the children's generation.

In general, the distinctive families appeared to be comparatively stable; the behavior of the alcoholic parent was relatively less intrusive. The quality of constancy of the distinctive families was due in part to the infrequency of intoxication relative to the intermediate subsumptive and subsumptive groups, the absence of an overt negative response by other family members when intoxication did occur, and the stability of participation of the alcoholic parent in family ritual life.

It is important to note that the characteristic of "no change in participation" can be interpreted in at least three ways: that the family insisted the alcoholic parent remain involved, even during the difficult times of his or her drinking, that the alcoholic parent was never very involved in family ritual life, or that family rituals occurred at intermittent intervals (i.e., holidays and vacations) rather than daily (i.e., dinner and evenings). In fact, our interview data indicated that in 4 of the 8 families the alcoholic parent (in these cases, always the father) had seldom participated in family ritual life before the period of heaviest drinking. Either due to isolation by other family members or self-imposed detachment, the alcoholic parent in these 4 families was never fully integrated into family rituals. Additionally, dinnertime—the most regular family ritual time—was not a ritual area for 3 of the distinctive families, indicating that in these families, at least, participation of the alcoholic parent did not occur on a daily basis.

There is evidence of considerable denial that an alcohol problem actually exists in some of these distinctive families, especially on the part of the children. Some of them stated in the interview that they never noticed any intoxicated behavior in their parents when they were growing up. Perhaps such denial is another means for the family to keep alcoholism distinct from family rituals. Whatever the strategy used by each of these families, the result is that there was significantly less transmission in the children's generation.

Intermediate Subsumptive Families

Although the overall categorization of the intermediate subsumptive group as intermediate was based on moderate change in family rituals, additional information about each of these 10 families indicates that their approach to the alcohol problem was clearly different from that of families in the other two groups. In the intermediate subsumptive families the alcoholic parent was usually present during ritual times, he or she was usually drinking, he or she was usually intoxicated, the family tended to reject that intoxicated behavior, the alcoholic parent participated less in ritual life, the family tended to reject that diminished participation, and there was less transmission of alcoholism than in the subsumptive families but more than in the distinctive families.

Our overall impression of these families was that much more activity occurred around the alcohol abuse behavior than in the distinctive families. They seemed to be families in flux, families that responded overtly to the alcohol abuse behavior and change in level of participation in such a way that the family's response

may have contributed to the moderate alteration of family ritual life. Ritual change might have been caused by the family's active struggle with the alcohol problem. Typically, there was less denial of the alcohol problem in intermediate subsumptive families than in distinctive families and less transmission than in subsumptive families.

Subsumptive Families

In direct contrast to the distinctive families, the ongoing ritual behavior in the 7 subsumptive families was invaded by the alcohol abuse behavior; it changed the fabric of family life. As a group the subsumptive families had the following characteristics in common: alcohol use by the alcoholic parent usually occurred, intoxication usually occurred, the alcoholic parent participated less in ritual life, family members tended to accept that change in level of participation, and the greatest transmission occurred.

These families exhibited a highly disrupted family life, and they were clearly the most vulnerable to the effects of the alcohol problem. In comparison with the other two groupings, the subsumptive families were less stable, as evidenced by the fact that all identified family rituals were altered during the period of heaviest drinking. As in the distinctive families but unlike the intermediate subsumptive families, subsumptive families had their ritual life concentrated at such intermittent intervals as holidays. Three of these families had no dinnertime rituals, perhaps indicating a deterioration of daily rituals in advance of the time the alcohol problem became most serious. Among the subsumptive families there was a far greater frequency of alcohol use and intoxication by the alcoholic parent at ritual times, with much less rejection of that behavior by other family members than in the intermediate subsumptive families. In their acceptance of less participation by the alcoholic parent at ritual times, these families might have been demonstrating their great relief at the decreased involvement of the alcoholic parent, who frequently wreaked havoc in ritual life, embarrassing other family members and making them anxious about future ritual activities. The decreased involvement of the alcoholic parent was often the most acceptable solution for these families. In short, the subsumptive families were incapable of keeping the impact of the alcohol problem under control.

We have found that there was a relationship between the maintenance or disruption of family rituals and the recurrence or nonrecurrence of alcoholism over generations in our 25 families. Furthermore, we have found that the degree of ritual change in family life was an overall reflection of specific family styles of handling a serious alcohol problem.

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Short-Term Spatial Memory Deficits in Men Arrested for Driving While Intoxicated

BY ERIC W. FINE, M.D., AND ROBERT A. STEER, ED.D.

In the light of the fact that ethyl alcohol can cause damage to the brain, especially in the area of motor control, a study of the incidence of alcohol-related neuropsychological impairment in individuals arrested for driving while intoxicated (DWI) would be of great value. The authors report on 100 DWI offenders given a Memory-for-Designs Test: 33% of the offenders were in the borderline range for organicity and a further 24% were in the critical range. Although they advise caution in interpreting these data, the authors point out a number of implications their analyses have for those interested in alcohol-related highway safety programs.

THE FACT THAT ethyl alcohol can cause damage to the brain is now widely accepted. However, there is a surprising lack of awareness as to how common alcoholic brain disorders really are, although Lemere (1) observed more than 20 years ago that "for each alcoholic with demonstrable pathology, there must be thousands in intermediate stages of danger." Since the 1960s, psychological studies of alcoholics have been accumulating in the scientific literature suggesting that alcohol produces neuropsychological impairment. In an excellent review of this literature, Tartar (2) concluded that "there is substantiative experimental evidence of a psychological nature to implicate a neurological impairment in chronic alcoholics."

Using the Halstead-Reitan Battery, several investigators (3, 4) have found that there is a neuropsychological impairment in alcoholics but that it is not as severe as that found in patients with traumatic brain injury. Tartar (5, 6) compared alcoholics, people with brain injuries, and experimentally lesioned animals and found many similarities among these groups. Among other impairments was a "marked inability of alcoholics to temporally organize behavior and persist with a cognitive act."

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It appears likely that alcoholic patients do not manifest global neuropsychological impairment but demonstrate particular deficits on tasks requiring visual-spatial information processing (7). There is also evidence suggesting that the duration of alcohol abuse is correlated positively with a progressive decline in competency (8–10). It is possible that the psychological deficits observed in alcoholics are influenced by the nutritional status of the subjects: thiamine deficiency is a well-known complication of chronic alcoholism. However, there is experimental evidence from animal studies (11, 12) indicating that alcohol has a deleterious effect on learning and timing behavior in spite of adequate nutritional status. Clarke and Haughton (13) found significant intellectual impairment among a group of heavy drinkers; they also found that these heavy drinkers remained impaired for a considerable time after drinking had been discontinued.

The most obvious effects of alcohol are on motor control. Deficient performances by alcoholics compared with normal subjects have been found in tests of finger tapping (14), finger and manual dexterity (15), and muscle strength (10). These disturbances are thought to be most likely cerebellar in origin.

It would obviously be of value to determine the incidence of alcohol-related, neuropsychological impairment in a group of people arrested for driving while intoxicated (DWI), but we found no such studies in the literature. Many investigators (16, 17) have confirmed the presence of heavy, prolonged alcohol intake in a substantial proportion of DWI offenders. Fine and Scoles (18) noted that about 64% of a group of DWI offenders were alcohol-dependent and were therefore likely candidates for early case-finding and secondary prevention of alcoholism. The average blood alcohol concentration (BAC) of these offenders at the time of arrest was .18%, and the subjects described themselves as having maintained the same drinking pattern for an average of 9.58 years. Given these degrees of involvement with alcohol, it is important to ascertain whether or not DWI offenders show any signs of organic brain syndromes.

METHOD

Study Group

The subjects for this study were 100 men admitted to the Alcohol Highway Safety Diagnostic Unit of the

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West Philadelphia Community Mental Health Consortium. The 100 men represented consecutive admissions referred for diagnostic appraisal as part of the city of Philadelphia's Alcohol Highway Safety Program over a period of 2 weeks. The referrals were made by the court, which used diagnostic assessments to determine whether or not the men should be sent to educational safe-driving classes or to the appropriate alcoholism treatment.

The sample was composed of 41 white men and 59 black men. Their mean age was 40.82±11.22 years, and their mean educational attainment was 9.69±3.53 years. The marital status of the men was complex: 17% had never married, 54% were currently married, 16% were separated, 10% were divorced, and 3% were widowed.

Only 9 of the men had been treated for alcoholism, but 31 had been arrested for driving while intoxicated before. The mean length of their current drinking pattern was 10.47±8.94 years. The mean breathalyzer score, furnished by the Philadelphia Police Department and obtained at the time of arrest, was .18%±.05%. The National Institute on Alcohol Abuse and Alcoholism's Quantity-Frequency (QF) Index (19), which represents the average number of ounces of alcohol intake per day over the past month, was calculated for the men; their mean QF Index score was 1.33±2.70 oz. We also used another measure of alcohol impairment developed for NIAAA, the Impairment Index (19), which is composed of 12 questions asking about the extent of self-perceived problems attributed to excessive alcohol use over the past month. The mean Impairment Index score was 3.93±5.00. The QF Index and Impairment Index scores were higher than those reported for drinking American adults (.8 oz. and 1.2, respectively).

Instrument

The instrument used to measure short-term spatial memory deficits was Graham and Kendall's Memory for Designs Test (MFD) (20). The MFD has been used in a number of diverse clinical investigations and has been found useful for assessing mild organic complications. Briefly, the test consists of 15 designs, each printed in black on a 5-inch cardboard square. Each of the designs is shown to the subject for 5 seconds. After the 5-second exposure, the design is withdrawn and the subject is asked to draw one exactly like it. It takes about 10 minutes to complete the entire test.

Higher total scores are associated with poorer performance. The scoring of each design is based on the following criteria: a score of zero reflects a satisfactory reproduction, an omitted figure, or incomplete reproduction; a score of 1 represents a drawing in which there were two easily identifiable errors but the overall design was adequate; a score of 2 indicates more than two easily identifiable errors; and a score of 3 indicates that a drawing has been reversed or rotated.

The MFD was administered to the 100 subjects by the clinicians who were conducting the diagnostic interviews. These clinicians were trained in the administration of the test by one of us (R.A.S.), who later scored all 100 tests. The MFD was presented at the end of the diagnostic interview, which lasted approximately an hour. None of the men was judged to be intoxicated by the interviewers, nor did any of the men admit to drinking just before the interview. The MFD drawings were given to the scorer without any information about background or drinking histories.

TABLE 1
Correlations of Background Characteristics and Drinking Behaviors with MFD Scores of 100 Subjects

Variable	r
Background	
Age	.34[a]
Race (white versus black)	.22[b]
Education	−.36[a]
Employment status (unemployed versus employed)	−.20[b]
Drinking behaviors	
BAC at time of arrest	.21[b]
Previous arrest for driving while intoxicated	.02
Length of current drinking pattern	.21[b]
QF Index	−.04
Impairment Index	.22[b]

[a]$p<.001$
[b]$p<.05$.

TABLE 2
Stepwise Regression Analysis for MFD Scores of 100 Subjects

Variable	r^2	F
Education	.13	11.35[a]
Age	.23	7.57[b]
Impairment Index	.28	6.49[c]
BAC at time of arrest	.30	2.18
QF Index	.31	1.86
Previous arrest for driving while intoxicated	.32	1.63
Race (white versus black)	.32	.36
Length of current drinking pattern	.33	.33
Employment status (unemployed versus employed)	.33	.03

[a]$p < .001$.
[b]$p < .01$.
[c]$p < .05$.

RESULTS

The mean MFD score for the 100 DWI offenders was 8.15±8.33; this mean is within the borderline range reported by Graham and Kendall (20). Diagnostically, 43% of the subjects were within the normal range (score of 0–4), 33% were within the borderline range (score of 5–11), and 24% were within the critical range (score of 12 or more). These percents differ from those given for control subjects in the MFD manual (20): 79% of the matched validation control group were normal, 17% were borderline, and 4% were critical. However, that control group represented a wide disparity in age. Focusing on the 22 control subjects whose ages were between 41 and 45 (the range in which the mean age of the present sample fell), the

average score was 4.00; the control group's mean score was thus approximately half that of the present sample.

To ascertain whether or not the men's background characteristics and drinking behaviors were related to their MFD total scores, nine background characteristics and drinking behaviors were correlated with the MFD scores (see table 1). Only previous arrest for driving while intoxicated and the QF Index were not significantly related to the MFD scores.

Since the background characteristics and drinking behaviors were intercorrelated, we decided to enter all of the variables into a stepwise regression analysis for the MFD scores. Table 2 presents the stepwise regression for the MFD scores. Education, age, and the Impairment Index contributed significant amounts of variance to the explanation of the MFD scores. The Impairment Index was significantly related to the MFD scores ($p<.05$), even after the influences of education and age were controlled for.

DISCUSSION

The findings reported here must be interpreted with caution: 33% of the men in this sample were within the borderline range for organicity, and a further 24% were in the critical range. Such results give great cause for concern because MFD performance has been shown significantly to differentiate subjects with brain disorders from those without brain disorder (20). The fact that the present results were found in individuals referred for evaluation after being arrested for driving while intoxicated suggests that the clinical and judicial management of such individuals requires serious reconsideration.

The relationships of the MFD scores to age, education, and the Impairment Index indicate that older, less educated men with higher levels of impairment due to alcohol had the most difficulty in remembering the spatial designs. When age and education were controlled for in the stepwise regression, there was still a positive relationship between the MFD scores and the Impairment Index. This finding might indicate an association between alcoholic symptoms and possible organicity.

Great emphasis has been given to the types of alcohol abuse and dependency in DWI offenders, and there has been considerable debate in the literature concerning the role of psychological factors in drinking drivers (21, 22). This study highlights another important variable that must be considered by people interested in alcohol highway safety programs.

In their study of alcoholic intellectual impairment Clarke and Haughton (13) found that heavy drinkers exhibited deficits in perceptual-motor coordination and abstract reasoning that would pose driving difficulties, especially in situations demanding sudden reactions and quick decisions. The findings of the present study suggest that some DWI offenders also display short-term spatial memory deficits; these drivers might not be able to remember road signs, traffic alerts, or emergency signals. This finding becomes particularly alarming when one realizes that the current sample was not drawn from chronic alcoholic patients but from people arrested for routine alcohol-related traffic violations. It is important to emphasize that these results were obtained when the subjects were clinically judged to be free of alcohol; they would be expected to be more impaired after alcohol consumption.

The results of this research might have been affected by the degree to which the subjects were under the influence of alcohol at the time of testing and by their high levels of anxiety (they were interviewed after having been arrested). We attempted to minimize these factors by excluding from the study anyone who admitted to drinking on the day of the interview or who showed any alcohol-related symptoms. The diagnostic interviews, including the MFD, were conducted by highly trained and very experienced personnel who worked exclusively with DWI offenders. The MFD was administered at the end of an hour-long interview, during which time any alcohol-induced interference with performance would have become apparent. Our experience has been that patients are much less anxious following the interview, which is conducted in a nonthreatening empathic fashion. Future investigators might well consider the use of a breathalyzer to ensure that the subjects' BAC is zero when they take the MFD.

One other possible source of bias should be mentioned. Although the person scoring the MFD had no knowledge of the drinking history of the subjects, he obviously knew they had all been arrested for driving while intoxicated. Therefore, we did not have the advantage of a truly blind evaluation.

The possibility of short-term memory impairment in DWI offenders has implications for the type of programs offered to them by the judicial and mental health systems. In general, these programs usually offer combinations of sanctions, education, and, occasionally, treatment. If there is a high incidence of organic brain damage in DWI offenders, each of these approaches will require modification. For example, depending on the extent of impairment, the courts might be obligated to impose long-term suspension of the driver's license with the option of reinstatement after appropriate medical and psychological review. Driver education courses for these offenders might have to be extended to allow for the repetition of content. Individual assessments of potential learning disabilities should also be included in these courses. If there is a suspicion of organicity during the course of psychological assessment, a neuropsychiatric evaluation should be performed. On the basis of the findings of this latter evaluation, a comprehensive treatment regimen should be prescribed. The regimen might include mandatory abstention from alcohol, appropriate medication, and highly structured, direct psychotherapy.

Further investigations of the validity and reliability of the short-term spatial memory deficits found here should be undertaken. The MFD must be regarded as a screening instrument only; it cannot substitute for a thorough neuropsychological testing procedure such as the Halstead-Reitan Battery. Ideally, DWI offenders suspected of having organic brain damage should be referred for neurological evaluation and for such possible specialized investigations as computerized axial tomography.

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Computerized Transaxial Tomographic and Neuropsychological Evaluations in Chronic Alcoholics and Heroin Abusers

BY SHIRLEY Y. HILL, PH.D., AND MICHAEL A. MIKHAEL, M.D.

Computerized transaxial tomographic (CTT) scans and the Halstead-Reitan Neuropsychological Battery were administered to 42 paid male volunteers (15 chronic alcoholics, 15 heroin abusers, and 12 matched control subjects). CTT scans revealed that only 1 of the chronic alcoholics had a ventricle/brain (V/B) index larger than normal. One unexpected finding was that the heroin abusers had significantly smaller sulci and V/B indices. Neuropsychological assessments of the alcoholics and heroin abusers revealed functional impairment in a majority of cases; the alcoholics were twice as likely as opiate abusers to show impairment.

CHRONIC ALCOHOLISM and heroin abuse may affect as many as one in five urban men (1, 2). The relationship between the abuse of psychoactive drugs and brain damage has been widely debated as substance abuse continues to be a persistent social and medical problem. A number of studies have assessed brain damage using a variety of psychological tests (3–6); others have used pneumoencephalography (7, 8), the EEG (9, 10) and, more recently, computerized transaxial tomography (CTT) (11–13).

Unfortunately, few studies have used the proper controls necessary to conclude that an association exists. Most studies involving psychological tests have ignored demographic variables that can alter the proportion of individuals diagnosed as impaired (14). In addition, the focus of previous studies concerned with the effects of opiate abuse has been restricted to polydrug abusers because of the great difficulty in finding individuals who have only abused opiates. Polydrug abuse may also complicate the study of alcoholism and its association with cerebral impairment. Only by eliminating polydrug abusers from studies of this type can the relative risk associated with the chronic abuse of particular drugs be determined.

Further, in the few studies completed to date that have utilized CTT, subject selection has been based on persistent abnormal functioning, neurological signs, or other indicators warranting CTT scans (11, 12). Clearly, data have previously not been available to determine the rate of cerebral atrophy in a randomly selected population of substance abusers.

The purpose of the present study was to determine whether alcoholics and heroin abusers exhibit comparable neuropsychological impairment as assessed by the Halstead-Reitan Neuropsychological Battery and whether structural alterations are manifest using CTT scans.

METHOD

Subjects

Forty-two male subjects were selected for neuropsychological testing and CTT. These subjects were alcoholics (N=15), heroin abusers (N=15), and controls who had never abused drugs (N=12). The subjects were part of a larger study of neuropsychological impairment in approximately 100 substance abusers. The alcoholics and heroin abusers having the least amount of multiple drug abuse were chosen from this larger pool of subjects for CTT study. Each had been given an extensive psychiatric interview covering lifetime history of substance abuse. The interviews ensured that subjects in the alcoholic group had never abused opiates and that none in the opiate group had ever abused alcohol. Similarly, the control group consisted of individuals who had never abused alcohol, opiates, barbiturates, stimulants, or hallucinogens.

The extensive drug history interviews also allowed for control for any possible acute effects of drugs on neuropsychological tests. It was determined that 8 of the alcoholics and 6 of the heroin abusers had been in remission for 1 month or more; 4 of the 8 alcoholics had not drunk for at least 6 months, and 3 of the heroin abusers had not used heroin for at least 6 months.

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Subjects were drawn from outpatient alcoholism and drug abuse programs in the St. Louis, Mo., area (i.e., Malcolm Bliss Mental Health Center, Veterans Administration Hospital, Narcotics Service Council). Persons included in the study had a minimum education of 10th grade, were at least 25 years old, and had no history of head trauma or high fever. The control group was drawn from churches and social groups in the St. Louis metropolitan area.

Because demographic variables may influence psychological test scores, the groups were matched as closely as possible for age, education, race, and socioeconomic status. The mean ages of the alcoholics, opiate abusers, and controls were 34.3, 29.2, and 28.0 years, respectively. Their respective educational levels were 11.0, 11.5, and 12.7 years of school. Most of the subjects were black, with only 3 of the alcoholics being white. The subjects were asked about the best job they had ever held; this occupation was coded using a socioeconomic index classification (15). The three groups were well matched for socioeconomic characteristics. The alcoholics had a socioeconomic index of 14.5, the heroin abusers 23.6, and the controls 28.1. These values correspond approximately to semiskilled to skilled labor.

All of the subjects in the alcoholic group met a diagnosis of alcoholism by the criteria of Feighner and associates (16). This diagnosis was based on criteria including physical dependence, social and legal problems resulting from alcohol abuse, and the person's own estimation that he drank too much. In addition, all of the subjects selected for study reported drinking a fifth of liquor per day when they were drinking. The average length of time they had drunk heavily was 14.3 years, with a range of 7–25 years.

The group of alcoholics chosen for study was characterized by an absence of opiate or other drug abuse. Nine of the 15 alcoholics had never used barbiturates, stimulants, or hallucinogens. The remaining 6 had never used these drugs on a regular basis (3 or more times per week). Six of the alcoholics had never tried marijuana, 6 had used marijuana infrequently (less than once a week), and 3 had used it as often as once or twice a week.

All of the subjects in the heroin abuse group met a diagnosis of drug abuse by the criteria of Feighner and associates (16) and had taken opiates three or more times a week for a minimum of 4 years. The group as a whole had abused opiates an average of 8.6 years; the longest use was 14 years. The available street drugs in St. Louis included heroin, morphine, codeine, hydromorphone (Dilaudid), and meperidine (Demerol). Nine of the 15 subjects had used methadone for more than a year, and 5 had used it for at least 4 years.

The attempt to select opiate abusers with minimal use of other drugs was largely successful. Although all but 1 subject drank alcohol socially, only 4 could be characterized as regular drinkers (7 or more drinks at one time at least once a week). All used marijuana; 8 used it one or more times a week, while 7 used it infrequently (less than once a week). All had tried stimulants, but more than half had used them less than 25 times in their life, with only 3 using them regularly (3 or more times per week). Two-thirds of the group had used barbiturates at some time in their life but never on a regular basis. These 10 subjects had a lifetime use averaging only 27 times. The remaining 5 subjects reported never having used barbiturates. LSD use was uncommon; 1 subject had used it twice, and 3 had used it once.

Twelve control subjects were chosen whose demographic characteristics matched as closely as possible the characteristics of the alcoholic and opiate abuser groups. These 12 subjects were selected from a population of 60 volunteers because they had no history of drug abuse. All but 1 of them drank alcohol socially, but none was a heavy user of alcohol. One control subject had experimented with barbiturates (15 times), while 4 had tried stimulants (all less than 10 times). One of the controls had inhaled heroin once, and 4 had used psychedelic drugs (none more than 3 times). All had tried marijuana, but none had ever smoked it more than 4 times a week.

CTT Scans

CTT scans were performed using the standard technique. A total of eight images were obtained based on four scanning sequences. Polaroid images were used to determine the first lower cut showing the lateral ventricles. Four consecutive slices were then used to measure the size of the lateral ventricles and the brain.

Evaluation of the CTT scans consisted of measuring the area of the lateral ventricles and the area outlined by tracing the inner table of the skull in each of the four slices under consideration. In those cases in which the ventricles were entirely included in three slices, the value of the ventricular area in the fourth slice was considered to be equal to zero. The sum of the areas of the ventricles in four slices was divided by the sum of the areas of the cranium, and the ratio was expressed as a percentage or ventricle/brain (V/B) index.

This method is a modification of that used by Synek and associates (17). However, measurements taken directly from Polaroid pictures have a minification factor of about 3.6. In addition, slight errors in the measurement on a minified image lead to larger errors in computed values. Therefore, the ventricular area was determined by tracing the perimeters of the lateral ventricles and the inner table of the skull using a transparency superimposed on the numerical computer printout. A hand planimeter, moved around the perimeters of the lateral ventricles and cranium that had been traced, allowed measurement of each slice. All measurements were completed twice. Using this technique, the areas of the ventricles rather than the linear distance across the ventricles could be determined (18).

The width of the sulci was judged blindly by two independent raters using the following numerical

TABLE 1
Comparison of Neuropsychological Performance Scores for 15 Alcoholic Subjects, 15 Heroin Abusers, and 12 Matched Controls

Test	Scores: Alcoholics Mean	Alcoholics SD	Heroin Abusers Mean	Heroin Abusers SD	Controls Mean	Controls SD	Normal Range	Significance: Alcoholics/Controls	Significance: Heroin Abusers/Controls
Category Test	72.7	27.0	56.6	30.2	44.9	22.9	≤51	p<.05	n.s.
Tactual Performance Test									
Total time	19.5	6.8	11.9	3.6	10.9	1.9	≤15.6	p<.001	n.s.
Memory	4.6	2.3	5.9	2.3	7.6	1.3	≥ 6	p<.001	p<.05
Location	1.9	1.5	2.8	2.1	6.6	1.5	≥ 5	p<.001	p<.05
Tapping Test									
Preferred hand	47.2	6.2	48.2	4.4	52.5	4.7	≥51	p<.05	p<.05
Impairment Index	2.2	0.6	1.6	0.6	0.9	0.4	≤ 1.55	p<.001	p<.05
Peabody Picture Vocabulary Scale	97.1	14.3	95.6	12.8	109.9	14.0	85-115	n.s.	n.s.
Shipley-Institute of Living Scale	85.2	17.4	87.9	10.9	95.8	10.7	≥85	n.s.	n.s.

TABLE 2
Number of Individuals in the Impairment Range According to Halstead-Reitan National Norms

Test	Alcoholics (N=15)	Heroin Abusers (N=15)	Controls (N=12)
Category Test	12	9	3
Tactual Performance Test			
Total time	11	2	0
Location	13	11	1
Memory	10	5	0
Tapping Test	12	9	3
Average Impairment Index	12	6	0

classification: 1=small or absent, 2=middle range, and 3=large. Although all of the sulci would ordinarily be considered within the normal range, obvious gradations were observed among the 42 subjects.

RESULTS

Neuropsychological Functioning

Results of the neuropsychological tests were analyzed in two ways. Duncan's multiple range test was used to compare the heroin abusers and controls and the alcoholics and controls. The number of individuals whose scores fell in the impairment range was evaluated according to established norms.

The Shipley-Institute of Living Scale for Measuring Intellectual Impairment and the Peabody Picture Vocabulary Test were used to ensure that the groups were equated for overall intelligence. No significant difference was found for the Shipley or the Peabody test, indicating that overall intelligence was equated across groups. All comparisons between the alcoholic and control subjects on tests of neuropsychological functioning were significant, as were all but two of the comparisons between the heroin abusers and the controls (see table 1). An important point is that unlike the alcoholic subjects, the opiate abusers performed as well as the controls on the Category Test, which measures abstract reasoning ability. Deficits in abstract reasoning are an especially prominent feature of impairment in chronic alcoholics. Results of a number of studies using the Category Test with chronic alcoholics have shown a remarkable consistency in the frequency of reported impairment (5, 19, 20). Significant neuropsychological impairment was noted for the alcoholics and heroin abusers (table 2), with 75% of the alcoholics and 37.5% of the heroin abusers exceeding the normal range for the Average Impairment Index. A cutoff score suggested by Russell and associates (21) was used to assess overall impairment.

A second set of norms was used to determine the proportion of subjects who fell outside the normal range. Cutoff scores developed in Oklahoma by Vega and Parsons (22), which have been found to discriminate brain-damaged from non-brain-damaged individuals quite well, were applied to the test results. According to these cutoff scores, twice as many alcoholics as heroin abusers once again showed overall impairment. Among the alcoholics, 40% had an Average Impairment Index exceeding the normal range, whereas 20% of the heroin abusers were similarly affected. None of the controls showed overall impairment.

CTT Scans

Statistical analyses of the V/B indices obtained for the three groups indicated that on the whole the alcoholics were not significantly different from the controls, and the opiate abusers were not significantly different from the controls. However, a comparison made between the alcoholics and the heroin abusers indicated a significant trend ($\chi^2=3.40$, df=2, $p<.10$), which was probably due to the large percentage of heroin abusers with small V/B indices (table 3). In a previous study (13) 34 control subjects without a drug history were found to have V/B indices less than 6.0. On-

TABLE 3
Distribution of V/B Index for Alcoholic Subjects, Heroin Abusers, and Controls

	V/B Index					
	<2.4		2.4–4.5		4.6+	
Group	N	%	N	%	N	%
Alcoholics	6	40.0	7	46.7	2	13.3
Heroin abusers	11	73.3[a]	3	20.0	1	6.7
Controls	7	58.3	4	33.3	1	8.3

[a]Significant trend compared with the alcoholics (χ^2=3.40, df=2, p<.10).

TABLE 4
Distribution of Sulci Size for Alcoholic Subjects, Heroin Abusers, and Controls

	Size of Sulci[a]		
Group	1	2	3
Alcoholics[b]	0	8	7
Heroin abusers[c]	4	11	0
Controls	0	9	3

[a]1=small or absent, 2=middle range, 3=large.
[b]There was no significant difference between the alcoholics and controls in size of the sulci (χ^2=1.34, df=2).
[c]The sulci of the heroin abusers were significantly smaller than those of the alcholics (χ^2=11.47, df=2, p<.001) and the controls (χ^2=6.95, df=2, p<.01).

ly 2 of the subjects in the present study exceeded this value—1 alcoholic with a V/B index of 7.60 and 1 heroin abuser with a V/B index of 8.70.

Since a disproportionate number of cases among the opiate abusers had a V/B ratio less than 2.4, it is particularly noteworthy that significantly more of the opiate abusers fell in the group characterized by small or absent sulci, as seen in table 4. The cerebral sulci of the opiate abusers were significantly smaller than those of both the controls (χ^2=6.95, df=2, p<.01) and the alcoholics (χ^2=11.47, df=2, p<.001). These findings suggest that opiates produce specific effects distinct from those seen with cerebral atrophy (enlarged ventricles and sulci).

DISCUSSION

Two recent CTT studies have demonstrated neuroanatomical changes in alcoholics without chronic brain syndrome. Ventricular enlargement was noted in 67% of the alcoholic patients in one study (11), and there was evidence of cerebral atrophy in 58% of the patients in another study (12). However, these studies cannot be taken as estimates of risk for cortical atrophy or intellectual impairment among alcoholics on the whole. Strict selection criteria were not used in these studies; CTT scans were obtained because the patients had displayed clinical criteria warranting further neurological consideration. Additional difficulties with previous CTT studies are the lack of age- and sex-matched control groups and the fact that measurements of the ventricles were linear. For these reasons, previous studies may have overestimated the degree of atrophy actually present among alcoholics. Further, these high rates for alcoholics are in direct contrast to our findings. Based on a V/B index of 10 as indicative of clear atrophy (17), none of our 30 alcoholics and heroin abusers showed atrophy, although 1 alcoholic and 1 control subject exceeded a score of 6.0.

Although patients with chronic brain syndrome or Korsakoff's syndrome exhibit the most striking intellectual impairment, many more alcoholics may suffer from subclinical forms of brain damage. It has been reported in many investigations that alcoholics have normal IQs and intact verbal capabilities, with deficits in abstracting ability as measured by the Category Test (5, 19, 20). The present results are in agreement with these previous findings concerning impairment in alcoholics on the Category Test.

Neuropsychological deficits in association with opiate abuse have previously not been tested in subjects without a history of abuse of other drugs, although polydrug abusers have been reported to be neuropsychologically impaired. Adams and associates (23), studying polydrug abusers of whom about one-third had abused opiates, found deficits on a number of tests, including the Category Test, Tactual Performance Test, and the Tapping Test. In another study, by Grant and Judd (24), 45% of the 66 polydrug abusers examined had impaired neuropsychological functioning as measured by the Halstead-Reitan battery. About one-third of these polydrug abusers had abused heroin, and one-fourth had abused alcohol. It was not possible in these studies to determine the importance of alcohol or opiate abuse alone.

The present investigation of individuals who were almost exclusively abusers of opiates demonstrated that significant neuropsychological impairment may occur as a result of abuse of this class of drugs. Unlike the alcoholics, who frequently exhibited marked impairment on the Category Test, the heroin abusers in the present study did not show this deficit as often or with as large a deviation from controls. Since impairment on this test is thought to correlate with frontal lobe damage or right hemisphere involvement (25), one may speculate that these areas are less vulnerable to the opiates. In other respects, the neuropsychological impairment seen in the opiate abusers was similar to that seen in the alcoholics, i.e., impaired performance on the Tactual Performance Test (Memory and Location) and the Tapping Test. However, overall impairment on these tests (Average Impairment Index) was present in twice as many alcoholics as opiate abusers. Further, using our criteria for probable cerebral atrophy as evidenced by CTT scans, as many alcoholics as opiate abusers showed a tendency toward enlarged ventricles. However, the number of individuals so affected was quite small.

One unexpected CTT finding that may be particularly noteworthy was the statistically significant number of heroin abusers who had very small or visually absent sulci. We can only speculate as to the possible

mechanism that could account for this difference. A number of reports (26–28) have indicated that antigenic sensitization to narcotics or accompanying adulterants may occur. Opiates are not antigenically inert. Morphine, codeine, meperidine, and opium are capable of producing acute allergic responses. Urticaria, angioneurotic edema, acute transverse myelitis, and anaphylactic shock have been reported to occur, presumably as a result of the capacity of opiates to release histamine (29). An allergic phenomenon has also been implicated in acute myoglobinuria seen in association with heroin addiction (30). Acute myoglobinuria is characterized by generalized muscle tenderness and edema. If chronic edema of the brain were a possible extension of these phenomena, resulting in chronic swelling, then the small V/B indices and small or absent sulci observed among the present group of heroin abusers might be a logical prediction. In any case, this finding appears to be worthy of further study.

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Craving for Alcohol in Sober Alcoholics

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The craving for alcohol experienced by 46 abstinent alcoholics was investigated with a structured interview, a personality inventory (the neuroticism Scale Questionnaire), and a mood state adjective checklist (Profile of Mood States). It was found that craving is a continuum, not an all-or-none phenomenon. The intensity of craving was positively correlated with the trait anxiety of the subject and inversely correlated with the duration of abstinence from alcohol. A large proportion of the craving was precipitated by environmental factors and relieved by psychological methods. A variety of mood changes accompanied craving. Only one mood state, anger-hostility, significantly differentiated subjects who experienced mild craving from those reporting severe craving.

"CRAVING" is used in this paper to denote an overpowering desire to drink alcoholic beverages (1). A sizable percentage of alcoholics experience craving before consuming alcohol, but systematic studies regarding this phenomenon are few.

Mello (2) questioned the very existence of the phenomenon of craving. However, Hore (3), in a mail questionnaire study of alcoholics in the United Kingdom, found that 33% of the respondents had definitely experienced craving in the last 7 days. Flaherty and associates (4) showed that periods of craving occurred frequently during the first year of sobriety.

Several investigators have hypothesized the existence of two types of craving: a psychological need to consume alcohol, which is often present before the onset of alcoholism, and a need to consume alcohol to relieve withdrawal symptoms (5). According to Ludwig and associates (6), the withdrawal syndrome and the craving are conditioned responses to a number of introceptive and extroceptive stimuli. Withdrawal symptoms and associated craving that occur as late as two months after termination of drinking have also been described (7). Since the diagnosis of alcohol withdrawal is made on a purely symptomatic basis, its differentiation from anxiety can be complicated. It is difficult to distinguish whether the anxiety experienced with craving is a delayed withdrawal syndrome or anxiety neurosis. It is equally difficult to decide whether the anxiety is primary or secondary to the craving experience. Other mood states also have been associated with craving. Hore (3) found that craving occurred under various circumstances, including social and drinking situations and altered mood states. Sixty-nine percent of his subjects described craving in terms of physical symptoms, 40.8% in terms of mental symptoms, and others claimed miscellaneous symptoms. In the Flaherty and associates study (4), craving spells corresponded with deep depressions and late withdrawal symptoms.

The association between alcoholism and affective disorders was investigated by Winokur and associates (8–11), who reported a possible underlying affective change in craving. Craving has also been studied under carefully controlled laboratory conditions using stimulation and simulation techniques. On the whole, these experiments demonstrated that craving is not always associated with drinking behavior. The majority of these studies investigated the craving experience that followed the first drink rather than the craving that preceded the drinking (2). It is debatable whether such data support the existence of the phenomenon of craving (12).

The present investigation was conducted to study the characteristics of craving and the factors that influence its appearance and disappearance.

METHOD

The subjects, 34 men and 12 women residents of three hostels for recovered alcoholics, were interviewed after the study was explained fully and they gave informed consent to participate. Only subjects given a "definite diagnosis of alcoholism," using the criteria described by Woodruff and associates (13, pp. 205–206) were included in the study. Craving was defined as the desire to drink alcohol before the consumption of the first drink. Assessments were obtained for each subject during a structured interview

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with a psychiatrist. A questionnaire on craving and an alcoholism questionnaire were constructed in order to systematically record information regarding the nature of craving, the duration of drinking patterns, complications of alcoholism, demographic information, etc.

Subjects were asked to complete the Neuroticism Scale Questionnaire (NSQ) and the Profile of Mood States (POMS). They were instructed to fill out the POMS according to the mood and feelings experienced during the most recent craving episode. Data for each participant were collected once.

The NSQ is a brief self-report inventory that measures the degree of an individual's neuroticism. In addition to a total neuroticism score, the 40-item test provides measures of tendermindedness, depression, submissiveness, and anxiety. The POMS is a 65-question self-report inventory that measures six clearly defined dimensions of mood: tension-anxiety, depression-dejection, anger-hostility, vigor-activity, fatigue-inertia, and confusion-bewilderment.

RESULTS

Subject Characteristics

Frequency tallies were calculated for each of the items on the alcoholism and craving questionnaires, and characteristics of the subjects emerged as follows: of the 46 subjects interviewed, 34 (74%) were male and 12 (26%) were female. Twenty-nine of the 46 subjects (63%) were separated or divorced, 11 (24%) were single, and 4 (9%) were married.

The mean age of the group was 37.8 years (SD=10.5). The alcoholism questionnaire indicated that the mean age of alcoholism onset was 25.9 years (SD=9.7), followed by a mean duration of alcoholism of 12.2 years (SD=8.7). The mean period of current abstinence from alcohol was 6.0 weeks (SD=5.3).

Precipitating Factors and Severity of Craving

The craving questionnaire was designed to indicate the various external factors that may precipitate the craving for alcohol.

Table 1 represents a subset of the craving questionnaire and gives the frequencies of subjects responding positively to each question. As the table indicates, craving appeared to be induced by a variety of factors, particularly unpleasant ones. It should be noted that the categories in table 1 are not mutually exclusive and that 15 subjects answered "yes" to both of the first two items and 11 answered "yes" to the first three questions.

The craving questionnaire included a series of items constructed to test our hypothesis that craving can be seen as a continuum (see table 2).

Response frequencies did not occur in a bimodal pattern, the expected distribution if craving were an all-or-none phenomenon. Instead, craving seemed to range from "none" to an intense presence so pervasive that drinking inevitably ensued. Thus it appears that craving can be conceptualized as a continuum.

TABLE 1
Responses to Questions Regarding Precipitating Factors

Precipitant	N	%
External events related to alcohol drinking	19	48
External non-alcohol-related events of an unpleasant nature	34	85
External non-alcohol-related events of a pleasant nature	23	58
Depression unrelated to external events	7	18
Elation unrelated to external events	4	10

TABLE 2
Frequency of Positive Responses to Questions on Severity of Craving

Severity	N	%
Never experienced craving (noncraver)	6	14
Recognized craving spontaneously but can ignore it (mild craver)	14	32
Recognized craving spontaneously but can keep it under control with great effort (moderate craver)	16	36
Craving invariably leads to drinking (severe craver)	8	18

TABLE 3
T Score Means for Profile of Mood States[a]

POMS Dimensions	Mild Cravers (N=14)	Moderate Cravers (N=16)	Severe Cravers (N=8)
Tension-anxiety	50.7	54.9	57.3
Confusion-bewilderment	51.7	51.1	55.6
Depression-dejection	56.1	55.6	61.4
Fatique-inertia	53.5	49.7	52.1
Vigor-activity	49.6	48.1	57.4
Anger-hostility	57.3	50.9	61.6

[a]The POMS was not given to the noncravers.

Duration of Alcoholism

An attempt was made to determine if the intensity of craving was related to the duration of alcoholism. It was found that noncravers had been alcoholics for a mean of 13.0 years, the mild cravers for 14.2 years, the moderate cravers for 10.3 years, and the severe cravers for 11.3 years. A one-way analysis of variance indicated no significant difference among the groups.

Duration of Current Abstinence

The relationship between the intensity of craving and the duration of current abstinence from alcohol was also investigated. The mean duration of abstinence for each group was as follows: noncravers, 19.4 weeks; mild cravers, 7.9 weeks; moderate cravers, 5.2 weeks; and severe cravers, 3.0 weeks. Initial inspection of the data included possible nonhomogeneity of group variances, and the Cochran C test confirmed this. Therefore, the data were transformed before the

TABLE 4
T Score Means for the Neuroticism Scale Questionnaire

NSQ Dimension	Noncravers (N=6)	Mild Cravers (N=14)	Moderate Cravers (N=16)	Severe Cravers (N=8)
Tendermindedness-overprotected	48.0	41.9	51.8	50.5
Depression-overseriousness	49.8	51.1	52.1	47.1
Submissiveness-dependence	53.7	49.6	55.9	57.0
Anxiety	51.8	60.3	63.9	66.1

one-way analysis of variance was calculated. A significant difference was found between the groups, $F(3, 40)=4.77$, $p \leq .01$, indicating a relationship between intensity of craving and duration of abstinence from alcohol. The relationship was inverse, with the severity of craving decreasing as the period of abstinence increased.

POMS Data

The POMS subscale scores were converted into T scores, and group means and standard deviations were calculated for each of the craving groups (see table 3).

A series of one-way analyses of variance was used to compare the three groups of cravers on each of the POMS subscale measures. Only the anger-hostility measure showed a significant difference among the craving groups, $F(2, 35)=3.33$, $p<.05$. It is of interest that, except for the fatigue-inertia measure, each of the POMS subscale measures was generally higher in the severe craving group than in the mild and moderate craving groups. It is also noteworthy that for the severe cravers the group means for the depression-dejection and anger-hostility subscales were more than 1 SD above the expected group mean.

The relationship between the severity of craving and POMS subscale measures was examined by means of a chi-square test comparing the craving groups in terms of the frequency of T scores above 60 across all subscales. The relationship was found to be highly significant ($\chi^2=15.22$, $df=2$, $p<.001$). The contingency coefficient assessing the degree of relationship between intensity of craving and the POMS scores was .25.

Subjects experiencing a severe craving for alcohol tended to report more severe mood changes than subjects experiencing mild and moderate craving. Finally, the mild, moderate, and severe craving groups differed significantly in mood changes involving anger-hostility.

NSQ Data

Raw data from the NSQ were converted into T scores (see table 4). A series of one-way analyses of variance was used to determine any differences among the craving groups with respect to the four NSQ measures. The craving groups were found to differ significantly only on the anxiety measure $F(3, 40)=5.03$, $p<.005$. The relationship between craving and anxiety is direct—the more intense the craving for alcohol, the greater the anxiety as measured by the NSQ. On the anxiety measure, the group mean for noncraving subjects is well within the expected normal range, while the group means for all three craving groups were at least moderately higher than the expected values. It can be concluded that among these subjects a strong relationship exists between the severity of craving and trait anxiety.

DISCUSSION

The cornerstone of the medical model of alcoholism is Jellinek's often quoted work (14). He has brought together several concepts concerning the nature of alcoholism as a disease: genetic factors, the natural course, and milestones in the natural course (craving, loss of control, amnesic spells, irreversibility, etc.). Thus he has erected a strong pillar of support for a biological model. However, several investigators have questioned the validity of the concepts used to support this pillar. Pattison (12) recently wrote an excellent review of these articles.

In the past, craving for alcohol was considered one of the strong supports for the disease concept. It was defined by many researchers as a sudden, overwhelming desire to consume alcohol that resulted from internal and possibly biological factors. However, several recent studies have supported the concept that craving is largely a conditioned response to internal and external stimuli (4, 6). Our data also tend to support this view.

The alcoholic and, to a lesser extent, the social drinker have many reasons for using alcohol—as an anxiolytic, antidepressant, euphoriant, aphrodisiac, social catalyst, confidence booster, and a means to suppress disturbing memories. Consequently, the sober alcoholic is confronted with numerous cues, both external and internal, that are capable of eliciting the craving response. The majority of subjects in the present study reported external cues as eliciting a craving for alcohol. These consisted of alcohol-related cues (48%), such as an empty beer can or a commercial for an alcoholic beverage, and non-alcohol-related events of an unpleasant (85%) or pleasant (58%) nature. Only 11 subjects reported craving associated with endogenous mood swings.

It is well known that the conditioned response extinguishes in the absence of reinforcement. We found that the frequency and intensity of craving decreased significantly with the duration of sobriety. In time, the sober alcoholic appears to learn more effective and re-

warding non-alcohol-related coping mechanisms to deal with stress. His immigration to a society of recovered alcoholics removes a number of drinking cues from his life, insulates him from other cues, propagates new insight into his drinking behavior, and provides support through this stressful period. Finally, the reminders of the "drinking days" lose their edge and slowly fade.

Our examination of sober alcoholics revealed subjects in different stages of deconditioning, ranging from those who experienced no craving (14%) to those who experienced severe craving for alcohol (18%). Such a cross-section further supports the hypothesis that craving is not necessarily an all-or-none phenomenon of a biological nature but a continuum that may be explained in terms of conditioning theory. However, our results do not rule out the possibility of a constitutional predisposition to such conditioning.

A majority of the subjects (68%) reported that association with nondrinking alcoholics was the most effective remedy for craving. Thus there is strong evidence that a substantial portion of craving is not a biological phenomenon resistant to psychological treatment. Fifteen percent of the subjects found anxiolytic drugs helpful to combat craving and 7% found relaxation useful. A large proportion (58%) used other methods including "working off steam," jogging, and having sex. This may be explained by the finding that the anger-hostility mood state differentiated mild from severe craving and that sublimation is known to be an effective means of dealing with anger. However, for 18% of the subjects craving was alcohol-specific and could be relieved only by alcohol.

An obvious corollary of the foregoing discussion is that a person who has a greater "need" for alcohol, has difficulty in keeping the craving response under control, is slow to learn new coping mechanisms, and has interpersonal difficulties, is more likely to experience severe craving. The strong relationship found between trait anxiety as measured by NSQ and the severity of craving substantiates this conclusion.

Some support for the association between depressive mood changes and alcoholism was found. Eighty-five percent of the subjects related craving to unpleasant environmental factors, 18% to endogenous depression, and 10% to endogenous elation. The mean POMS depression score for the severe cravers was more than one standard deviation above the expected mean.

In conclusion it can be said that craving, to a large extent, is a conditioned response to environmental cues. Craving tends to diminish in intensity with time and can be most effectively dealt with by psychological methods. Craving is not an all-or-none phenomenon but a continuum, with an absence of desire to drink at one pole and an intense desire at the other. Craving is most intense during the early days of sobriety and then diminishes progressively. Anxious people tend to experience more intense craving than nonanxious people.

Our data do not address the issue of whether there are two types of craving: a symbolic and a withdrawal craving (5, 6). Although there is some tentative support for the symbolic-learned hypothesis, the results do not disprove the existence of withdrawal craving. In addition, the degree to which biological factors are involved in craving remains unanswered, but there is a strong suggestion that conditioning plays a significant role.

The results reported herein should be generalized with caution to alcoholics as a group because hostel-facility clientele differ from alcoholics residing in other types of facilities on a number of psychological variables.

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Drug Abuse Among Alcoholic Women

BY MARC A. SCHUCKIT, M.D., AND ELIZABETH R. MORRISSEY, M.A.

Of 293 women in an alcoholic treatment center, two-thirds had received prescriptions for drugs of potential abuse, usually hypnotic and antianxiety drugs. One-third of the women admitted abusing substances; 80% of these subjects got prescriptions for potential drugs of abuse while actively abusing. These figures are alarming in light of the lack of evidence that antianxiety drugs, hypnotics, and stimulants are effective in treating alcoholics and drug abusers. Drug-misusing women in this sample reported more suicide attempts and early antisocial problems and had received more psychiatric care than nonabusers. The authors urge that hypnotics, antianxiety drugs, and stimulants almost never be prescribed to outpatient alcoholics and that analgesics be prescribed only with great care.

PRESCRIPTION DRUG MISUSE or deliberate abuse may be a relatively common phenomenon, especially among middle- to upper-socioeconomic status (SES) individuals with complex medical histories (1). Misuse of prescription medications is even more prevalent in clinical samples of multidrug users and alcoholics. A recent review (2) indicates that somewhere between 5% and 50% of alcoholics use other drugs (average around 20%), with the chance of misuse being greater among young alcoholics (3) and in older middle-SES alcoholic women. The drugs usually misused are depressants (4, 5), which are usually obtained from physicians by prescription (5).

Most alcoholics come to their family physician with medical or emotional complaints such as sleeplessness and anxiety, a pattern said to be even more frequent among alcoholic women than men (6). The physician may respond to these nebulous complaints by prescribing hypnotics or antianxiety drugs. This is unfortunate because these two classes of drugs have not been proven to be effective when used chronically and have serious potential dangers for addiction and adverse drug interactions.

Insomnia is reported by most alcoholics and may continue for many months, either as part of the chronic alcohol-abusing picture or perhaps as a secondary abstinence phenomenon (7). However, sleeping medications tend to lose their effectiveness after 10 days to two weeks (8) and are dangerous when mixed with alcohol because they potentiate respiratory depression, with possible subsequent accidental or deliberate overdose (4). They may also interact with alcohol to increase levels of cognitive impairment in the elderly (9). Thus, hypnotics are almost contraindicated in outpatient alcoholics, as their period of effectiveness is too short and their physical dangers and abuse potential too great to justify their use.

All of the problems noted for hypnotics generalize to potential difficulties with antianxiety medication. Although anxiety can be seen during active drinking or over an extended period of time in abstinent alcoholics, controlled investigations have not demonstrated antianxiety drugs to be effective for longer than two to three weeks (10). At the same time, these depressant medications can potentiate the adverse effects of alcohol on cognition and autonomic nervous system functioning (9, 11). If these substances are taken frequently enough over a long period of time, physical dependence can be produced and, for the alcoholic, a complex withdrawal syndrome can ensue (12, 13). This is an especially difficult problem with meprobamate, a drug that also carries a high potential for overdose deaths (14).

Stimulants are the third type of drug frequently received by alcoholic outpatients. These drugs, usually prescribed for depression or lethargy (symptoms frequently seen in alcoholics), are ineffective and frequently produce subjective feelings of depression and lethargy themselves (15). Finally, care must also be taken in giving analgesics to alcoholics because of their potential drug-drug interactions with alcohol, which include increased respiratory depression and possible death (9).

The drugs most frequently abused by alcoholics are stimulants, opiates, hypnotics, and antianxiety drugs. In each instance the potential benefits are extremely limited and the risks are relatively high. Yet, due to a variety of factors, most physicians prescribe these medications to individuals who might be identified as substance misusers. We will examine one specific population of alcoholics in an attempt to document the nature and dimensions of this problem.

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METHOD

The 293 subjects were drawn from 339 consecutive admissions to the King County (Seattle) Detoxification Facility in 1976. Every woman admitted was approached by a trained interviewer within 24 hours; the interview was delayed if signs of confusion were present. Four percent of the original 339 admissions refused to complete the interview, and an additional 10% were either too psychologically ill or signed out before an interview could be done.

Systematic material was collected on social and developmental histories, psychiatric syndromes as defined by Woodruff and associates (16), alcohol and drug patterns, and family history of psychiatric disorders. All data were obtained from self-reports, but the clinical records of patients were reviewed if inconsistencies occurred within the interview.

A detailed history of substance use was taken. Alcoholism or drug abuse was defined as the occurrence of a major life problem related to the use of a substance, including one of the following: a marital separation or divorce related to drugs or alcohol, multiple alcohol or drug related arrests, physical evidence that the substance had harmed health, loss of a job because of substance use, or, for drugs other than alcohol, intravenous use. As outlined by other authors (16), antisocial personality refers to the occurrence of pervasive antisocial problems beginning before age 16, and primary affective disorder indicates a persistent depression lasting for two or more weeks in the absence of preexisting alcoholism, drug use, or psychiatric disorders.

For each drug or medication, including alcohol, the individual was asked her age at first use, the frequency of occurrence of related problems, and the ages at which the first and the most recent problems occurred. In the cases of antianxiety drugs, barbiturates, amphetamines, and opiates, the patient was asked if the drug had ever been received by prescription and, if so, whether this had happened during a time when she was actively abusing other drugs or alcohol. Responses to these questions were validated by referring to the patients' reports of ages at which alcohol-related problems had occurred.

For purposes of data analysis, the women in the sample were divided into four groups. First, those who reported abusing antianxiety drugs, barbiturates, amphetamines, or opiates were separated from those who had not. Then the drug abusers were subdivided into those who abused prescription drugs and those using only "street" drugs. The nonabusers were divided according to whether they had received prescriptions for analgesics, hypnotics, antianxiety drugs, or stimulants.

Data from the interviews were numerically coded and analyzed by computer. Comparisons between groups were made using chi square, corrected for continuity where appropriate, and Student's t test for comparing means.

TABLE 1
Background Characteristics of 293 Women with Alcohol Problems

Variable[a,b]	Patient Group: Drug Abusers: Prescription (N=95)	Drug Abusers: No Prescription (N=17)	Nonabusers: Prescription (N=108)	Nonabusers: No Prescription (N=73)
Percent	32	6	37	25
Mean age (years)	36.8[c,d,e]	25.9[d,e]	45.9	45.9
Marital status				
Never married	14	35	4	7
Married	20	0	34	22
Cohabiting	14	18	9	18
Separated/divorced/ widowed	52	47[d,e]	53	53
Married 3 or more times	34[c,e]	0	29	16
Social class I, II, or III[f]				
Father	21	20	23	15
Husband	22	25	20	18
Patient	20	0	30	22
Race				
Caucasian	82	59	75	60
Native American	13	35	18	29
Black	1	6	6	7
Other	4[e]	0[d]	1	4
Mean high school GPA[g]	2.3[e]	2.3	2.5	2.7
Antisocial problems				
Suspended/expelled from school	33[d,e]	41[d,e]	5	7
Runaway	41[d,e]	53[d,e]	13	14
Delinquency	32[d,e]	53[d,e]	5	6
Reform school	32[d,e]	53[d,e]	6	8
Incorrigible	42[d,e]	59[d,e]	6	10

[a]In percents except when specified as mean.
[b]Statistical comparisons of percentage differences based on chi-square; statistical comparisons of differences between means based on Student's t test.
[c]Difference from no-prescription drug abusers is statistically significant ($p<.05$).
[d]Difference from prescription nonabusers is statistically significant ($p<.05$).
[e]Difference from no-prescription nonabusers is statistically significant ($p<.05$).
[f]Based on Hollingshead's Two-Factor Index of Social Position (unpublished monograph, Yale University Department of Sociology, 1957).
[g]For those who attended high school.

RESULTS

Two-thirds of the subjects had received the relevant prescription drugs: 57% received hypnotic/antianxiety medications, 27% opiates, and 22% stimulants (usually amphetamines or methylphenidate). Of the entire sample, approximately one-third met the criteria for abusing substances, including 29% who abused depressants, 27% stimulants, and 16% opiates. Eighty percent of the drug-abusing subjects who used depressants had received prescriptions for these drugs while abusing. Similarly, over half of the opiate abusers and 40% of the stimulant abusers had received prescriptions while actively abusing. Thus, between two-thirds and 90% of abusers who had received prescriptions had received them while actively abusing some substance.

Table 1 outlines the demographic and antisocial backgrounds of the four groups. The two drug-abusing populations were relatively young, the least likely to be married and most likely to have never married (es-

TABLE 2
Alcohol and Drug Use in 293 Women with Alcohol Problems

Variable[a,b]	Patient Group: Drug Abusers: Prescription (N=95)	Drug Abusers: No Prescription (N=17)	Nonabusers: Prescription (N=108)	Nonabusers: No Prescription (N=73)
Drinking patterns				
Daily	40	53	32	32
Usually alone	45	24	44	32
Binges	74[c,d]	71	49	42
Mean number of drinks/day	14[c,d]	18[c,d]	9	9
Drinking history				
Blackouts	87[c,d]	94[c,d]	66	47
Missed work	53[c,d]	71[c,d]	29	24
Outpatient treatment	55[c,d]	41	31	22
Withdrawal	68[c,d]	76[c,d]	45	41
Liver damage	28[d]	29	17	14
Hospitalization	44[c,d]	24	29	22
Drug use				
Depressants	81[c,d,e]	53[c,d]	—	—
Stimulants	63[c,d]	88[c,d]	—	—
Opiates	44[c,d]	35[c,d]	—	—
Cocaine	27[c,d]	35[c,d]	0	0
Hallucinogens	40[c,d]	41[c,d]	1	1
Marijuana	70[c,d]	94[c,d]	32	25
Intravenous drugs	30[c,d]	35[c,d]	0	0
Mean number of drugs used	3.3[c,d]	3.5	1.0	1.0
Drug-related problems[f]				
Separation/divorce	30	35	0	0
Loss of job	14	0	0	0
Medical attention	80	25	0	0
Withdrawal	74	75	0	0
Arrests	23	25	0	0

[a]In percents except when specified as mean.
[b]Statistical comparisons of percentage differences based on chi-square; statistical comparisons of differences between means based on Student's t test.
[c]Difference from prescription nonabusers is statistically significant ($p<.05$).
[d]Difference from no-prescription nonabusers is statistically significant ($p<.05$).
[e]Difference from no-prescription drug abusers is statistically significant ($p<.05$).
[f]Of those who used drugs.

TABLE 3
Psychiatric Diagnosis and Mental Health Problems of 293 Women with Alcohol Problems

Variable[a,b]	Patient Group: Drug Abusers: Prescription (N=95)	Drug Abusers: No Prescription (N=17)	Nonabusers: Prescription (N=108)	Nonabusers: No Prescription (N=73)
Primary diagnosis				
Alcoholism	29	41	62	70
Drug abuse	18	6	0	0
Antisocial personality	30	41	3	3
Affective disorder	18	12	17	4
Other	0	0	1	1
No diagnosis	5[c,d]	0[c,d]	17	22
Psychological problems				
Depression	33[c,d]	18	24	6
Outpatient treatment	56[c,d]	41	34	20
Hospitalization	25[c]	12	15	11
Suicide attempts	62[c,d]	59[c,d]	28	20
Suicide attempts while abusing drugs[e]	39[c,d]	0	3	0
Familial alcoholism				
Mother	17[c]	29[c,d]	7	3
Father	37[c,d]	35[c]	22	11
Brother(s)	17	12	12	16
Sister(s)	6	18	12	10

[a]In percents.
[b]Statistical comparisons based on chi-square.
[c]Difference from no-prescription nonabusers is statistically significant ($p<.05$).
[d]Difference from prescription nonabusers is statistically significant ($p<.05$).
[e]Of those who attempted suicide.

pecially the non-prescription drug abusers). Data not shown in the table indicate that 34% of abusers of prescription drugs, 29% of those who had received prescriptions but did not abuse them, none of the street drug abusers, and 16% of the nonabusers had been married three or more times. The women abusing nonprescription medications included the highest proportion of minorities, especially Native Americans, and had the lowest present social class.

The two drug-abusing populations demonstrated more severe alcohol histories, as shown in table 2. These women were more likely to drink daily, more frequently reported binges (drinking and disregarding usual activities for three or more days at a time), and had higher rates of all alcohol-related difficulties, including blackouts (having been alert and oriented but forgetting what one had done while intoxicated). The two groups of abusers reported the highest average number of drinks per day and were more likely to receive both outpatient and inpatient treatment for alcohol problems.

Both of the drug-abusing populations took multiple drugs, with the prescription drug abusers more likely to abuse depressants or opiates (usually oral codeine) but not stimulants. Drug-related problems were seen fairly equally for the two groups, but prescription abusers were more likely to have received medical attention for their drug problem (usually not the overt reason for seeing the prescribing physician). The prescription and nonprescription drug abusers had used each substance on multiple occasions: depressants an average of 144 and 181 times, stimulants 313 and 179 times, opiates 176 and 50 times, and cocaine 23 and 25 times, respectively.

Primary psychiatric diagnoses were based on the age of occurrence of the first appearing psychiatric disorder. As can be seen in table 3, subjects with histories of drug abuse were less likely to receive an alcoholic label and more likely to be noted as antisocial personalities or drug abusers. Almost all antisocial and drug-abusing women were secondary alcoholics. Primary affective disorder was seen in 10%–20% of each group with the exception of the nonabusing women who had not received prescriptions. Between 0% and 20% of the sample fulfilled no diagnostic criteria—most of them had alcohol-related problems that were not severe enough to meet the rather rigid criteria for this diagnosis.

Consistent with the psychiatric labels are the reports of sadness not pervasive enough to meet the criteria for affective disorder, which was seen in approximately one-quarter of the sample, especially in those having received prescriptions. Although suicide attempts were seen in all groups, they were threefold higher among the drug abusers. Also, a substantial minority of patients received outpatient or inpatient care for psychiatric problems—a trend more frequently noted for drug abusers.

The most disturbed family histories of psychiatric disorders were seen for drug abusers for all family diagnoses, especially alcoholism, as shown in table 3. In addition, 5% of the brothers and sisters of both drug-abusing groups, versus 1% of the remainder, were themselves drug abusers.

DISCUSSION

Two-thirds of the subjects received prescriptions for antianxiety drugs, hypnotics, stimulants, or opiates. Almost half of those who received prescriptions admitted to abusing them. Those drug-abusing women tended to subdivide into the younger, somewhat more antisocial "street" drug abusers and the older, relatively higher status, primarily white, abusers who took only prescription medications (17). Despite these demographic differences, both samples of abusers demonstrated an elevated rate of single marital status and high rates of antisocial problems in school, at home, and with police. In addition, the two drug-abusing populations had the heaviest alcohol use patterns, the most pervasive histories of alcohol-related life problems, and the highest rates of subsequent drug-related serious social and medical consequences.

Psychiatric disturbances, including suicide attempts, were three times as high for the abusing groups and must be considered by physicians dealing with this population. Although many subjects tended to report depression, relatively few met the criteria for primary affective disorder (16). The distinction between sadness in the midst of drug or alcohol abuse and a depressive episode of a severe nature occurring irrespective of substance problems must be kept in mind because primary affective disorder patients will probably respond to antidepressant medications, whereas the sadness noted secondary to substance abuse tends to be evanescent and does not require formal treatment (18).

Women demonstrating drug-abuse problems are more likely to consult a physician for psychological difficulties, including insomnia, depression, suicide attempts, and family or interpersonal disorganization (19), as is consistent with the data related in this study. Thus, physicians in practice are faced with a difficult dilemma. The average alcoholic is a middle-class individual who does not complain to the physician of alcohol problems but rather physical, emotional, and life difficulties. To diagnose alcoholism, one needs to have a high index of suspicion, and this is probably even more important in diagnosing drug abuse or substance misuse among alcoholics. These women are more likely to live alone, to have histories of early antisocial problems, and to report to the physician with depression, insomnia, or pain.

Hypnotics and antianxiety drugs are contraindicated in the average alcoholic. Their length of action is too short in comparison to the amount of time the patient will have symptoms to make them of use, and alcoholics should be considered at high risk for drug misuse problems. Treatment of anxiety can usually be handled by referral to an alcoholism treatment clinic, gentle understanding by the physician who sympathizes with the patient's problems, education about the probable physiologic basis of the symptoms, and warnings that any medications will cause more harm than good. Insomnia will improve with abstinence, but normal sleep patterns may not appear for a period of months (7). There are a number of nonhypnotic approaches to handling sleep problems, including exercise, establishing a rigid sleeping pattern, avoiding naps during the day, and shunning caffeinated beverages (20). Patients can be given these aids without the dangers inherent in hypnotics.

In summary, the average alcoholic is a middle-class worker or housewife consulting the physician for physical or emotional problems (6). These people, who make up one-third of the hospitalized general medical or surgical patients (21), are at high risk for abuse of hypnotics, antianxiety drugs, stimulants, and analgesics. The first three drugs should *almost never* be prescribed to this group on an outpatient basis and the latter only with great care.

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Psychiatric Problems in Women Admitted to an Alcoholic Detoxification Center

MARC A. SCHUCKIT, M.D., AND ELIZABETH R. MORRISSEY, M.A.

The authors studied 293 consecutive female admissions to a public detoxification facility. One-half had a primary diagnosis of alcoholism; the rest had a primary diagnosis of affective disorder (14%), antisocial personality (14%), drug abuse (6%) or did not meet the criteria for alcoholism (13%). Each subgroup had unique social backgrounds and characteristic histories of involvement with alcohol, drugs, and antisocial problems. The primary alcoholic woman in the detoxification center resembles the primary alcoholic man in the same setting. The authors underscore the importance of socioeconomic strata and primary versus secondary diagnosis in understanding the prognosis for alcoholics.

THE COURSE OF ALCOHOLISM in women has been studied primarily in middle-class samples (1, 2), whereas most studies of male alcoholics deal with lower socioeconomic status (SES) groups, a factor that might explain many of the reported differences between alcoholic men and women (1). The two sexes also differ on histories of prior psychiatric illness, i.e., on diagnoses of primary versus secondary alcoholism (1, 3). The three predominant diagnostic groups of alcoholics—those with primary alcoholism, those with primary affective disorder and secondary alcoholism, and those with primary antisocial personality and secondary alcoholism—have different clinical pictures and prognoses (4-7). A third factor affecting comparisons of male and female alcoholics is the differential processing by public agencies; the police are less likely to confront and arrest a woman than a man for alcohol problems (8-11).

All of these factors contribute to the fact that alcohol problems in women, especially women who are public inebriates, are not clearly understood. Therefore we have studied the course of alcohol problems and the potential importance of diagnostic subgroups in women entering the King County Detoxification Center in Seattle, Washington, a facility that maintains a population of approximately 8% women.

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METHOD

Of the 339 consecutive female admissions to the detoxification center between April and September 1976, we studied 293 (86%); 13 women refused to take part,

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TABLE 1
Demographic and Background Characteristics of 29 Women Seen in a County Alcohol Detoxification Center

Variable[a,b]	Primary Diagnosis				
	Alcoholism (N=154)	Drug Abuse (N=18)	Affective Disorder (N=40)	Antisocial Personality (N=40	No (
Percent of total	53	6	14	14	
Mean age (years)	45.4[c,d,e]	33.3[d,f]	41.8[e]	29.9[f]	
Race					
Caucasian	70.1	83.3	80.0	73.2	
Native American	22.7	16.7	15.0	17.1	
Black	3.9	0	2.5	4.9	
Other	3.3	0	2.5	4.9	
Marital status					
Single	4.5	27.8	2.5	19.5	
Married	24.7	11.1	22.5	12.2	
Cohabiting	13.6	22.2	12.5	17.1	
Separated/divorced	48.0	38.9	50	36.6	
Widowed	9.1[c,e,f]	0[d,f]	12.5[e,f]	14.6[f]	
Lives alone	37.0	27.8	37.5	29.3	
Resides on skid row	9.9	0	5.1	21.6[f]	
No religious preference	10.4[c,e]	33.3	10	26.8	
Employed	19.5	16.7	17.5	19.5	
Pregnancy history	76.0	55.6	77.5	61.0	
Deceased child	17.4[e]	30.0	15.2	37.0	
Handicapped child	9.2[e]	20.0	15.2	29.6	
Miscarriage	37.0	27.8	45.0	43.9	
Abortion	13.6	33.3	22.5	24.4	
Class of Origin I, II, or III	18.3	26.7	31.6	18.4	
Social Mobility					
Upward	27.6	0	32.4	14.7	
None	44.9	41.7	38.2	38.2	
Downward	27.6[e]	58.3[d,f]	29.4	47.1	

[a]In percents except when specified as mean.
[b]Statistical comparisons of percentage differences based on chi-square; statistical comparisons of differences between means based on Student's
[c]Similarity to drug abuse group statistically significant ($p \leq .05$).
[d]Similarity to affective disorder group statistically significant ($p \leq .05$).
[e]Similarity to antisocial personality group statistically significant ($p \leq .05$).
[f]Similarity to no-diagnosis group statistically significant ($p \leq .05$).

and 33 women were excluded during the study because of premature discharge or such serious organic brain syndromes that they could not be interviewed. Each eligible woman was given a structured interview covering demographic background, milestones of drinking, psychiatric history, drug use history, levels of social functioning, and family history of psychiatric and substance abuse problems.

The subjects were divided into groups based on their primary psychiatric diagnoses, which were assigned by the age of first occurrence of their first major psychiatric disorder. Alcoholism was defined as the occurrence of any one of a number of alcohol-related problems (12): a marital separation or divorce, multiple arrests, a job loss or layoff or inability to carry out usual tasks around the home, or physical evidence that alcohol had harmed their health. Drug abuse was defined as the presence of a similar series of drug-related problems or the use of intravenous drugs. The criteria for primary affective disorder followed those outlined by Woodruff and associates (3): a period of depression lasting at least two weeks and accompanied by changes in body functioning (e.g., insomnia, constipation, lethargy, etc.) as well as in mind functioning (e.g., inability to concentrate, mixed-up thoughts, loss of interest in things) (13). The diagnosis of a personality or sociopathy was established if, age 16, the woman had had antisocial problem of the following: family (e.g., running awa corrigibility), school (e.g., repeated truancy tiple suspensions and expulsions), police (e. tiple arrests), and peers (e.g., using weapons ing someone seriously in a fight) (14). Bec definition requires antisocial behavior in all areas prior to age 16, it was relatively easy t guish between primary sociopathy and antis havior of a limited nature secondary to alcoho misuse (6). Diagnostic criteria for other ps syndromes such as schizophrenia, Brique drome, anxiety neurosis, etc., were extracted work of Woodruff and associates (3) and Spi associates (15).

The women were divided into groups acc primary diagnosis, and their backgrounds w pared. An additional three psychiatrically ill two meeting the criteria for schizophrenia (3) having an unknown psychiatric disorder (schizophrenia), were excluded from the data a cases that would add little to the comparisons. cal analyses were made using chi square ana

TABLE 2
Antisocial and Drug Use History of Women Seen in a County Alcohol Detoxification Center

Variable[a,b]	Primary Diagnosis				
	Alcoholism (N=154)	Drug Abuse (N=18)	Affective Disorder (N=40)	Antisocial Personality (N=40)	No Diagnosis (N=38)
Mean grade completed	10.8[c,d]	11.7[d]	12.1[d,e]	9.9	10.8
Mean high school GPA	2.57[d]	2.47[d]	2.4[d,e]	2.0[e]	2.7
School problems					
Failed course	26.3[d]	17.6[d]	27.0[d]	62.5[e]	10.0
Suspended/expelled	7.8[d,f]	38.9[c,e]	5[d]	58.5[e]	7.7
Truancy	21.4[d]	38.9[e]	22.5[d]	63.4[e]	7.7
Repeated one or more grades	16.9	27.8	10	17.1	23.1
Family problems					
Runaway	17.5[d]	33.3[d,e]	15[d]	75.6[e]	5.1
Incorrigible	10.4[d,f]	50[c,e]	12.5[d]	78.0[e]	5.1
Legal problems					
Reform school	9.7[d]	23.5[d]	5[d]	68.3[e]	5.1
One or more misdemeanors	14.3[d,f]	52.9[e]	25[e]	43.9[e]	5.1
One or more felonies	4.5[d]	0[d]	2.5[d]	39.0[e]	0
Use of alias	3.2[d]	11.1	7.5[d]	36.6[e]	2.6
Prostitution	3.9[d]	11.1[d]	0[d]	56.1[e]	2.6
Acceptance of food or alcohol in exchange for sex	1.9[d]	5.6	5.0	19.5[e]	0
Mean age at first juvenile arrest	14.9[d]	13.2	14.0	13.4	15.0
Drugs used					
Depressants	13.6[c,d,f]	77.8[c,e]	42.5[d,e]	75.6[e]	7.7
Stimulants	11.0[d,f]	83.3[c,e]	22.5[d,e]	85.4[e]	5.1
Opiates	7.1[d,f]	61.1[c,e]	10[d]	53.7[e]	0
Hallucinogens	3.9[d,f]	50[c,e]	12.5[d]	61.0[e]	5.1
Cocaine	1.3[d,f]	38.9[c,e]	5[d]	48.8[e]	2.6
Intravenous drugs	1.9[d,f]	55.6[c,e]	5[d]	46.3[e]	0
Drug-related problems	1.3[d,f]	83.3[c,d,e]	5[d]	48.8[e]	0
Mean number of drugs used	1.6[c,d,f]	3.9[c,e]	2.2[d]	4.5[e]	1.5

[a]In percents except when specified as mean.
[b]Statistical comparisons of percentage differences based on chi-square; statistical comparisons of differences between means based on Student's t test.
[c]Similarity to affective disorder group statistically significant ($p \leq .05$).
[d]Similarity to antisocial personality group statistically significant ($p \leq .05$).
[e]Similarity to no-diagnosis group statistically significant ($p \leq .05$).
[f]Similarity to drug abuse group statistically significant ($p \leq .05$).

categorical data, with Yates's correction for continuity where appropriate and a Student's t test for comparison of means.

RESULTS

A total of 53% of the patients met the criteria for primary alcoholism, 14% demonstrated primary affective disorder, 14% met the diagnostic criteria for primary antisocial personality, 6% had serious drug problems prior to the onset of alcohol difficulties, and 13% had no established diagnosis. Although the demographic backgrounds of these groups (table 1) were similar, the drug abusers and patients with antisocial personality were the most unusual; they tended to be young, single, and, if they had been pregnant, to have lost a child or given birth to an offspring with handicaps. These women tended to come from lower SES origins as measured by Hollingshead's Two-Factor Index of Social Position (unpublished monograph, Yale University Department of Sociology, 1957) with a striking downward SES mobility.

The alcoholic women with affective disorder were demographically similar to the primary alcoholics but tended to be more isolated and to live in geographic areas other than skid row (as determined geographically [16]). The socioeconomic status of origin for these women was relatively high and their social status mobility patterns were nearly identical to those women with no diagnoses. The primary alcoholics tended to be middle-aged, Caucasian, and separated or divorced, coming from relatively low SES origins with slight upward mobility. Over one-third of these women lived alone, with the second highest proportion residing on skid row. The no-diagnosis group was demographically similar to the primary alcoholics but more likely to have never been married, less likely to live alone, and most likely to be working full- or part-time.

The antisocial and drug use histories for the five groups are reported in table 2. Once again, the most deviant were the patients with antisocial personality and the drug abusers, both demonstrating pervasive antisocial and drug problems, even in those areas not used to establish the original diagnoses. The primary alcoholics and alcoholics with affective disorder both demonstrated intermediate rates of school problems and difficulties with the parental family and the law,

TABLE 3
Drinking Patterns and Alcohol-Related Problems of 290 Women Seen in a County Detoxification Center

Variable[a,b]	Primary Diagnosis				
	Alcoholism (N=154)	Drug Abuse (N=18)	Affective Disorder (N=40)	Antisocial Personality (N=40)	No Diagnosis (N=38)
Drinking patterns					
Daily	37.0[f]	33.3[c,d]	35[c,d]	58.5[d]	10.3
In bars	51.3[c]	41.2[c]	47.5[c]	75.6	61.5
In parks	13[c]	29.4	12.5[c]	39[d]	10.3
In other public places	7.8	17.6[d,e]	0[c]	17.1[d]	0
Usually alone	40.9	47.1	57.5[d]	31.7	25.6
Binge (ever)	56.5[c,d,f]	83.3[d]	60[d]	80.5[d]	15.4
Uses alcohol substitutes	7.8[c]	16.7	10[c]	29.3[d]	2.6
Mean number of drinks per 24 hours	10.6[c,d]	12.9[c,d]	10.4[c,d]	19.3[d]	5.2
Mean age at first drink	16.9[c,d,f]	14.7[d,e]	16.9[c]	13.4[d]	19.2
Mean age at first intoxication	21.2[c,d]	19.4[c,d]	21.9[c]	14.8[d]	26.1
Mean age at first alcohol problem	34.1[c,f]	27.6[c,d,e]	34.6[c]	21.5[d]	38.9
Alcohol problems					
Shakes	90.9[d]	77.8	82.5[d]	92.7[d]	53.8
Blackouts	70.8[d]	88.9[d]	77.5[d]	82.9[d]	33.3
Missing work[g]	38.8[c,d]	50[d]	37.5[d]	58.5[d]	8.3
Fired[g]	24.7[c,d]	12.5	20[c,d]	45[d]	0
Fights	40.9[c,d]	55.6[d]	42.5[c,d]	73.2[d]	13.2
Auto accidents	31.0	35.7	32.4	24.2	19.4
Arrests					
Driving while intoxicated	32.8	21.4	32.4	24.2	19.4
Drinking in public	50.6[e]	38.9	25.0[c]	68.3[d]	33.3
Separated/divorced[h]	25.9[c,d]	7.7[c]	20.5[c,d]	45.5[d]	0
Withdrawal	60.4[d]	66.7[d]	52.5[d]	73.2[d]	0
Cirrhosis	22.1[d]	27.8[d]	22.5[d]	29.3[d]	0
Treatment history					
Tried to stop	79.9[d]	66.7	75.0[d]	70.7	48.7
Taken disulfiram	34.4[d]	27.8[d]	32.5[d]	26.8[d]	2.6
Outpatient	35.1[c,d]	50[d]	52.5[d]	56.1[d]	0
Inpatient	35.1[d]	38.9[d]	30[d]	46.3[d]	0

[a] In percents except when specified as mean.
[b] Statistical comparisons of percentage differences based on chi-square; statistical comparisons of differences between means based on Student's t test.
[c] Similarity to antisocial personality group statistically significant ($p \leq .05$).
[d] Similarity to no-diagnosis group statistically significant ($p \leq .05$).
[e] Similarity to affective disorder group statistically significant ($p \leq .05$).
[f] Similarity to drug abuse group statistically significant ($p \leq .05$).
[g] Of those ever employed.
[h] Of those ever married.

but the two groups differed on drug histories, with relatively low rates of drug abuse among the primary alcoholics and higher rates for patients with affective disorder. The no-diagnosis group had the best history of school performance and relatively low rates of school-related difficulties and problems with the parental family and drugs.

The drinking patterns and alcohol problem histories for the four groups are shown in table 3. The most serious and earliest-onset alcohol problems characterized the subjects with antisocial personality; women in this group were also the most likely to have seen a mental health worker or to have been hospitalized because of their drinking. The alcohol histories of the drug abusers were generally similar to those of the subjects with antisocial personality. The alcoholics with affective disorder, once again, were similar to the primary alcoholics in their drinking patterns, but the former were more likely to have seen a mental health worker. Although, almost by definition, the drinking patterns and problem histories of the no-diagnosis group were the most benign, they had been drinking enough for over half to have reported experiencing shakes and for many to have noted blackouts (forgetting what they had done while intoxicated but awake), auto accidents, or being arrested for alcohol-related problems. This last finding was biased by the fact that it was via auto accidents while drinking that many of these women were admitted to the detoxification center.

The psychiatric histories of the 290 subjects are given in table 4. Depressive symptoms—relatively persistent sadness that did not qualify for a diagnosis of affective disorder—were seen most frequently in the subjects with antisocial personality. The highest rates of psychiatric symptomatology and use of mental health services were for the subjects with affective disorder, but the women with antisocial personality and the drug abusers also made heavy use of such treatments and had the highest incidence of suicide attempts. Data not shown in the table indicate that the sociopaths were the only individuals to report transient psychoses, seen for 5%–10% as evanescent pictures of delusions, paranoia, or hallucinations that were not pervasive enough to warrant a diagnosis of schizo-

TABLE 4
Personal and Family Psychiatric Histories of 290 Women Seen in a County Detoxification Center

Variable[a,b]	Primary Diagnosis				
	Alcoholism (N=154)	Drug Abuse (N=18)	Affective Disorder (N=40)	Antisocial Personality (N=40)	No Diagnosis (N=38)
Personal history					
Depression	3.9[c,d]	5.6[c,d]	100[d,e]	34.1[e]	5.1
Outpatient treatment	24[c,d,f]	61.1[e]	80[d,e]	53.7[e]	23.1
Inpatient treatment	10.4[c,d]	16.7	37.5[e]	29.3[e]	7.7
Suicide attempts	28.6[c,d,f]	61.1[e]	55.0[e]	68.3[e]	23.1
Family history					
Alcoholism					
Father	22.7[d]	38.9[e]	22.5	43.9[e]	10.3
Mother	9.7[d]	11.1	5[d]	29.3[e]	0
Brother(s)	14.3	5.6	17.5	17.1	15.4
Sister(s)	9.7	0	15	7.3	12.8
Affective disorder					
Father	0[c]	0	5	0	0
Mother	1.9[d]	0	5	17.1	5.1
Brother(s)	0[c]	0	5	0	0
Sister(s)	4.5	5.6	7.5	2.4	2.6
Other psychiatric illness					
Father	0	0	0	0	0
Mother	0.6	0	0	2.4	0
Brother(s)	1.9[d]	11.1	7.5	14.6[e]	0
Sister(s)	2.6	5.6	5.0	2.4	2.6

[a]In percents.
[b]Statistical comparisons of percentage differences based on chi-square; statistical comparisons of differences between means based on Student's t test.
[c]Similarity to affective disorder group statistically significant ($p \leq .05$).
[d]Similarity to antisocial personality group statistically significant ($p \leq .05$).
[e]Similarity to no-diagnosis group statistically significant ($p \leq .05$).

phrenia (3). It is also of interest that the primary alcoholics had relatively high rates of both outpatient and inpatient treatment for what may have appeared to be non-alcohol-related psychiatric problems. These occurred after the onset of alcohol abuse and although not recognized as such by the mental health professional were probably related to ethanol.

The family history data are consistent with the patient diagnostic groups. The subjects with antisocial personality had the most disordered families and had high rates of alcoholism, affective disorder, and other psychiatric disorders, usually drug or antisocial in nature. The drug abusers also reported high rates of alcoholism in fathers, with brothers showing antisocial or drug problems. The alcoholics had less disordered families, but a predominant diagnosis of alcoholism, and the no-diagnosis group had the lowest rate of ill relatives.

DISCUSSION

The women admitted to the detoxification facility were heterogeneous with respect to primary diagnosis. More than half met the criteria for primary alcoholism—a rate similar to those reported in both public and private hospital admission samples of women (4, 7, 17) and men (5). As was true in the earlier studies, only a very small percentage (about 1%) met the fairly rigid diagnostic criteria for schizophrenia.

The distribution of women with secondary alcoholism differed from previous reports on inpatient facilities. The rate of affective disorder of 14% is midway between that expected for inpatient men (about 5%) and that for women (25%). The rate of antisocial personality falls almost midway between the previously reported 5%–9% for women and 20% reported for men (4, 5), and an additional 6% met the criteria for serious drug problems.

Slightly more than one in ten of the women met no diagnostic criteria for psychiatric disorders. These patients were in their mid 40s at the time of entering the program and appeared to be a relatively heterogeneous population. Examination of individual patient records revealed that one-third of the subgroup were almost certainly not alcoholic and found their way to the detoxification center for the first time because of a first drunk driving charge, public drunkenness arrest, or because they suffered an injury at a time when they were intoxicated. However, almost two-thirds of this group were relatively heavy drinkers who did have alcohol-related problems of a somewhat limited nature. Despite their age, these women might be in the relatively early stages of alcoholism or represent a less severe course of the disorder. Certainly, underreporting might be a problem with this sample, and future follow-ups will help determine the predictive validity of assigning these women to the no-diagnosis group.

The women diagnosed as having antisocial personality were distinctive, being the youngest, coming from

the lowest SES stratum, and showing marked downward mobility despite their young age. In part by definition, they were the most deviant as children, and their problems continued into adulthood, when they were likely to drink in bars and in public places, imbibe alcohol substitutes such as perfume, demonstrate an early age of onset of drinking and related problems, and show high rates of all types of alcohol-related social difficulties and the most severe drug histories. These women do not resemble the picture of a typical alcoholic reported in the literature (1, 2, 18).

Suicide attempts were much more frequent in the antisocial personality group, which, according to the literature, were probably spur-of-the-moment attempts involving periods of frustration rather than affective episodes (19, 20). With such chaotic backgrounds, it is not surprising that the antisocial women spent much time in treatment. Follow-ups of sociopaths (14) and individuals with both alcohol and antisocial problems (7) demonstrate a poor prognosis. Thus it is important to identify these women, as they are likely to have more social difficulties within the treatment center and may show more serious antisocial problems after they are discharged, and their potentially poor prognosis must be taken into consideration in evaluating the success of treatment efforts by the center.

The 6% of the sample with primary drug abuse and secondary alcoholism were midway between the antisocial and drug/alcohol problem histories of the women with antisocial personality and those with primary alcoholism. With these women it is important to remember the necessity of dealing with other drugs of abuse as part of the treatment regimen and of taking special care regarding the probably high propensity for future antisocial problems.

In prior studies (1, 7, 13) of middle-class samples alcoholic women with histories of affective episodes before the onset of alcohol problems have been shown to have relatively unique properties. In the present study the alcoholics with affective disorder more closely resembled the primary alcoholics than was true in prior investigations, probably reflecting the lower SES of this sample. However, there was still a trend for the affective disorder alcoholics to be younger, to come from higher socioeconomic strata of origin, to be more isolated than primary alcoholics, to be less likely to get into trouble from drinking in public places, and to be less likely to live in a skid row area. The affective disorder alcoholics were also more likely than the primary alcoholics to have attempted suicide, to have seen mental health workers for non-alcohol-related problems, and to have family histories of affective disease. The importance of the affective disorder subgroup in the present sample will be determined at follow-up, but the possibility of an affective disorder subtype running higher risks for suicide attempts and having greater needs for counseling unrelated to alcohol problems should be kept in mind by anyone dealing with alcoholic women.

The primary alcoholics in many ways resemble descriptions of the male public inebriates, with an average age in the mid 40s (21), the majority being Caucasian, and one-third to one-half being separated or divorced (22). For both men and women, public inebriates have about a one-third chance of living alone and only 20% hold a job. Likewise, both groups show relatively low rates of school achievement, although a mean grade of 10.8 in the present sample of primary alcoholics was higher than the seventh- to ninth-grade education reported for most male groups (21–23). The primary alcoholic women studied here were likely to drink daily, drink rather heavily (approximately 11 drinks per drinking day), drink in public places, and have relatively high rates of alcoholic problems. Thus, primary alcoholic women were more similar to lower-class alcoholic men than to the usual sample of middle-class women. The similarity is even greater when one considers that most samples of public inebriate men (10, 24) do not control for the presence of sociopaths.

One ancillary finding is worthy of comment. These women, especially those who have drug or antisocial histories, showed very high rates of miscarriage (25%–45%), abortion (15%–33%), and having handicapped children (10%–30%). It is impossible to determine whether lifestyle, genetic propensities, or alcohol/drug abuse was responsible for this, nor can we be sure how many of these handicapped children had the fetal alcohol syndrome (25). The high probability of such a syndrome in children of heavy drinking mothers needs to be borne in mind in any attempt at meeting the social needs of this group.

A variety of factors may help explain why these women came to a detoxification center. First, most had relatively low SES classes of origin, sliding into even lower positions as their alcohol problems progressed. Within this setting, they became relatively isolated, having high rates of divorce, separation, and living alone. Also, probably related to economics, their place of residence in the skid row area, along with their lack of resources, might have made them more likely to find themselves intoxicated in public.

To further understand this population we plan to gather data on a similar sample of men at detoxification and to carry out a future follow-up of both the samples of men and women. We hope that this study will stimulate others to look at women within this population and to recognize the probable importance of diagnostic subtypes for alcoholics in any treatment facility.

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Interpersonal Attractiveness and Clinical Decisions in Alcoholism Treatment

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The authors investigated the interactive contribution of patient characteristics and staff perceptions of the patient to decisions concerning the provision of continued alcoholism treatment following an initial 2-week evaluation period. Staff perceptions contributed significantly more to an individual's acceptance or rejection than did patient characteristics. A high interpersonal attractiveness rating also led to a significantly more positive evaluation of the patients' level of intelligence, knowledge of current events, morality, and adjustment. The present results and research findings with general psychiatric populations are compared.

DECISION-MAKING in the areas of clinical judgment, diagnosis, and the provision of treatment service, although of obvious importance, is still poorly understood (1). In any conceptualization of the clinical decision-making process, three main classes of variables must be considered: client or patient characteristics, characteristics of the health care provider, and the resulting interaction. While client and therapist variables have often been investigated independently, the study of the interaction between these two classes has been rare (1, 2).

The client variable that has received the most intensive investigation has been social class, typically defined in terms of occupational status, income, and/or educational achievement. In reviewing a large body of early literature on client characteristics, Garfield (2) concluded that social class variables were more predictive of both acceptance into and duration of psychotherapy than were personality variables. The results of early epidemiological studies (3, 4) and more recent analogue investigations (1) show a relatively consistent pattern: upper- and middle-class clients are more likely to be judged favorably, are more likely to be accepted into treatment, and are likely to receive more extensive care. The effects of social class may be mediated by the evocation of certain stereotypic biases among treatment personnel (1, 5). Stern (5), summarizing the relevant literature, indicated that staff members in many psychiatric facilities believe that persons of high social class possess attitudes and abilities which allow them to benefit maximally from individual psychotherapy. For instance, Brill and Storrow (6) found that lower social class status was significantly related to therapist evaluations of low estimated intelligence, a tendency for clients to view their problems as physical rather than emotional, a desire for symptomatic relief, and a lack of understanding of and desire for psychotherapy. The intake interviewer had less positive feeling toward lower-class patients and saw them as less treatable by means of psychotherapy than middle-class patients.

Strupp and his coworkers (7, 8) evaluated staff members' interpersonal attraction toward clients and found that the interviewer's liking for the patient as a person was positively related to judgments concerning the clients' emotional maturity, motivation for treatment, expected level of improvement in therapy, and recommendations for insight-oriented therapy. Furthermore, liking the client was inversely related to ratings of hostility, defensiveness, and the degree of disturbance. Such findings, taken together, suggest that variables such as client social status and a therapist's perception of a client's interpersonal attractiveness may operate interactively to influence decision making in diagnosis, acceptability for treatment, and the nature of treatment.

Blane (9) has indicated that client characteristics generally bear the same association to treatment for alcoholics that holds for general psychiatric patients. Smart and associates (10) noted that the patient's social class was chief among factors extraneous to alcoholic clients' personality and disease process, which influenced the type of service provided. Socioeconomic status and social stability also appeared to be positively related to treatment outcome (11–16).

In contrast to the relatively well-documented effects of client characteristics on the selection for and out-

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come of alcoholism treatment, relatively little information is available concerning the effects of therapist characteristics or therapist-client interactions. Therapist variables are potentially crucial to the successful treatment of the alcoholic. They include such background characteristics as age, sex, and therapeutic experience, as well as psychological factors such as receptivity to treating alcoholics and interpersonal attraction (9). Furthermore, it has been suggested (9, 17, 18) that the interaction between client and therapist variables may have its maximum impact during the initial stages of treatment and may contribute to premature treatment termination. However, there is little empirical support for these contentions.

The purpose of the present study was to investigate the relative contribution of patient and treatment staff variables to clinical decisions to offer continued inpatient therapy to alcoholics following a two-week evaluation period. The patient variables included measures of social class and demographic characteristics, personality functioning, and the client's perception of the treatment ward atmosphere. The staff variable under consideration was the treatment personnel's attitude toward the clients, measured by ratings of their perceived interpersonal attractiveness. Based on the pattern of results found with general psychiatric populations, we anticipated that the major contributors to the decision concerning continued treatment would be the patients' social status and the staff members' ratings of the clients' interpersonal attractiveness; client personality variables were expected to have less influence on the decision process.

METHOD

Subjects

The subjects were 50 consecutive admissions to the inpatient phase of an alcoholism treatment program. Prior to admission the subjects, all males, were screened for gross evidence of severe thought disorder, psychotic symptoms, and organic brain syndrome. The primary diagnosis for each patient was alcohol addiction. The sample had a mean age of 45.08 years (SD=10.57) and a mean education level of 12.60 years (SD=2.66). All subjects provided informed consent to participate in the research after the procedures of the study were fully explained.

Treatment Setting

The research was conducted in the alcoholism treatment program of the Seattle Veterans Administration Medical Center. The primary treatment staff consisted of a multidisciplinary team composed of a psychiatrist, a clinical psychologist, social workers, a vocational rehabilitation specialist, alcoholism counselors, and nursing personnel. The therapeutic regimen consisted largely of interpersonal skills training and small-group therapy with a process orientation (19), both within the broader framework of a therapeutic community. Psychological theories of alcoholism development and maintenance were stressed; patients were helped to focus on the contingencies contributing to their drinking behavior and were encouraged to take responsibility for making life changes necessary to increase the probability of their achieving abstinence and overall social and personal adjustment. Following admission, and detoxification if necessary, patients completed a 2-week evaluation period. At the end of this period the staff consensually decided whether or not to offer the patient continued treatment, i.e., 3 more weeks of inpatient treatment followed by 1 month of day treatment and 9 months in a weekly aftercare group.

Patient Measures

The clients' age, educational level, and social status were employed as primary demographic variables. Hollingshead and Redlich's procedure (3) was used to generate a two-factor index of social position (the sum of the weighted scaled scores for occupational and educational levels), which was then converted into one of five levels of social class. Higher scores on each of these variables is related to a lower social status.

A variety of personality and cognitive tests was completed by the patients approximately 10 days following admission. The Minnesota Multiphasic Personality Inventory (MMPI) provided a measure of generalized psychopathology. Schutz's FIRO-B test (20) was given to assess the clients' orientation toward interpersonal relationships. The scale measures three basic dimensions of interpersonal relationships: inclusion, control, and affection (21). Inclusion assesses the degree to which a person associates with others. Control measures the extent to which the individual assumes responsibility, makes decisions, and dominates people. Affection assesses the degree to which a person becomes emotionally involved with others. Each of these three dimensions is further differentiated into two subscores reflecting expressed or manifest behavior versus wanted or desired behavior.

Rotter's internal-external locus of control scale (22) was employed as a measure of the general level of personal responsibility the individual assumes for his/her behavior. A low score on the scale reflects the individual's perception that the outcome of significant life events is contingent on personal initiative and action. A specially constructed drinking-related locus of control scale was also included (23, 24). An external score on the scale has been found to be related to increased levels of physical, psychological, and social impairment due to chronic drinking (24).

Intellectual functioning was assessed by the Shipley-Hartford Scale (25) and the Trail Making Test (26). Measures of verbal ability, abstracting ability, cognitive impairment (Conceptual Quotient), and general intelligence (IQ) were derived from the Shipley-Hartford Scale. Higher scores on each of these measures indicate a higher level of cognitive functioning. Higher scores on the two portions of the Trail Making Test

(parts A and B) indicate higher levels of cognitive impairment.

On the day the staff members were to make their decision concerning continued treatment, the patient completed the Ward Atmosphere Scale (WAS), Form R (27, 28), which assesses the patient's perception of the social climate of the treatment unit. The scale measures 10 dimensions of the ward atmosphere falling within 3 broad categories. The first, including the involvement, support, and spontaneity subscales, measures aspects of patient and staff relationships. The next four subscales, autonomy, practical orientation, personal problem orientation, and anger and aggression, measure personal development and the program's treatment orientation. The final scales, order and organization, program clarity, and staff control, assess system maintenance dimensions.

Staff Measures

Prior to discussing a particular patient's clinical status and reaching a consensual decision concerning his suitability for continued treatment, each member of the primary treatment team rated the patient on Byrne's Interpersonal Judgment Scale (IJS) (29). The IJS consists of 6 subscales (intelligence, knowledge of current events, morality, adjustment, personal feelings of likability, and working together in a group). Staff members were to express their opinions about and evaluations of the patient by rating on each of the 7-point subscales. The first 4 items deal with various aspects of personal evaluation. The last 2 items deal specifically with interpersonal attractiveness.

Following the completion of the IJS scales, the staff members discussed the patient's case and arrived at a decision concerning continued treatment. The outcome of this staff decision was used to classify the subjects as accepted or rejected for the purposes of the present analysis. Of the 50 subjects, 22 (44%) were rejected and 28 (56%) were accepted.

RESULTS

The 6 primary staff members' ratings for each patient were averaged to provide an overall evaluation of the individual on each of the 6 IJS subscales. A patient's total interpersonal attraction score was then derived by averaging the sum of the staff's scores on the personal feelings of likability and working together in a group subscales. These IJS subscale scores, as well as all of the other patient personality and demographic variables, were evaluated by means of one-way analyses of variance for the accepted and rejected patients.

The results of this analysis indicate that the patient variables related minimally to the decision concerning treatment disposition. Of the 40 patient variables, only 4 significantly differentiated between the accepted and rejected groups. Those patients who were selected for continued treatment had a significantly higher educational level ($p<.05$), a greater need for others to initiate close, intimate relationships with them (FIRO-B, Want Affection scale) ($p<.01$), a higher level of ego strength ($p<.05$), and a higher level of functional defensiveness (MMPI K scale) ($p<.05$). No differences were found between groups on age, social class, cognitive functioning, general or drinking-specific locus of control, perception of ward atmosphere, or other measures of interpersonal and psychological function.

TABLE 1
Accept and Reject Rates Based on Staff Ratings on Interpersonal Judgment Scale

IJS Variables	Low Level (%)		High Level (%)			
	Accept	Reject	Accept	Reject	χ^{2a}	ϕ
Intelligence	22.0	30.0	34.0	14.0	3.05	.29
Current events	18.0	32.0	38.0	12.0	6.57[b]	.40
Morality	20.0	28.0	36.0	16.0	2.81	.28
Adjustment	20.0	28.0	36.0	16.0	2.81	.28
Likability	12.0	36.0	44.0	8.0	15.66[c]	.60
Group membership	10.0	38.0	46.0	6.0	20.50[c]	.68

[a]Yates' correction applied.
[b]$p<.01$.
[c]$p<.0001$.

In contrast to the relative noncontributory nature of these patient variables, the staff ratings on the Interpersonal Judgment Scale significantly differentiated between the two groups. Subjects who were accepted for treatment were rated as being more intelligent ($p<.05$), having a greater knowledge of current events ($p<.05$), having higher moral standards ($p<.01$), and being better adjusted ($p<.01$). Furthermore, accepted patients were seen as being more interpersonally attractive by the staff, as indicated by the significantly higher ratings of personal likability and suitability for group membership ($p<.01$).

The relationship of the individual IJS variables to the acceptance or rejection of patients was further evaluated by 2×2 chi-square analyses. Subjects were classified as falling above (high level) or below (low level) the median on each of the IJS subscales. The percentage of subjects within each of these categories who were accepted or rejected is found in table 1. Of the IJS subscales, the perceived level of knowledge of current events, the staff's liking of the patient as a person, and the judged suitability for group membership were the most predictive of the staff decision process.

A stepwise multivariate discriminant functions analysis (30) was conducted to determine that combination of variables which provided the greatest level of differentiation between the accepted and rejected patient groups. The 9 most discriminating original variables were entered into this analysis based on the results of the initial analysis of variance. These variables were the patient's educational level, MMPI K and ego strength scales, the FIRO-B Want Affection score, the 4 evaluative subscales of the IJS, and the total interpersonal attractiveness score. The patient's social position index was also included in order to determine the multivariate contribution of this variable. The results indicated that 3 of the 10 variables made signifi-

cant contributions to this discrimination: total interpersonal attractiveness ($p<.001$), wanted affection ($p<.002$), and ego strength ($p<.02$).

DISCUSSION

Garfield (2) noted that not all individuals who apply for psychotherapeutic help receive it, and not all individuals who are offered psychotherapy accept it. Previous research in the area of clinical decision making has found that a major determinant of the outcome of such decisions is the patient's social status (1–4, 6). It had been assumed that lower-class clients had values, attitudes, and conceptions concerning treatment that differed from those of middle-class clients, as well as therapists, which interfered with their active participation in the therapeutic process. More recent findings have challenged these assumptions. Frank and associates (31) found that the conceptions clients have concerning therapy is highly similar across social classes. Stern (5) also found that client social status was not related to acceptance into treatment, assignment to different forms of treatment, or to the number of therapeutic sessions provided. It has been suggested (5, 31) that this discrepancy between the older body of literature, collected primarily in the 1950s and 1960s, and more recent findings may be related to relatively widespread societal changes in attitudes toward mental illness and the increased availability of professional services to all levels of social class.

Our results with alcoholics in an inpatient rehabilitation program also tend to be discrepant from earlier findings. Prior research indicated that the social class of alcoholic subjects exerted a great degree of influence on acceptance into treatment, type of therapy assignment, and outcome (9-16). However, the present findings do not support the pervasiveness of social class influence. We found that alcoholics' social position did not contribute significantly to staff decisions concerning the provision of continued treatment. A possible interpretation for this finding is that patients within the Veterans Administration system are drawn from a relatively low and restricted range of social class; however, an examination of the two-factor social position index indicates that while the mean for the present sample fell within class IV (a mean of 52.5 out of a possible range from 11 to 77), the sample was also characterized by a high degree of variability ($SD=12.3$).

It may be that with an increased emphasis being placed on the provision of therapeutic services to a broadly based constituency, both within the VA and community mental health systems, the potentially biasing influence of social class is diminishing. If social class played a minimal role in clinical decisions concerning the provision of treatment, what other factors may have affected this decision? Patient's personality characteristics appeared to play a limited role. Of the 40 demographic, personality, and cognitive variables investigated, only 4 were found to differentiate between those alcoholics accepted or rejected for continued therapy. Those individuals accepted for treatment were better educated, desired others to initate close, intimate relationships with them, presented themselves in a more positive light, and had a higher level of ego strength; this latter scale has been shown in previous research to be predictive of prognosis in psychotherapy (32).

The variable found to be most predictive of staff members' decisions was their perception of the patient's interpersonal attractiveness. Those individuals who were judged to be highly attractive on an interpersonal level, as defined by staff ratings of their personal likability and suitability for group membership, were highly likely to be accepted for continued treatment. This finding is consistent with the suggestion of Smart and associates (10) that a number of factors extraneous to the alcoholic patient's personality characteristics and disease state may influence treatment-related decisions. The primary variable appears to be the staff members' collective attitude toward the patient rather than psychometrically measured patient characteristics.

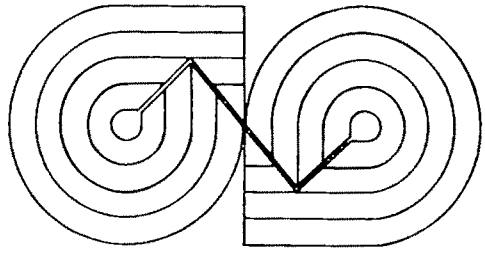
The present findings may be reflective not so much of social class as of the professional relationship between staff and patient. One might speculate that therapists rate as likable those types of patients who, in their experience, are most likely to benefit from the therapeutic modalities at which they are most skilled, thus reinforcing the therapists' sense of professional effectiveness. Thus, ratings of likability may have objective behavioral correlates that are at least partially independent of social class biases.

We have attempted to determine how alcoholics' personal characteristics and staff members' perceptions interact to influence aspects of treatment at the entry boundary of a therapeutic system. Continued research is needed in this area (9, 17, 18) as well as in attempts to determine the effects of such interactions on assignment to differing treatment modalities and on therapeutic outcome. If these findings are replicated in other mental health settings as a stable feature of clinical decision making, they have important implications for training and education programs in psychiatry and the behavioral sciences.

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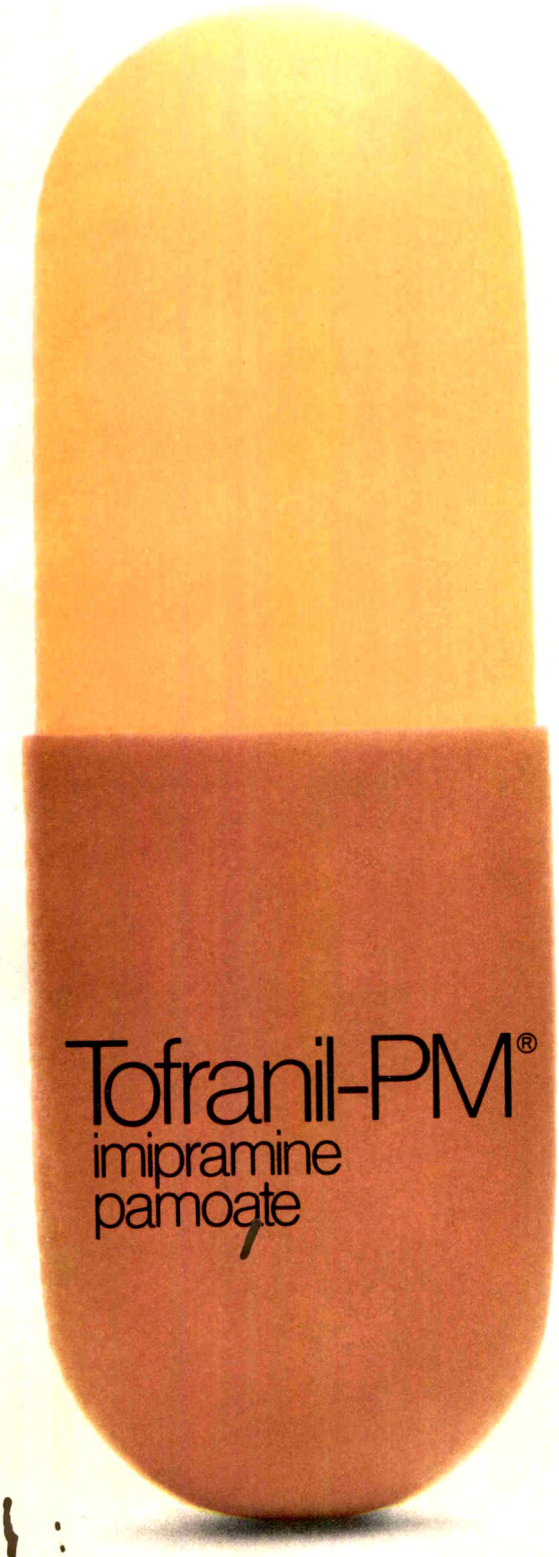
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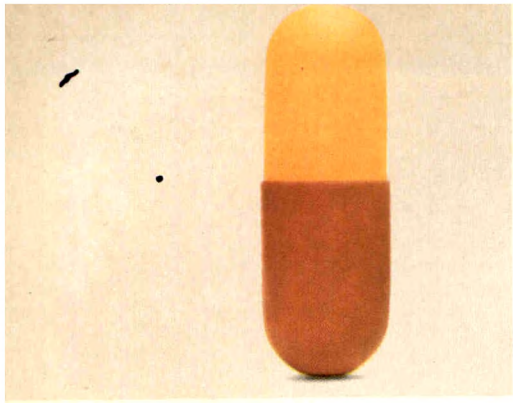
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Tofranil-PM may enhance the CNS depressant effects of alcohol. Therefore, it should be borne in mind that the dangers inherent in a suicide attempt or accidental overdosage with the drug may be increased for the patient who uses excessive amounts of alcohol. (See **Precautions**.)

Usage in Children: Tofranil-PM should not be used in children of any age because of the increased potential for acute overdosage due to the high unit potency (75 mg, 100 mg, 125 mg and 150 mg). Each capsule contains imipramine pamoate equivalent to 75 mg, 100 mg, 125 mg or 150 mg imipramine hydrochloride.

Precautions An ECG recording should be taken prior to the initiation of larger-than-usual doses of imipramine pamoate and at appropriate intervals thereafter until steady state is achieved. (Patients with any evidence of cardiovascular disease require cardiac surveillance at all dosage levels of the drug. See **Warnings**.) Elderly patients and patients with cardiac disease or a prior history of cardiac disease are at special risk of developing the cardiac abnormalities associated with the use of imipramine pamoate. It should be kept in mind that the possibility of suicide in seriously depressed patients is inherent in the illness and may persist until significant remission occurs. Such patients should be carefully supervised during the early phase of treatment with imipramine pamoate and may require hospitalization. Prescriptions should be written for the smallest amount feasible.

Hypomanic or manic episodes may occur, particularly in patients with cyclic disorders. Such reactions may necessitate discontinuation of the drug. If needed, imipramine pamoate may be resumed in lower dosage when these episodes are relieved. Administration of a tranquilizer may be useful in controlling such episodes. An activation of the psychosis may occasionally be observed in schizophrenic patients and may require reduction of dosage and the addition of a phenothiazine. Concurrent administration of imipramine pamoate with electroshock therapy may increase the hazards; such treatment should be limited to those patients for whom it is essential, since there is limited clinical experience.

Usage During Pregnancy and Lactation:
Animal reproduction studies have yielded inconclusive results.

There have been no well-controlled studies conducted with pregnant women to determine the effect of imipramine on the fetus. However, there have been clinical reports of congenital malformations associated with the use of the drug. Although a causal relationship [illegible] these effects and the drug could not be establis[illegible] possibility of fetal risk from the maternal ingestio[illegible] imipramine cannot be excluded. Therefore, imip[illegible] should be used in women who are or might bec[illegible] pregnant only if the clinical condition clearly just[illegible] potential risk to the fetus.

Limited data suggest that imipramine is likely to [illegible] creted in human breast milk. As a general rule, [illegible] woman taking a drug should not nurse since the [illegible] possibility exists that the drug may be excreted [illegible] breast milk and be harmful to the child.

Patients should be warned that imipramine pam[illegible] may enhance the CNS depressant effects of alc[illegible] (See **Warnings.**)

Imipramine pamoate should be used with cautio[illegible] patients with significantly impaired renal or hep[illegible] function.

Patients who develop a fever and a sore throat [illegible] therapy with imipramine pamoate should have [illegible] cyte and differential blood counts performed. In[illegible] ramine pamoate should be discontinued if there[illegible] dence of pathological neutrophil depression.

Prior to elective surgery, imipramine pamoate sh[illegible] be discontinued for as long as the clinical situa[illegible] allow.

In occasional susceptible patients or in those re[illegible] anticholinergic drugs (including antiparkinsoni[illegible] agents) in addition, the atropine-like effects ma[illegible] come more pronounced (e.g., paralytic ileus). [illegible] supervision and careful adjustment of dosage [illegible] quired when imipramine pamoate is administer[illegible] comitantly with anticholinergic drugs.

Avoid the use of preparations, such as decong[illegible] and local anesthetics, which contain any sympa[illegible] mimetic amine (e.g., epinephrine, norepinephr[illegible] since it has been reported that tricyclic antidep[illegible] can potentiate the effects of catecholamines.

Caution should be exercised when imipramine [illegible] oate is used with agents that lower blood press[illegible] Imipramine pamoate may potentiate the effects [illegible] depressant drugs.

Patients taking imipramine pamoate should av[illegible] cessive exposure to sunlight since there have b[illegible] ports of photosensitization.

Both elevation and lowering of blood sugar lev[illegible] been reported with imipramine pamoate use.

Adverse Reactions Note: Although the listing [illegible] follows includes a few adverse reactions which [illegible] not been reported with this specific drug, the p[illegible] macological similarities among the tricyclic ant[illegible] sant drugs require that each of the reactions be[illegible] ered when imipramine is administered.

Cardiovascular: Orthostatic hypotension, hype[illegible] tachycardia, palpitation, myocardial infarction, [illegible] mias, heart block, ECG changes, precipitation [illegible] gestive heart failure, stroke.

Psychiatric: Confusional states (especially in t[illegible] derly) with hallucinations, disorientation, delusi[illegible] iety, restlessness, agitation; insomnia and nigh[illegible] hypomania; exacerbation of psychosis.

Neurological: Numbness, tingling, paresthesia[illegible] tremities; incoordination, ataxia, tremors; perip[illegible] neuropathy; extrapyramidal symptoms; seizure[illegible] tions in EEG patterns; tinnitus.

Anticholinergic: Dry mouth, and, rarely, associ[illegible] lingual adenitis; blurred vision, disturbances o[illegible] commodation, mydriasis; constipation, paralyt[illegible] urinary retention, delayed micturition, dilation [illegible] nary tract.

Allergic: Skin rash, petechiae, urticaria, itching[illegible] sensitization; edema (general or of face and t[illegible] drug fever; cross-sensitivity with desipramine.

Hematologic: Bone marrow depression includi[illegible] agranulocytosis; eosinophilia; purpura; thromb[illegible] penia.

Gastrointestinal: Nausea and vomiting, anorex[illegible] epigastric distress, diarrhea; peculiar taste, st[illegible] abdominal cramps, black tongue.

Endocrine: Gynecomastia in the male; breast [illegible] ment and galactorrhea in the female; increase[illegible] creased libido, impotence; testicular swelling; [illegible] or depression of blood sugar levels.

Other: Jaundice (simulating obstructive); alte[illegible] function; weight gain or loss; perspiration; flus[illegible] nary frequency; drowsiness, dizziness, weakn[illegible] fatigue; headache; parotid swelling; alopecia; [illegible] ness to falling.

Withdrawal Symptoms: Though not indicative [illegible] tion, abrupt cessation of treatment after prolon[illegible] therapy may produce nausea, headache and [illegible]

How Supplied Tofranil-PM is available as follo[illegible] 75-mg, coral-colored capsules equivalent to [illegible] imipramine hydrochloride in bottles of 30, 100[illegible] 1,000 capsules, and unit strip packages of 10[illegible] 100-mg, dark yellow/coral-colored capsules e[illegible] to 100 mg imipramine hydrochloride in bottles [illegible] and 1,000 capsules.

125-mg, light yellow/coral-colored capsules e[illegible] to 125 mg imipramine hydrochloride in bottles [illegible] and 1,000 capsules.

150-mg, coral-colored capsules equivalent to [illegible] imipramine hydrochloride in bottles of 30, 100[illegible] capsules, and unit strip packages of 100.

For complete details, including description, a[illegible] dosage and administration, and overdosage, see full prescribing information.

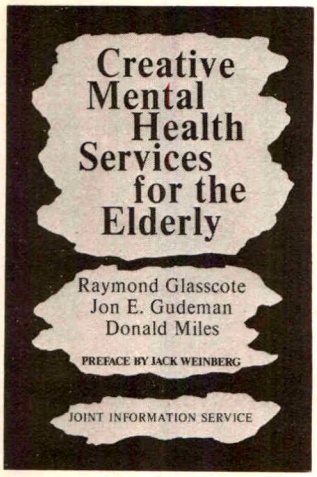
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Geigy

The Joint Information Service of the American Psychiatric Association and the Mental Health Association releases Two New Volumes That

Focus on the Elderly



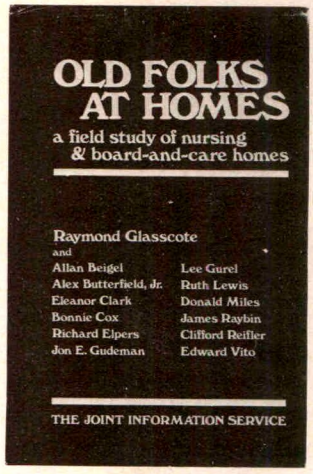
This carefully documented book reports a field study of ten programs, four in America and six in England and Scandinavia, which serve as practical demonstrations of creative mental health services for the elderly. They include:

- a novel "Neighborhood Family" providing vigorous support to elderly residents of several trailer parks
- a high school for the elderly which sends its students abroad for study trips
- a "Human Development Project" that focuses on responding to the psychological needs of the elderly
- a carefully coordinated system of "respite hospitalization," which promotes the health of the elderly while allowing maximum use of hospital beds and family resources
- a "Lucy Booth," patterned after the Peanuts comic strip, which provides easy access to many kinds of services
- and many other innovative, successful approaches.

Each program is subjected to intensive scrutiny. The resulting document becomes a handbook, and, as well, a forum through which some of the world's most experienced practitioners of "the psychiatry of old age" present their views.

Dr. Wilma Donohue, director of the International Center for Social Gerontology, characterizes this volume as "a masterful job of putting everything in relief . . . the guidelines for action are right here."

190 pages. Casebound. Price $8.50.



Concerned about the very bad reputation that nursing homes have received from many quarters in recent years, the Joint Information Service set up a field study to visit a systematically chosen sample of nursing homes, and board-and-care homes as well, to see to what extent the care of and the quality of life for the patients differed from the geriatrics units of state hospitals. Altogether, sixty nursing homes and 31 board-and-care homes in ten locales were visited, in each case by the senior author accompanied by two mental health professionals.

The teams were surpised to learn that care and quality of life in many of the nursing and board-and-care homes they visited were better—sometimes dramatically better—than that in the mental health facilities and often at lower cost.

This unique publication provides an overview of all facilities visited plus vignettes of approximately half. Federal and state rules and regulations are also expertly synthesized into brief and readable style.

148 pages. Casebound. Price $6.50

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1700 18th St., N.W., Washington, D.C. 20009

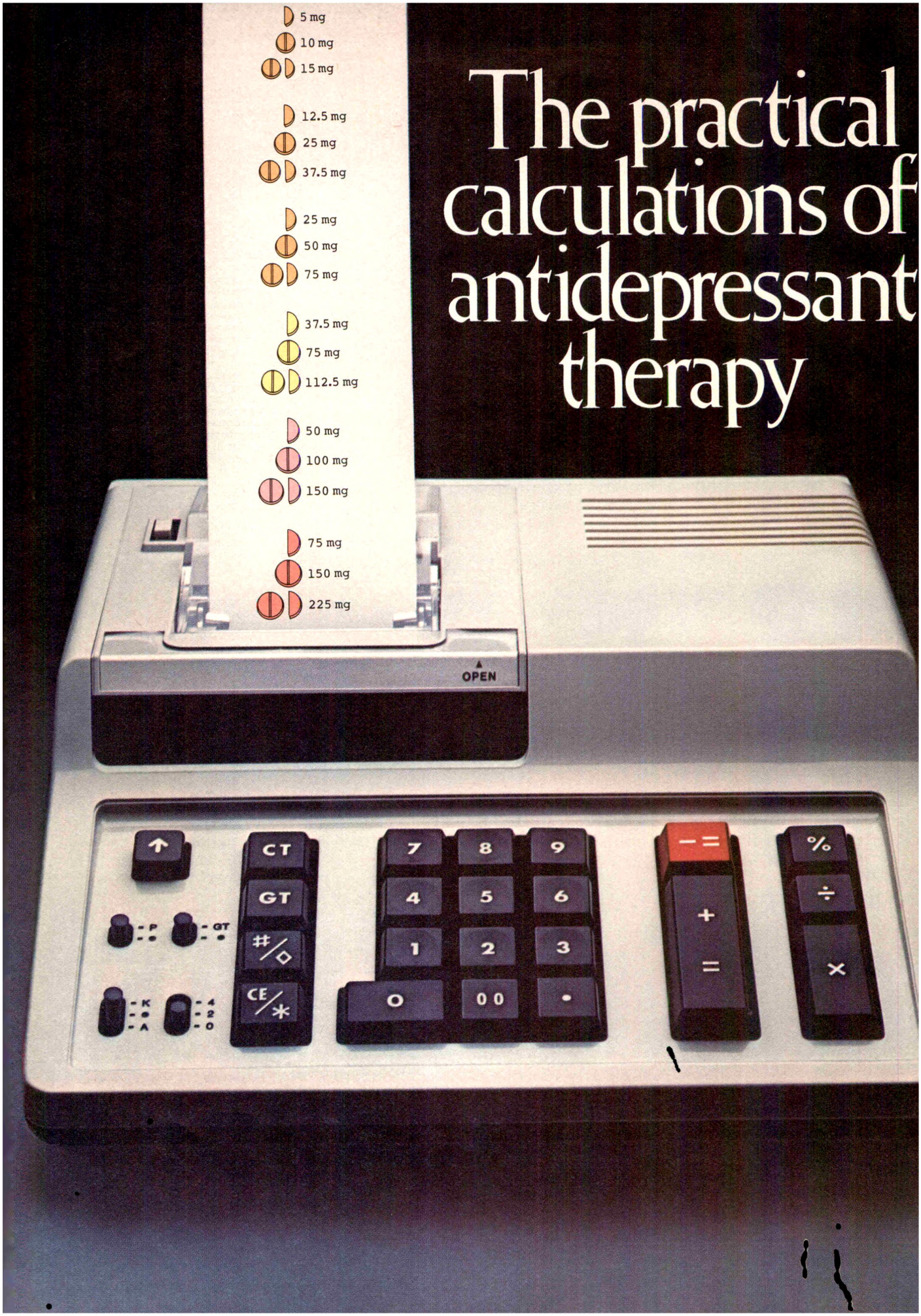
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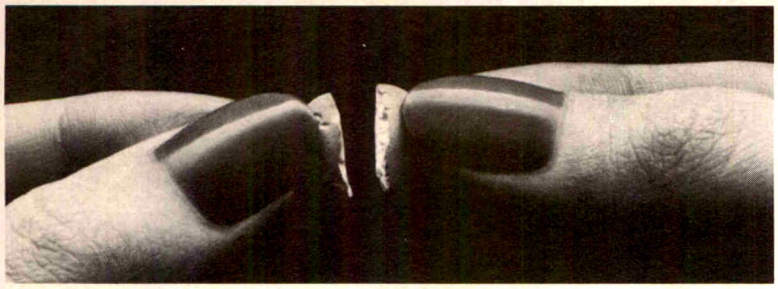
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12.5 mg
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The practical calculations of antidepressant therapy
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Divide scored Endep tablets



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Add flexibility to dosage titration

Long term treatment of depression with amitriptyline requires precise calculation of dosage to meet the individual needs of each patient. Because all six strengths of Endep are scored, you can easily increase or decrease dosage in half-tablet steps, without the bother and expense of a new prescription.

−

Subtract waste and cost

Scored Endep tablets permit you to adjust the regimen without wasting unused tablets or burdening the patient with the cost of a new prescription.

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Multiply therapeutic options

The pharmacokinetic properties of Endep are such that its clinical effect is the same whether prescribed *t.i.d.* or as a single daily dose. Thus, once you have established the proper dosage, you can prescribe the entire dose *h.s.*, minimizing anticholinergic and sedative side effects, and helping to assure patient compliance with this simple regimen.

By prescribing the higher strength tablets *h.s.*— secure in the knowledge that the dosage can be adjusted without resorting to a new prescription—your patient will economize further on a per milligram basis.

Endep®
amitriptyline HCl/Roche

The only antidepressant scored for easy division

Endep
amitriptyline HCl/Roche

Before prescribing, please consult complete product information, a summary of which follows:

Contraindications: Known hypersensitivity. Do not use with monoamine oxidase (MAO) inhibitors or within at least 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use; then initiate cautiously, gradually increasing dosage until optimal response is achieved. Use not recommended during acute recovery phase after myocardial infarction.

Warnings: May block action of guanethidine or similar antihypertensives. Use with caution in patients with history of seizures, urinary retention, angle-closure glaucoma, increased intraocular pressure. Closely supervise cardiovascular patients, hyperthyroid patients and those receiving thyroid medications. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, including amitriptyline HCl, especially in high doses. Myocardial infarction and stroke reported with use of this class of drugs.) May impair alertness; warn against hazardous occupations or driving a motor vehicle during therapy. Weigh possible benefits against hazards during pregnancy, the nursing period and in women of childbearing potential. Not recommended in children under 12.

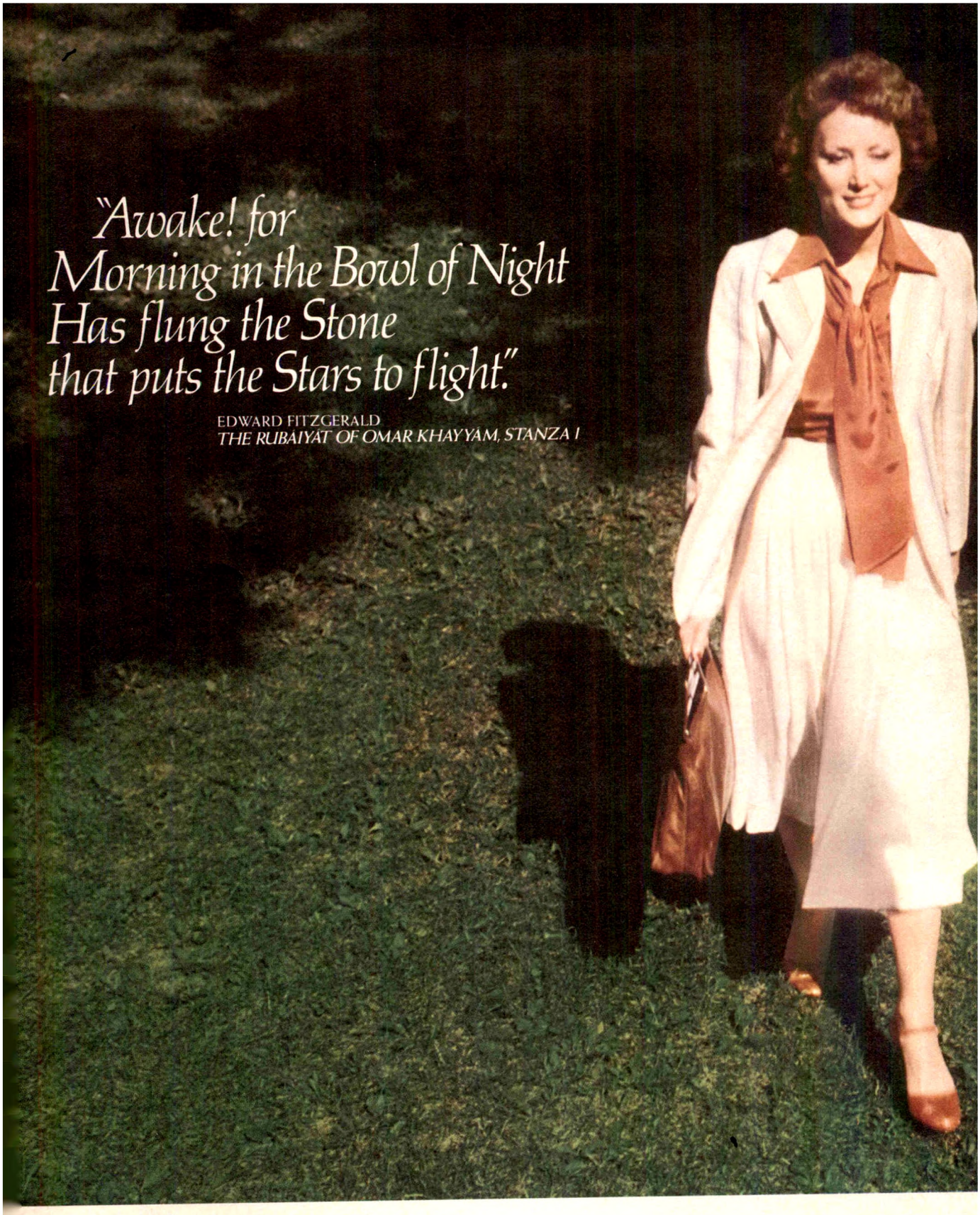
Precautions: May exaggerate symptoms in schizophrenic and paranoid patients, or shift manic-depressives to manic stage; reduce dose or administer major tranquilizer concomitantly. Close supervision and careful dose adjustments required when given with anticholinergic or sympathomimetic agents. Exercise care in patients receiving large doses of ethchlorvynol; transient delirium reported with concomitant administration. May enhance effects of alcohol, barbiturates and other CNS depressants. Because of the possibility of suicide in depressed patients, do not permit easy access to large drug quantities in these patients. Because it may increase hazards of electroshock therapy, limit concomitant use to essential treatment. If possible, discontinue drug several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported.

Adverse Reactions: Note: This list includes a few adverse reactions not reported with this specific drug but requiring consideration because of similarities of tricyclic antidepressants. *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, constipation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, black tongue. *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, jaundice, alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache and malaise. These are not indicative of addiction.

Supplied: *Scored Tablets:* 10, 25, 50, 75, 100, 150 mg.

ROCHE

Roche Laboratories
Division of Hoffmann-La Roche Inc.
Nutley, New Jersey 07110



Brief Summary of Prescribing Information

ADAPIN® (doxepin HCl) Capsules

Indications—Relief of symptoms of anxiety and depression.

Contraindications—Glaucoma, tendency toward urinary retention, or hypersensitivity to doxepin.

Warnings—Adapin has not been evaluated for safety in pregnancy. No evidence of harm to the animal fetus has been shown in reproductive studies. There are no data concerning secretion in human milk, nor on effect in nursing infants.

Usage in children under 12 years of age is not recommended. MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with this drug, as serious side-effects and death have been reported with the concomitant use of certain drugs and MAO inhibitors.

In patients who may use alcohol excessively potentiation may increase the danger inherent in any suicide attempt or overdosage.

Precautions—Drowsiness may occur and patients should be cautioned against driving a motor vehicle or operating hazardous machinery. Since suicide is an inherent risk in depressed patients they should be closely supervised while receiving treatment. Although Adapin has shown effective tranquilizing activity, the possibility of activating or unmasking latent psychotic symptoms should be kept in mind.

Adverse Reactions—Dry mouth, blurred vision and constipation have been reported. Drowsiness has also been observed.

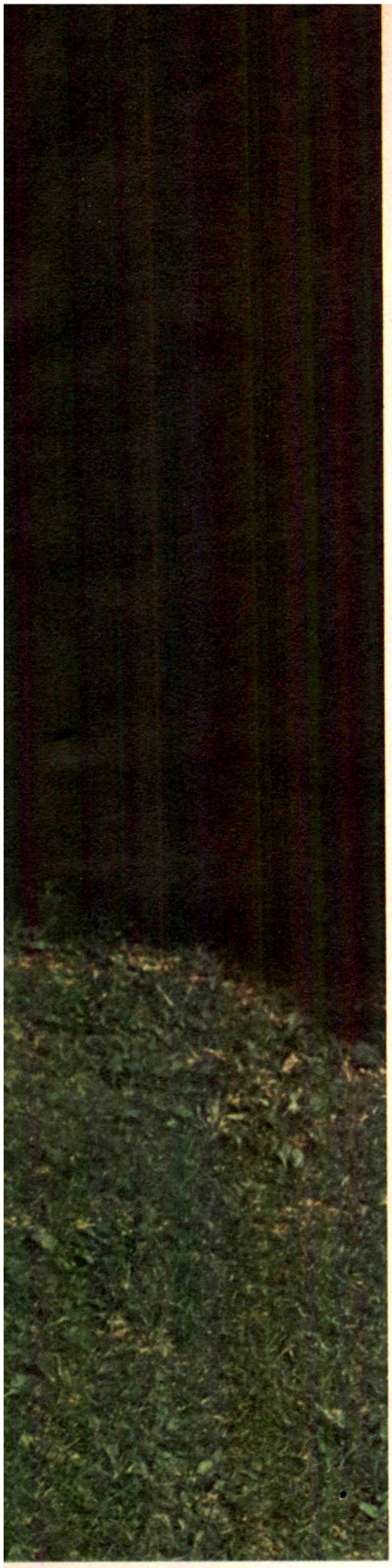
Adverse effects occurring infrequently include extrapyramidal symptoms, gastrointestinal reactions, secretory effects such as sweating, tachycardia and hypotension. Weakness, dizziness, fatigue, weight gain, edema, paresthesias, flushing, chills, tinnitus, photophobia, decreased libido, rash and pruritus may a occur.

Dosage and Administration—In mild to moderate anxiety an depression: 25 mg t.i.d. Increase or decrease the dosage accor to individual response. Daily dosage, up to 150 mg may be take bedtime without loss of effectiveness. Usual optimum daily dos is 75 mg to 150 mg per day not to exceed 300 mg per day.

Antianxiety effect usually precedes the antidepressant effec two or three weeks.

How Supplied—Each capsule conta s doxepin, as the drochloride: **10 mg, 25 mg, 50 mg, and 00 mg** apsules in bo of 100 and 1000.

For complete prescribing information please e package in or PDR.



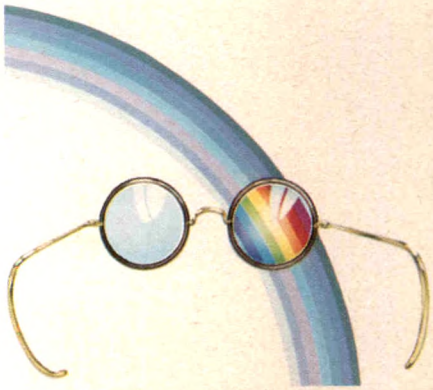
in anxiety/depression...

a good awakening from a good night's sleep

Most depressed patients experience some form of sleep disturbance. Adapin® helps relieve the accompanying anxiety, and restores a more normal sleep pattern right from the start of therapy.

☐ **convenient, flexible therapy**
The usual optimal dose is 75 to 150 mg daily. The entire dose may be given at night without loss of therapeutic effect. Adapin is available as 10 mg, 25 mg, 50 mg, and 100 mg capsules.

Adapin®
(doxepin HCl)
useful adjunctive therapy in your psychiatric practice



When they see life in shades of blue... help them see life in all its colors

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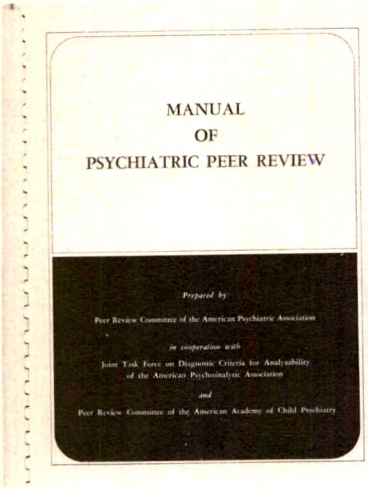
You know what to expect from antidepressant therapy;



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"This Manual provides a set of guidelines for district branches of APA and their peer review committees which should be adapted and modified in terms of local criteria, standards, and practices. The guidelines are not intended to be hard and fast rules. They are screening criteria, and not regulations to control practice. They are so designed that they can be utilized by non-physicians to identify those cases that can profitably be reviewed by psychiatrists serving on peer review committees. This document should be viewed as transitional, subject to revision as experience is gained, and continually updated in response to advances in psychiatric knowledge and changes in patterns of practice. . . . The application of these standards, and refinement through experience must rest with the peer review committees of district branches. . . . It should help them to move ahead in the development of a peer review for American psychiatry."

Robert W. Gibson, M.D.
President

Part I titled PSYCHIATRIC PEER REVIEW contains sections on Basic Relationships and Responsibilities, Appointment of Peer Review Committees, Funding, Operations and Procedures, Medical Care Evaluation Studies, Appeals Processes, Special Problems, Confidentiality, Educational Aspects, Medical Ethics, Advocacy, Interdisciplinary Aspects, with Appendices on Model Screening Format for Inpatient Treatment for Alcoholism, Model Screening Format for Outpatient Treatment, Sample Form for Requesting Extension of Office Treatment, Claims Review Guidelines Suggestions, APA Position Papers on Peer Review, Bibliography.

Part II titled PSYCHOANALYTIC PEER REVIEW has sections on Steps in Peer Review for Psychoanalysis, Guidelines for Model Criteria Sets, Pilot Application of Guidelines for Model Criteria Sets, Sample Peer Review Form, Sample data from Pilot Applications of Guidelines, and Glossary of Terms Used.

Part III titled PEER REVIEW AND CHILD PSYCHIATRY contains sections of Special Considerations, Legal Rights of Children, Issue of Confidentiality, Conclusions, References, and Appendices on Model Screen Criteria Format for Intermediate Care, for Acute Care in a Short Stay Facilities, and for Partial Hospitalization, Outpatient Diagnostic Evaluation Procedures, and Outpatient Treatment for Children and Adolescents.

Published by the American Psychiatric Association, Washington, D.C., September 1976. 114 pages. Spiral binding. Single copies, $6.00. For orders of 10 or more, $5.00 per copy.

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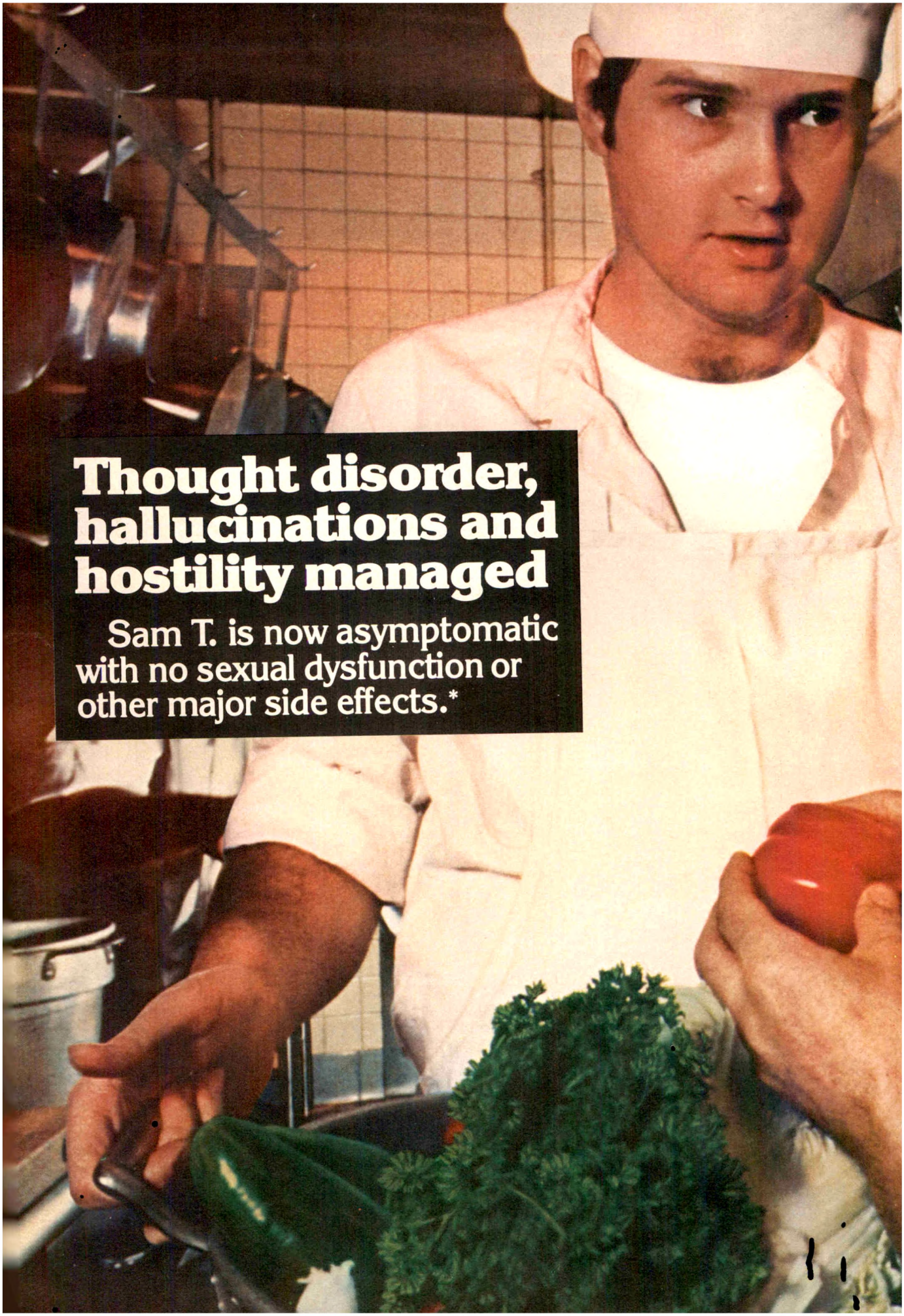
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Thought disorder, hallucinations and hostility managed
Sam T. is now asymptomatic with no sexual dysfunction or other major side effects.*



LOXITANE® Loxapine Succinate

Permits rapid management of Sam's hostility and excited behavior

Thirty-six year old salad maker, claiming to be the "Wild Man of the West" was brought to the hospital by his kitchen supervisor. History revealed prior psychiatric hospitalizations and therapies including psychotropic drugs. Hypertension was noted as a complication of earlier treatment with anti-psychotic drugs. Diagnosis: chronic schizophrenia, acute exacerbation. He responded promptly to LOXITANE® C Loxapine HCl Concentrate 50 mg stat, followed by LOXITANE® Loxapine Succinate capsules 30 mg t.i.d.

Permits aggressive titration for rapid establishment of optimal management of Sam's thought disorder and hallucinations

During first week, LOXITANE dosage increased to 50 mg q.i.d. Significant improvement in thought disorder noted by third week. Auditory hallucinations and delusions much less frequent. Patient calmer and more cooperative.

Permits sustained symptom management with minimal risk of serious adverse effects, such as sexual dysfunction

Patient revealed that while on outpatient status, he failed to take his previously prescribed antipsychotic for a ten-month period after he met and married his girlfriend. He stated the drug "didn't let me be a man." No sexual dysfunction reported with LOXITANE.

Side effects are usually mild, transitory and easily managed

Patient felt anxious because his "heart was beating so fast," and he "could not sit still." Increased pulse rate and restlessness subsided following decrease of LOXITANE to 20 mg q.i.d.

Consistence in schizophrenic symptom management and tolerance promotes patient compliance

All admitting symptomatology under control by the fourth week. By the sixth week, he was anxious to return home. Stated "this medicine doesn't make me feel drowsy like the other did." Discharged by the eighth week. He returned to work fully managed on LOXITANE 20 mg t.i.d., and free from recurrence of previously reported side effects.

*Painful ejaculation and urine containing sperm have not been reported. Endocrine disturbances such as galactorrhea and menstrual irregularities have only been rarely reported. Other sexual side effects infrequently reported. Although not reported to date, possibility of hepatic, renal, ocular, or phototoxicity cannot be ruled out at this time. Transient liver enzyme changes not definitely related to LOXITANE have been reported. Not an actual patient, but a simulation to illustrate the action of LOXITANE as reported in clinical studies.

See LOXITANE prescribing information on following page for indications, warnings and precautions and for more detailed information concerning side effects.

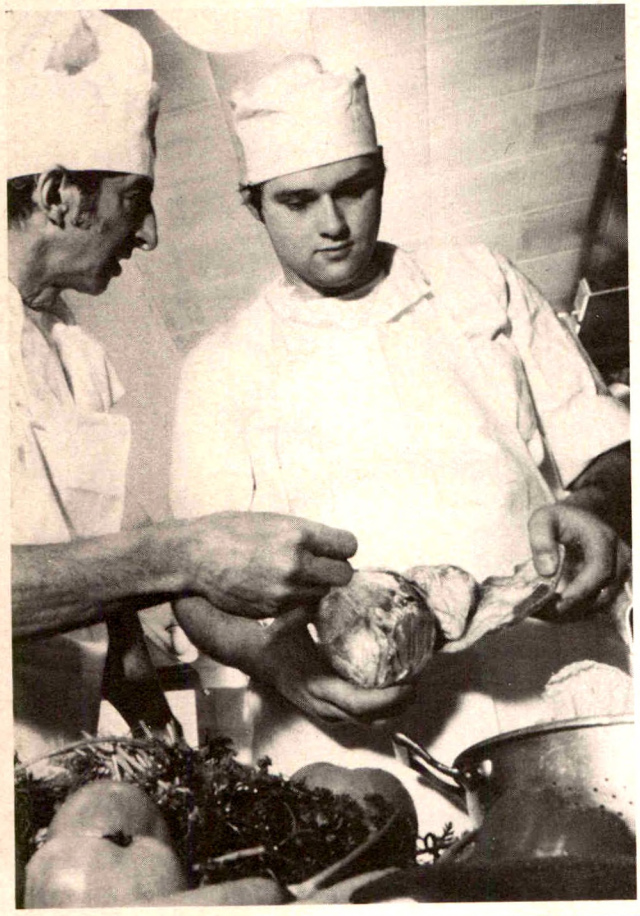
Loxitane®

LOXAPINE SUCCINATE Lederle

Capsules:
5 mg, 10 mg,
25 mg, 50 mg

Maximum management... minimum risk

Maximum management... minimum risk



Loxitane®
LOXAPINE SUCCINATE
Lederle

Recommended Daily Dosage

	MILD	MODERATE	SEVERE
Initial Dosage	10 mg b.i.d.	10 mg t.i.d or q.i.d.	25 mg b.i.d.
First 7 to 10 days	Increase dosage until psychotic symptoms are controlled. Dosage should not exceed 250 mg/day. Usual dosage during titration: 50 to 150 mg/day.		
Maintenance Dosage	Adjust to lowest effective level. Usual maintenance dosage: 60 to 100 mg/day. Many patients are controlled with dosages as low as 20 to 60 mg/day.		

Brief Summary
LOXITANE® Loxapine Succinate Capsules
LOXITANE® C Loxapine Hydrochloride Oral Concentrate
INDICATIONS: Manifestations of schizophrenia.
CONTRAINDICATIONS: Comatose or severely depressed states; hypersensitivity to the drug.
WARNINGS: Safe use during pregnancy or lactation has not been established; weigh potential benefits to possible hazards. Not recommended for use in children under 16. May impair mental and/or physical abilities especially during early therapy; warn ambulatory patients about activities requiring alertness and concomitant use of alcohol or other CNS depressants. Not recommended for management of behavioral complications in mentally retarded patients.
PRECAUTIONS: Use with extreme caution in patients with a history of convulsive disorders; use with caution in patients with cardiovascular disease or in those with glaucoma or a tendency to urinary retention particularly when on concomitant anticholinergic medication. Loxapine has an antiemetic effect in animals which might occur in man masking signs of overdosage of toxic drugs and obscuring intestinal obstruction or brain tumor. Since possible ocular toxicity cannot be excluded, observe carefully for pigmentary retinopathy and lenticular pigmentation.
ADVERSE REACTIONS: CNS effects, other than extrapyramidal, infrequent. Mild drowsiness may occur at beginning of therapy or upon dosage increase, usually subsides with continued therapy. Sedation, dizziness, faintness, staggering gait, muscle twitching, weakness and confusional states have been reported. Extrapyramidal reactions often occur early in treatment manifested by Parkinson-like symptoms (tremor, rigidity, excessive salivation, masked facies, akathisia) controllable by dosage reduction or antiparkinson drugs at usual dosages. Dystonic and dyskinetic reactions, while less frequently occurring, may be more severe, requiring dosage reduction or temporary withdrawal plus appropriate counteractive drugs. Persistent Tardive Dyskinesia may appear during prolonged therapy or following discontinuance, the risk greater in the elderly, especially females, on high dosage. Symptoms, persistent and in some patients apparently irreversible, are characterized by rhythmical involuntary movement of the tongue, face, mouth and jaw sometimes accompanied by involuntary movement of extremities. Since there is no known effective treatment, discontinue all antipsychotic drugs if symptoms appear. Reinstitution of treatment, increased dosage, or switching to another agent may mask syndrome. The syndrome may not develop if medication is stopped when fine vermicular movements of the tongue first appear. Cardiovascular Effects: Tachycardia, hypotension, hypertension, lightheadedness and syncope. ECG changes, not known to be related to loxapine use, have been reported. Skin: Dermatitis, edema of face, pruritus, seborrhea. Possible photosensitivity and/or phototoxicity; skin rashes of unknown etiology seen in a few patients in hot summer months. Anticholinergic: Dry mouth, nasal congestion, constipation, blurred vision (more likely to occur with concomitant use of antiparkinson agents). Other: Nausea, vomiting, weight gain or loss, dyspnea, ptosis, hyperpyrexia, flushed facies, headache, paresthesia, polydipsia. Rarely, galactorrhea and menstrual irregularity of unknown etiology.

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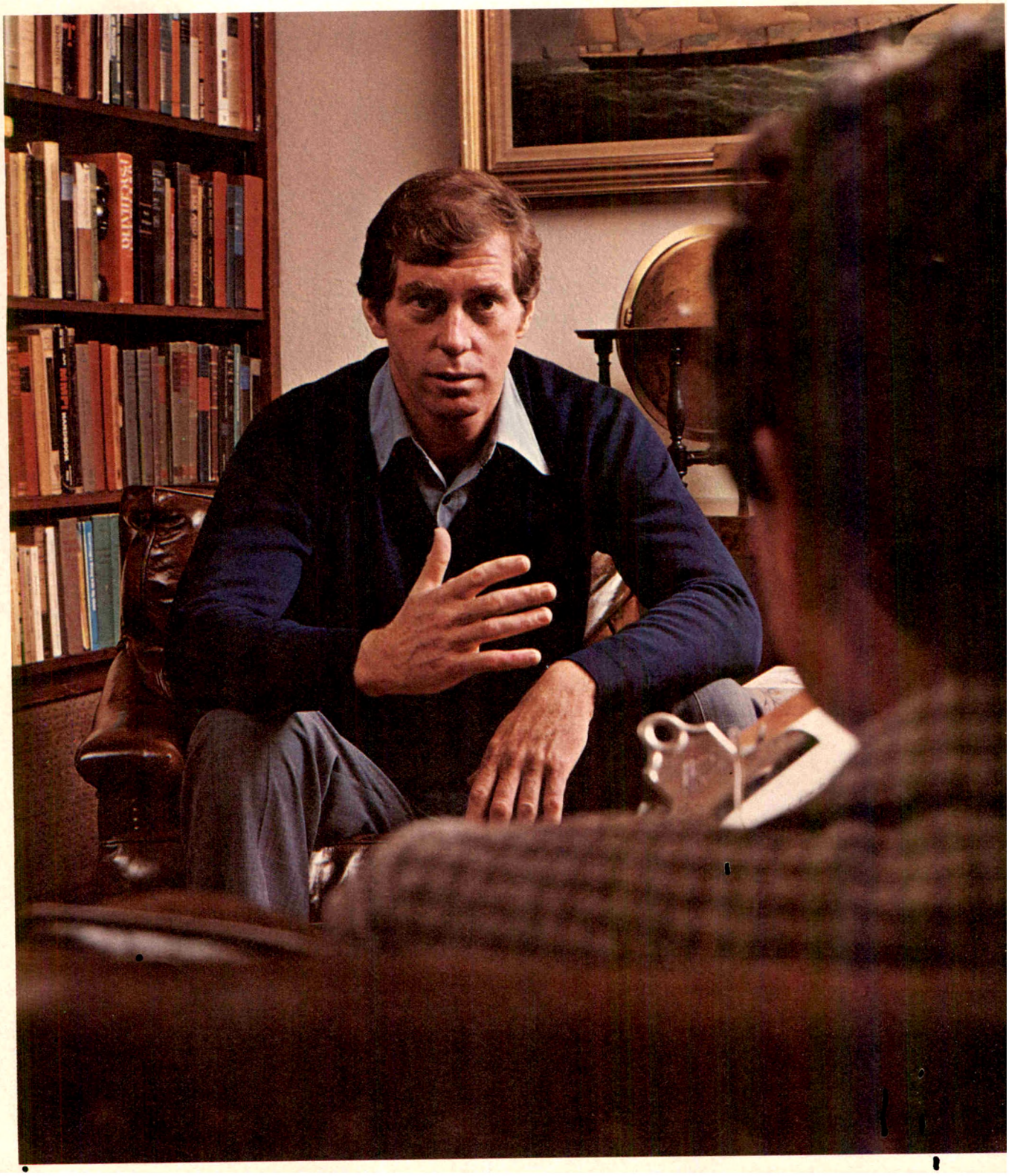
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ELAVIL usually relieves a broad range of symptoms associated with depression, including sleep disturbance, one of the most frequently observed in the "constellation of symptoms," and usually the first symptom to respond to therapy.

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Contraindications: Known hypersensitivity. Should not be given concomitantly with a monoamine oxidase inhibitor since hyperpyretic crises, severe convulsions, and deaths have occurred. When used to replace a monoamine oxidase inhibitor, allow a minimum of 14 days to elapse before initiating therapy with amitriptyline HCl. Initiate dosage of amitriptyline HCl cautiously with gradual increase in dosage until optimum response is achieved. Not recommended during the acute recovery phase following myocardial infarction.

Warnings: May block the antihypertensive action of guanethidine or similarly acting compounds. Should be used with caution in patients with a history of seizures or a history of urinary retention, or with angle-closure glaucoma or increased intraocular pressure; in patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely; arrhythmias, sinus tachycardia and prolongation of the conduction time have been reported, particularly with high doses; myocardial infarction and stroke have been reported with drugs of this class. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. May impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle. In patients who use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage. Safe use during pregnancy and lactation has not been established; in pregnant patients, nursing mothers, or women who may become pregnant, weigh possible benefits against possible hazards to mother and child. Not recommended for patients under 12 years of age.

Precautions: Schizophrenic patients may develop increased symptoms of psychosis; patients with paranoid symptomatology may have an exaggeration of such symptoms; manic depressive patients may experience a shift to the manic phase. In these circumstances, the dose of amitriptyline HCl may be reduced or a major tranquilizer, such as perphenazine, may be administered concurrently.

When given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required; paralytic ileus may occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs. Use cautiously in patients receiving large doses of ethchlorvynol, since transient delirium has been reported on concurrent administration. May enhance the response to alcohol and the effects of barbiturates and other CNS depressants. The possibility of suicide in depressed patients remains until significant remission occurs. Potentially suicidal patients should not have access to large quantities of this drug. Prescriptions should be written for the smallest amount feasible. Concurrent electroshock therapy may increase the hazards associated with such therapy; such treatment should be limited to patients for whom it is essential. When possible, discontinue the drug several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported. Use with caution in patients with impaired liver function.

Adverse Reactions: *Note:* Included in this listing are a few adverse reactions not reported with this specific drug. However, pharmacological similarities among the tricyclic antidepressant drugs require that each reaction be considered when amitriptyline is administered. *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, increased intraocular pressure, constipation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, leukopenia, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, black tongue, rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache, and malaise; these are not indicative of addiction.

Overdosage: Hospitalize as soon as possible all patients suspected of having taken an overdose. Treatment is symptomatic and supportive. In addition, the intravenous administration of 1 to 3 mg physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage should be repeated as required, particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine.

How Supplied: Tablets containing 10 mg and 25 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100, 1000, and 5000; tablets containing 50 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100 and 1000; tablets containing 75 mg and 100 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100; tablets containing 150 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 30 and 100; for intramuscular use, in 10-ml vials containing per ml: 10 mg amitriptyline HCl, 44 mg dextrose, 1.5 mg methylparaben and 0.2 mg propylparaben as preservatives, and water for injection q.s. 1 ml.

For more detailed information, consult your MSD representative or see full prescribing information. Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa. 19486

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The American Psychiatric Association announces publication of two volumes reporting the Conference on Education of Psychiatrists

held at Lake of the Ozarks, Missouri, June 9-15, 1975. Organized by the A.P.A. in cooperation with the American Academy of Child Psychiatry, the American Association of Chairmen of Departments of Psychiatry, and the American Association of Directors of Psychiatric Residency Training.

1. PSYCHIATRIC EDUCATION: PROLOGUE TO THE 1980's, Library Bound, 544 pages, $15.00.

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The volume contains ten chapters covering the topical structure of the Conference: Goals and Values; Competence, Evaluation, and Quality Control; The Resident; Faculty; Educational Methods: Means in the Service of Ends; The Program Environment; Research; The Economics of Residency Training; Psychiatry in Its Social Context; and Retrospect and Prospect. It also contains nine appendices describing the organization of the Conference and its participants, together with selected model programs and related informational materials of value.

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Papers commenting on the reports of the commissions, published here for the first time, were presented at the Conference by Melvin Sabshin, M.D., Eugene B. Brody, M.D., Jeanne Spurlock, M.D., Jack R. Ewalt, M.D., Robert J. Stoller, M.D., James R. Comer, M.D., and Bernard Holland, M.D.

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Brief Summary

ACTIONS — Pharmacologically, clorazepate dipotassium has the characteristics of the benzodiazepines. It has depressant effects on the central nervous system. The primary metabolite, nordiazepam, quickly appears in the blood stream. The serum half-life is about 2 days. The drug is metabolized in the liver and excreted primarily in the urine. (See CLINICAL PHARMACOLOGY section.)

INDICATIONS — TRANXENE is indicated for the symptomatic relief of anxiety associated with anxiety neurosis, in other psychoneuroses in which anxiety symptoms are prominent features, and as an adjunct in disease states in which anxiety is manifested.

TRANXENE is indicated for the symptomatic relief of acute alcohol withdrawal.

The effectiveness of TRANXENE in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should reassess periodically the usefulness of the drug for the individual patient.

CONTRAINDICATIONS — TRANXENE is contraindicated in patients with a known hypersensitivity to the drug, and in those with acute narrow angle glaucoma.

WARNINGS — TRANXENE is not recommended for use in depressive neuroses or in psychotic reactions.

Patients on TRANXENE should be cautioned against engaging in hazardous occupations requiring mental alertness, such as operating dangerous machinery including motor vehicles.

Since TRANXENE has a central nervous system depressant effect, patients should be advised against the simultaneous use of other CNS-depressant drugs, and cautioned that the effects of alcohol may be increased.

Because of the lack of sufficient clinical experience, TRANXENE is not recommended for use in patients less than 18 years of age.

Physical and Psychological Dependence:

Withdrawal symptoms (similar in character to those noted with barbiturates and alcohol) have occurred following abrupt discontinuance of clorazepate. Symptoms of nervousness, insomnia, irritability, diarrhea, muscle aches and memory impairment have followed abrupt withdrawal after long-term use of high dosage.

Caution should be observed in patients who are considered to have a psychological potential for drug dependence.

Evidence of drug dependence has been observed in dogs and rabbits which was characterized by convulsive seizures when the drug was abruptly withdrawn or the dose was reduced; the syndrome in dogs could be abolished by administration of clorazepate.

Usage in Pregnancy:

An increased risk of congenital malformations associated with the use of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) during the first trimester of pregnancy has been suggested in several studies. TRANXENE, a benzodiazepine derivative, has not been studied adequately to determine whether it, too, may be associated with an increased risk of fetal abnormality. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physician about the desirability of discontinuing the drug.

Usage during Lactation:

TRANXENE should not be given to nursing mothers since it has been reported that nordiazepam is excreted in human breast milk.

PRECAUTIONS — In those patients in which a degree of depression accompanies the anxiety, suicidal tendencies may be present and protective measures may be required. The least amount of drug that is feasible should be available to the patient.

Patients on TRANXENE for prolonged periods should have blood counts and liver function tests periodically. The usual precautions in treating patients with impaired renal or hepatic function should also be observed.

In elderly or debilitated patients, the initial dose should be small, and increments should be made gradually, in accordance with the response of the patient, to preclude ataxia or excessive sedation.

ADVERSE REACTIONS — The side effect most frequently reported was drowsiness. Less commonly reported (in descending order of occurrence) were: dizziness, various gastrointestinal complaints, nervousness, blurred vision, dry mouth, headache, and mental confusion. Other side effects included insomnia, transient skin rashes, fatigue, ataxia, genitourinary complaints, irritability, diplopia, depression and slurred speech.

There have been reports of abnormal liver and kidney function tests and of decrease in hematocrit.

Decrease in systolic blood pressure has been observed.

DOSAGE AND ADMINISTRATION

For the symptomatic relief of anxiety:

TRANXENE is administered orally. The capsules may be given in divided doses. The usual daily dose is 30 mg. The dose should be adjusted gradually within the range of 15 to 60 mg. daily in accordance with the response of the patient. In elderly or debilitated patients it is advisable to initiate treatment at a daily dose of 7.5 to 15 mg.

TRANXENE capsules may also be administered as a single dose daily at bedtime; the recommended initial dose is 15 mg. After the initial dose, the response of the patient may require adjustment of subsequent dosage. Lower doses may be indicated in the elderly patient. Drowsiness may occur at the initiation of treatment and with dosage increment.

TRANXENE-SD tablets (22.5 mg.) may be administered as a single dose every 24 hours. This tablet is intended as an alternate dosage form for the convenience of patients stabilized on a dose of 7.5 mg. capsules three times a day. TRANXENE-SD tablets should not be used to initiate therapy.

TRANXENE-SD HALF STRENGTH tablets (11.25 mg.) may be administered as a single dose every 24 hours.

For the symptomatic relief of acute alcohol withdrawal:

Recommended schedule: 1st 24 hours, 30 mg. TRANXENE initially, followed by 30 to 60 mg. in divided doses; 2nd 24 hours, 45 to 90 mg. in divided doses; 3rd 24 hours, 22.5 to 45 mg. in divided doses; 4th day, 15 to 30 mg. in divided doses. Thereafter gradually reduce to 7.5 to 15 mg. daily, and discontinue as soon as condition is stable. Maximum daily dose is 90 mg. Avoid excessive reductions in total drug on successive days.

DRUG INTERACTIONS — If TRANXENE is to be combined with other drugs acting on the central nervous system, careful consideration should be given to the pharmacology of the agents to be employed. Animal experience indicates that TRANXENE prolongs the sleeping time after hexobarbital or after ethyl alcohol, increases the inhibitory effects of chlorpromazine, but does not exhibit monoamine oxidase inhibition. Clinical studies have shown increased sedation with concurrent hypnotic medications. The actions of the benzodiazepines may be potentiated by barbiturates, narcotics, phenothiazines, monoamine oxidase inhibitors or other antidepressants.

If TRANXENE is used to treat anxiety associated with somatic disease states, careful attention must be paid to possible drug interaction with concomitant medication.

MANAGEMENT OF OVERDOSAGE — Overdosage is usually manifested by varying degrees of CNS depression ranging from slight sedation to coma. As in the management of overdosage with any drug, it should be borne in mind that multiple agents may have been taken.

There are no specific antidotes for the benzodiazepines. The treatment of overdosage should consist of the general measures employed in the management of overdosage of any CNS depressant. Gastric evacuation either by the induction of emesis, lavage, or both, should be performed immediately. General supportive care, including frequent monitoring of the vital signs and close observation of the patient, is indicated. Hypotension, though rarely reported, may occur with large overdoses. In such cases the use of agents such as Levophed® (levarterenol) or Aramine® (metaraminol) should be considered.

While reports indicate that individuals have survived overdoses of TRANXENE (clorazepate dipotassium) as high as 450 to 675 mg., these doses are not necessarily an accurate indication of the amount of drug absorbed since the time interval between ingestion and the institution of treatment was not always known. Sedation in varying degrees was the most common physiological manifestation of TRANXENE overdosage. Deep coma when it occurred was usually associated with the ingestion of other drugs in addition to TRANXENE.

CLINICAL PHARMACOLOGY — Studies in healthy men have shown that TRANXENE has depressant effects on the central nervous system. Prolonged administration of single daily doses as high as 120 mg. was without toxic effects. Abrupt cessation of high doses was followed in some patients by nervousness, insomnia, irritability, diarrhea, muscle aches, or memory impairment.

Absorption — Excretion:

After oral administration of TRANXENE, there is essentially no circulating parent drug. Nordiazepam, its primary metabolite, quickly appears in the blood stream. In 2 volunteers given 15 mg. (50 μC) of ^{14}C-TRANXENE, about 80% was recovered in the urine and feces within 10 days. Excretion was primarily in the urine with about 1% excreted per day on day 10.

HOW SUPPLIED — TRANXENE (clorazepate dipotassium) is supplied as capsules in three dosage strengths:

3.75 mg. capsules (gray with white cap) in bottles of 100 (NDC 0074-3417-13) and 500 (NDC 0074-3417-53). Also available in ABBO-PAC® unit dose packages of 100 capsules (NDC 0074-3417-11).

7.5 mg. capsules (gray with maroon cap) in bottles of 30 (NDC 0074-3418-30), 100 (NDC 0074-3418-13) and 500 (NDC 0074-3418-53). Also available in ABBO-PAC unit dose packages of 100 capsules (NDC 0074-3418-11).

15 mg. capsules (all gray) in bottles of 100 (NDC 0074-3419-13) and 500 (NDC 0074-3419-53). Also available in ABBO-PAC unit dose packages of 100 capsules (NDC 0074-3419-11).

TRANXENE-SD single dose tablets are supplied in two dosage strengths:

TRANXENE-SD 22.5 mg. tablets (tan-colored) in bottles of 100 (NDC 0074-2997-13).

TRANXENE-SD HALF STRENGTH 11.25 mg. tablets (blue-colored) in bottles of 30 (NDC 0074-2699-30) and 100 (NDC 0074-2699-13).

To help limit the risk of complications during alcohol withdrawal

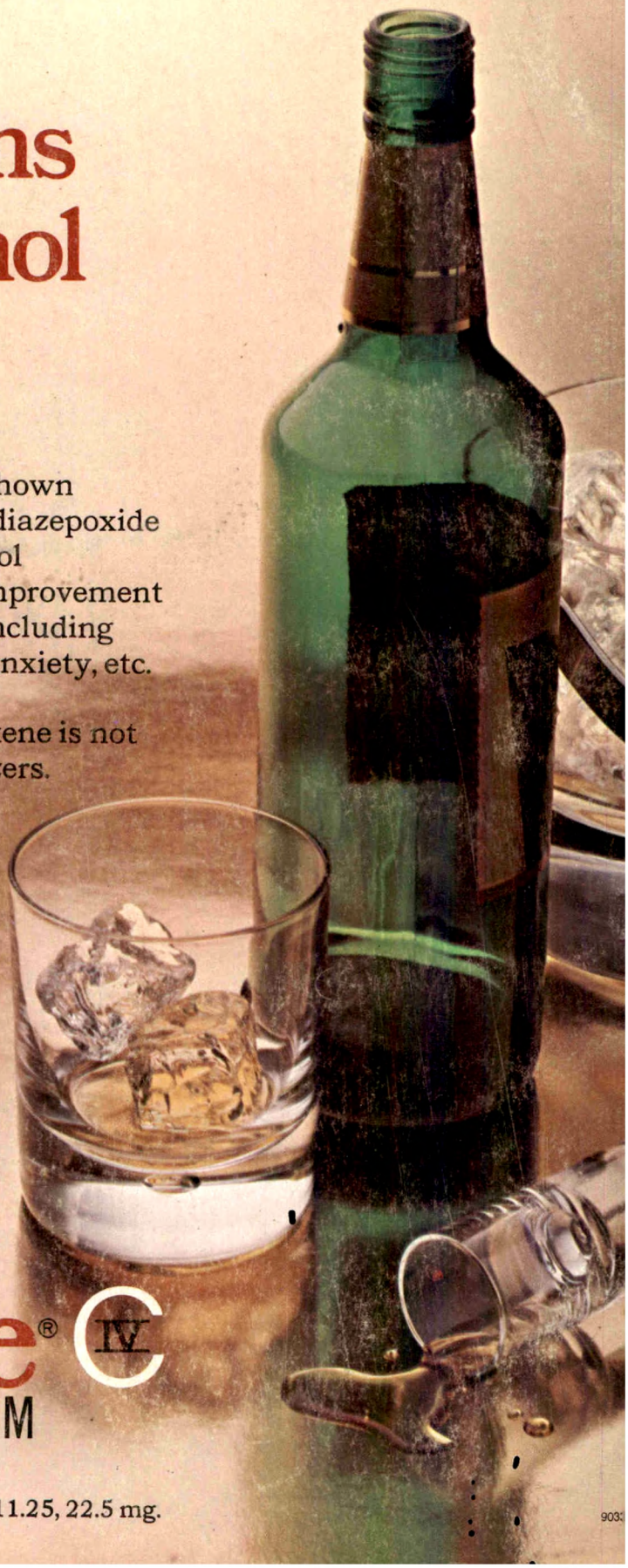
Effective Tranxene has been shown therapeutically equivalent to chlordiazepoxide in easing the distress of acute alcohol withdrawal. Rapid symptomatic improvement has been demonstrated in studies including tremors, agitation, hallucinations, anxiety, etc.

Not widely known Tranxene is not as widely known as some tranquilizers. Thus it will be unfamiliar to most patients, as well as to paramedical alcoholic support organizations.

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The Tranxene regimen is easy to manage. A simple tapering schedule is recommended, with decreasing amounts over five days. Discontinue as soon as the patient's condition is stable.

ABBOTT

See overleaf for prescribing information.



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CAPSULES: 3.75, 7.5, 15 mg; TABLETS: 11.25, 22.5 mg.

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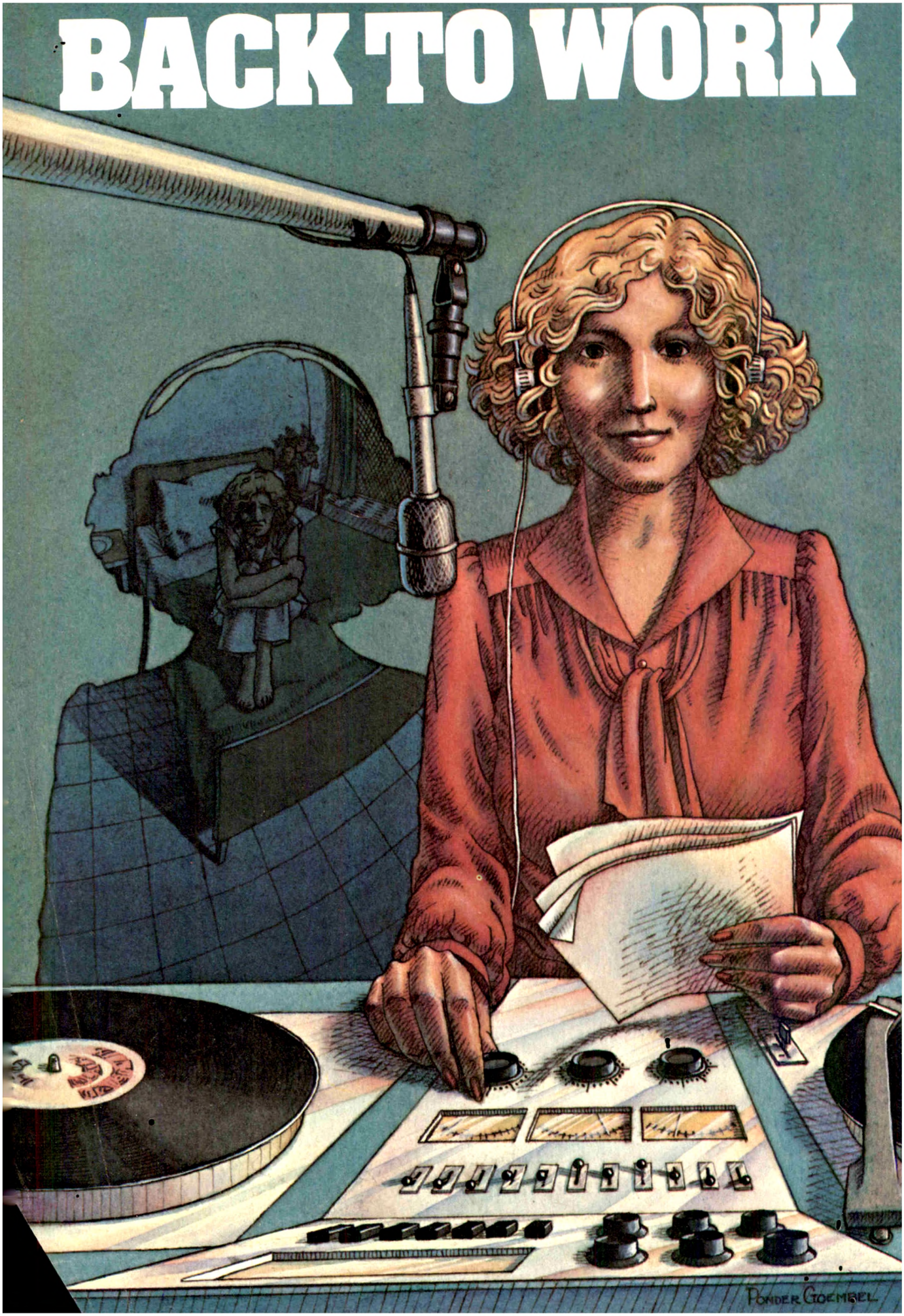
In this issue

Research on the Psychosocial Treatment of Schizophrenia: A Summary Report

By Loren R. Mosher and Samuel J. Keith

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Before prescribing, see complete prescribing information in SK&F literature or PDR. The following is a brief summary.

Indications

Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

Effective: For the management of manifestations of psychotic disorders. For control of the manifestations of manic-depressive illness (manic phase).

Probably effective: For the control of moderate to severe agitation, hyperactivity or aggressiveness in disturbed children.

Possibly effective: For control of excessive anxiety, tension and agitation as seen in neuroses.

Final classification of the less-than-effective indications requires further investigation.

Contraindications: Comatose states, presence of large amounts of C.N.S. depressants, or bone marrow depression.

Warnings: The possibility of extrapyramidal reactions from 'Thorazine' may confuse the diagnosis of Reye's syndrome or other encephalopathy. Therefore, avoid use in children or adolescents with suspected Reye's syndrome.

Avoid using in patients hypersensitive (e.g., blood dyscrasia, jaundice) to any phenothiazine. Caution patients about activities requiring alertness (e.g., operating vehicles or machinery) especially during the first few days' therapy. Avoid concomitant use with alcohol. May counteract antihypertensive effect of guanethidine and related compounds.

Use in pregnancy only when essential. There are reported instances of jaundice or prolonged extrapyramidal signs in newborn whose mothers had received chlorpromazine.

Precautions: Use cautiously in persons with cardiovascular, liver or chronic respiratory disease, or with acute respiratory infections. Due to cough reflex suppression, aspiration of vomitus is possible. May prolong or intensify the action of C.N.S. depressants, organophosphorus insecticides, heat, atropine and related drugs. (Reduce dosage of concomitant C.N.S. depressants.) Anticonvulsant action of barbiturates is not intensified. Antiemetic effect may mask signs of toxic drug overdosage or obscure diagnosis of conditions such as intestinal obstruction, brain tumor, and Reye's syndrome (see Warnings). Discontinue high-dose, long-term therapy gradually.

Patients on long-term therapy, especially high doses, should be evaluated periodically for possible adjustment or discontinuance of drug therapy.

Adverse Reactions: Drowsiness, cholestatic jaundice, agranulocytosis, eosinophilia, leukopenia, hemolytic anemia, thrombocytopenic purpura and pancytopenia; postural hypotension, tachycardia, fainting, dizziness and, occasionally, a shock-like condition; reversal of epinephrine effects; EKG changes have been reported, but relationship to myocardial damage is not confirmed; neuromuscular (extrapyramidal) reactions; pseudoparkinsonism, motor restlessness, dystonias, persistent tardive dyskinesia, hyperreflexia in the newborn; psychotic symptoms, catatonic-like states, cerebral edema; convulsive seizures; abnormality of the cerebrospinal fluid proteins; urticarial reactions and photosensitivity, exfoliative dermatitis, contact dermatitis; lactation and breast engorgement (in females on large doses), false positive pregnancy tests, amenorrhea, gynecomastia; hyperglycemia, hypoglycemia, glycosuria; dry mouth, nasal congestion, constipation, adynamic ileus, urinary retention, miosis, mydriasis; after prolonged substantial doses, skin pigmentation, epithelial keratopathy, lenticular and corneal deposits and pigmentary retinopathy, visual impairment; mild fever (after large I.M. dosage); hyperpyrexia; increased appetite and weight; a systemic lupus erythematosus-like syndrome; peripheral edema.

NOTE: Sudden death in patients taking phenothiazines (apparently due to cardiac arrest or asphyxia due to failure of cough reflex) has been reported, but no causal relationship has been established.

Supplied: Tablets, 10 mg., 25 mg., 50 mg., 100 mg. and 200 mg., in bottles of 100; Single Unit Packages of 100 (intended for institutional use only).

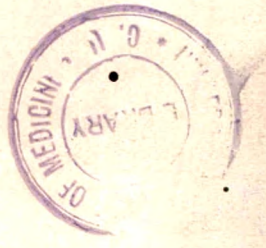
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ANNOUNCING PUBLICATION OF

THE CHRONIC MENTAL PATIENT

Report of a Conference held in January 1978
Edited by
John A. Talbott, M.D.

with contributions by James T. Barter, M.D., Paul R. Friedman, J.D., Raymond Glasscote, M.A., Trevor D. Glenn, M.D., Norman V. Lourie, M.S.W., W. Walter Menninger, M.D., Arthur T. Meyerson, M.D., Kenneth Minkoff, M.D., Samuel Muszynski, M.S.W., Lucy D. Ozarin, M.D., Gordon L. Paul, Ph.D., Ronald Peterson, Steven S. Sharfstein, M.D., Judith Clark Turner, Jane Bloom Yohalem, J.D.

This 277-page Report of the Conference, sponsored by APA and President Carter's Commission on Mental Health, identifies the chronic mental patient population, spells out where they are and what their needs and rights are. It specifies what programs work and what programs do not work in meeting the needs of these patients. It elaborates on the obstacles to implementing effective programs and the economic issues involved. It delineates the pros and cons of case management and specifies responsibility for coordinating, implementing, and monitoring services to chronic mental patients.

Finally, it proposes a *Call to Action* which opens with this statement: "There is no more urgent concern than the needs of the chronic mentally ill who suffer from severe, persistent, or recurrent mental illnesses with residual social and vocational disabilities. As a result of the deinstitutionalization programs of the past decade and the continuing growth of high risk populations that generate chronically ill, the problems associated with the care of these patients constitute a national crisis."

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1. Berne E: Principles of Group Treatment. New York, Oxford University Press, 1966, p 26
2. Blackwell B, Marley E, Price J, et al: Hypertensive interactions between monoamine oxidase inhibitors and foodstuffs. Br J Psychiatry 113:349-365, 1967
3. Gold PW, Goodwin FK: Urinary free cortisol in depression and mania. Presented at the 130th annual meeting of the American Psychiatric Association, Toronto, Ont, Canada, May 2-6, 1977
4. Brosin H: Communication systems of the consultation process, in The Psychiatric Consultation. Edited by Mendel W, Solomon P. New York, Grune & Stratton, 1968

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Tables should be self-explanatory and should supplement rather than duplicate text. Authors may be asked to delete tables that present data which could be given succinctly in text or repeat information in the Results section. Tables are generally reserved for data presentation and not used as lists or charts. They should be no wider than 120 typewriter characters, including spaces. Consult recent issues of the *Journal* for table style. Please note that no vertical rules or internal horizontal rules are used. It is usually desirable to give both numbers and percentages where applicable. All units of measurement must be specified within the table. Metric measurements are used in tables and throughout the text. Each table should be identified numerically and include a concise title.

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Figures should be used only for data that cannot be adequately presented in text or tabular form. Figures should be submitted as glossy prints with one photocopy attached to each manuscript copy. The author's name and the title of the paper should appear on a gummed label affixed to the back of each glossy. All figures should be able to withstand reduction to about 8 cm (3¼ inches). Authors are urged to consider carefully the necessity of figures; they may be asked to delete figures that repeat information available in text. It is often advisable to obtain professional assistance in the preparation of figures.

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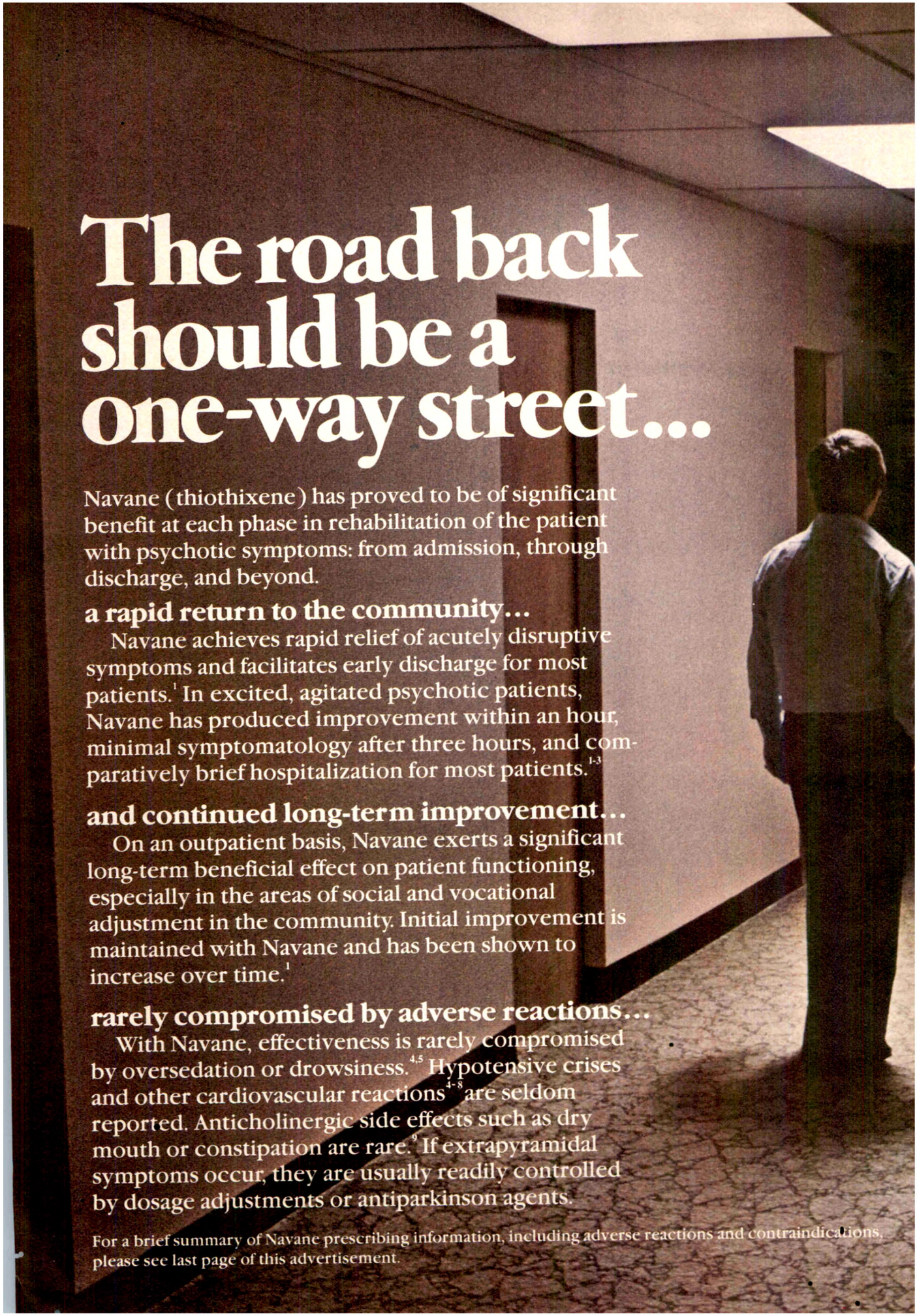
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- **"Folk" Criteria for the Diagnosis of Mental Illness in Rural Laos: On Being Insane in Sane Places**

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The road back should be a one-way street...

Navane (thiothixene) has proved to be of significant benefit at each phase in rehabilitation of the patient with psychotic symptoms: from admission, through discharge, and beyond.

a rapid return to the community...

Navane achieves rapid relief of acutely disruptive symptoms and facilitates early discharge for most patients.[1] In excited, agitated psychotic patients, Navane has produced improvement within an hour, minimal symptomatology after three hours, and comparatively brief hospitalization for most patients.[1-3]

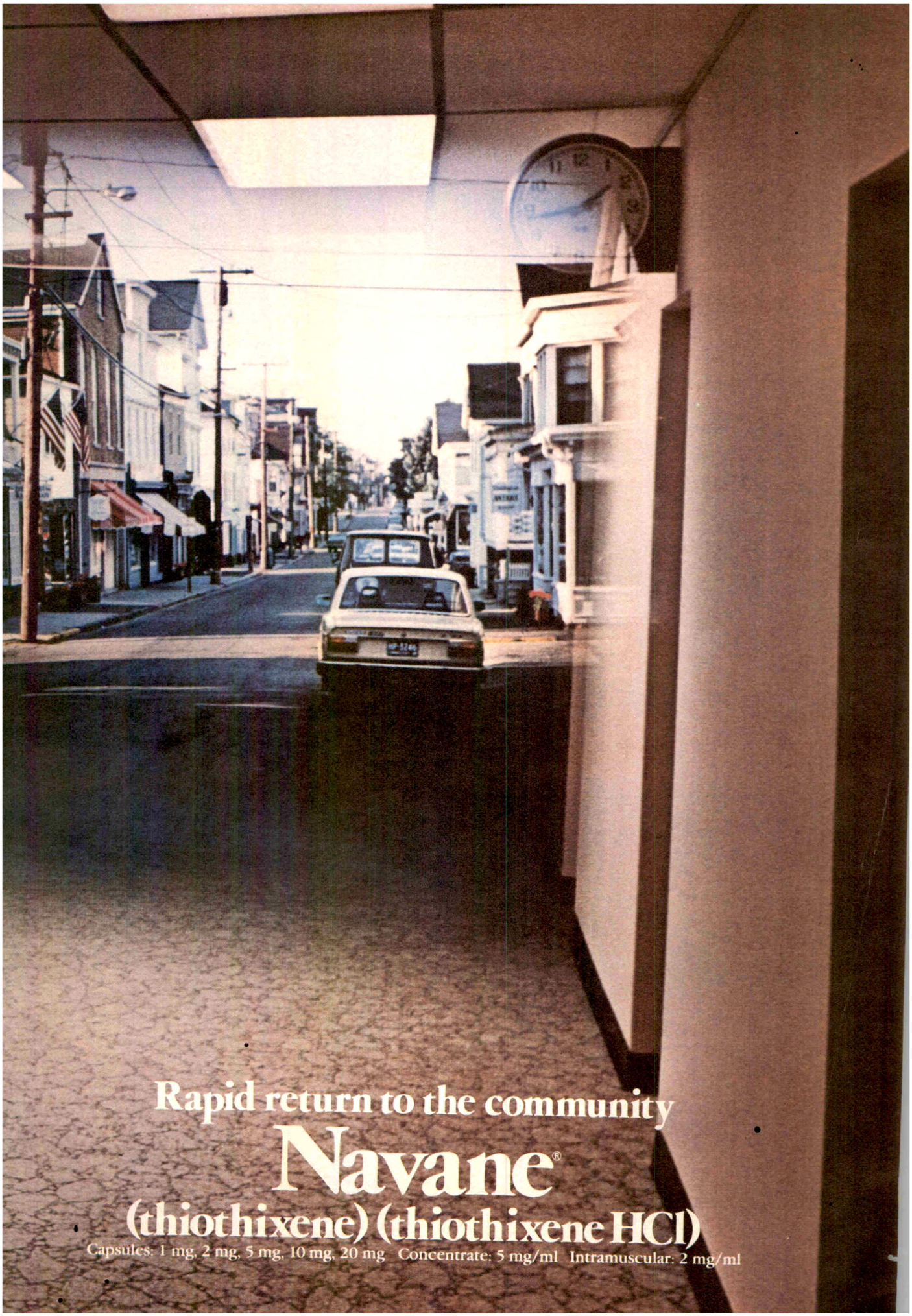
and continued long-term improvement...

On an outpatient basis, Navane exerts a significant long-term beneficial effect on patient functioning, especially in the areas of social and vocational adjustment in the community. Initial improvement is maintained with Navane and has been shown to increase over time.[1]

rarely compromised by adverse reactions...

With Navane, effectiveness is rarely compromised by oversedation or drowsiness.[4,5] Hypotensive crises and other cardiovascular reactions[4-8] are seldom reported. Anticholinergic side effects such as dry mouth or constipation are rare.[9] If extrapyramidal symptoms occur, they are usually readily controlled by dosage adjustments or antiparkinson agents.

For a brief summary of Navane prescribing information, including adverse reactions and contraindications, please see last page of this advertisement.



Rapid return to the community

Navane®

(thiothixene) (thiothixene HCl)

Capsules: 1 mg, 2 mg, 5 mg, 10 mg, 20 mg Concentrate: 5 mg/ml Intramuscular: 2 mg/ml

Rapid return to the community

Navane® (thiothixene) (thiothixene hydrochloride)

Capsules: 1 mg, 2 mg, 5 mg, 10 mg, 20 mg Concentrate: 5 mg/ml Intramuscular: 2 mg/ml

BRIEF SUMMARY OF PRESCRIBING INFORMATION
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Capsules: 1 mg, 2 mg, 5 mg, 10 mg, 20 mg (thiothixene hydrochloride) Concentrate: 5 mg/ml, Intramuscular: 2 mg/ml

Contraindications. Navane (thiothixene) is contraindicated in patients with circulatory collapse, comatose states, central nervous system depression due to any cause, and blood dyscrasias. Navane is contraindicated in individuals who have shown hypersensitivity to the drug. It is not known whether there is a cross-sensitivity between the thioxanthenes and the phenothiazine derivatives, but this possibility should be considered.

Warnings. *Usage in Pregnancy*– Safe use of Navane during pregnancy has not been established. Therefore, this drug should be given to pregnant patients only when, in the judgment of the physician, the expected benefits from the treatment exceed the possible risks to mother and fetus. Animal reproduction studies and clinical experience to date have not demonstrated any teratogenic effects.

In the animal reproduction studies with Navane, there was some decrease in conception rate and litter size, and an increase in resorption rate in rats and rabbits, changes which have been similarly reported with other psychotropic agents. After repeated oral administration of Navane to rats (5 to 15 mg/kg/day), rabbits (3 to 50 mg/kg/day), and monkeys (1 to 3 mg/kg/day) before and during gestation, no teratogenic effects were seen. (See Precautions.)

Usage in Children– The use of Navane in children under 12 years of age is not recommended because safety and efficacy in the pediatric age group have not been established.

As is true with many CNS drugs, Navane may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery, especially during the first few days of therapy. Therefore, the patient should be cautioned accordingly.

As in the case of other CNS-acting drugs, patients receiving Navane should be cautioned about the possible additive effects (which may include hypotension) with CNS depressants and with alcohol.

Precautions. An antiemetic effect was observed in animal studies with Navane; since this effect may also occur in man, it is possible that Navane may mask signs of overdosage of toxic drugs and may obscure conditions such as intestinal obstruction and brain tumor.

In consideration of the known capability of Navane and certain other psychotropic drugs to precipitate convulsions, extreme caution should be used in patients with a history of convulsive disorders or those in a state of alcohol withdrawal since it may lower the convulsive threshold. Although Navane potentiates the actions of the barbiturates, the dosage of the anticonvulsant therapy should not be reduced when Navane is administered concurrently.

Caution as well as careful adjustment of the dosage is indicated when Navane is used in conjunction with other CNS depressants other than anticonvulsant drugs.

Though exhibiting rather weak anticholinergic properties, Navane should be used with caution in patients who are known or suspected to have glaucoma, or who might be exposed to extreme heat, or who are receiving atropine or related drugs.

Use with caution in patients with cardiovascular disease.

Also, careful observation should be made for pigmentary retinopathy, and lenticular pigmentation (fine lenticular pigmentation has been noted in a small number of patients treated with Navane for prolonged periods). Blood dyscrasias (agranulocytosis, pancytopenia, thrombocytopenic purpura), and liver damage (jaundice, biliary stasis) have been reported with related drugs.

Undue exposure to sunlight should be avoided. Photosensitive reactions have been reported in patients on Navane.

Intramuscular Administration– As with all intramuscular preparations, Navane Intramuscular should be injected well within the body of a relatively large muscle. The preferred sites are the upper outer quadrant of the buttock (i.e., gluteus maximus) and the mid-lateral thigh.

The deltoid area should be used only if well developed, such as in certain adults and older children, and then only with caution to avoid radial nerve injury. Intramuscular injections should not be made into the lower and mid-thirds of the upper arm. As with all intramuscular injections, aspiration is necessary to help avoid inadvertent injection into a blood vessel.

Adverse Reactions. *Note:* Not all of the following adverse reactions have been reported with Navane (thiothixene). However, since Navane has certain chemical and pharmacologic similarities to the phenothiazines, all of the known side effects and toxicity associated with phenothiazine therapy should be borne in mind when Navane is used.

Cardiovascular effects: Tachycardia, hypotension, lightheadedness, and syncope. In the event hypotension occurs, epinephrine should not be used as a pressor agent since a paradoxical further lowering of blood pressure may result. Nonspecific EKG changes have been observed in some patients receiving Navane. These changes are usually reversible and frequently disappear on continued Navane therapy. The incidence of these changes is lower than that observed with some phenothiazines. The clinical significance of these changes is not known.

CNS effects: Drowsiness, usually mild, may occur although it usually subsides with continuation of Navane therapy. The incidence of sedation appears similar to that of the piperazine group of phenothiazines, but less than that of certain aliphatic phenothiazines. Restlessness, agitation and insomnia have been noted with Navane (thiothixene). Seizures and paradoxical exacerbation of psychotic symptoms have occurred with Navane infrequently.

Hyperreflexia has been reported in infants delivered from mothers having received structurally related drugs.

In addition, phenothiazine derivatives have been associated with cerebral edema and cerebrospinal fluid abnormalities.

Extrapyramidal symptoms, such as pseudo-parkinsonism, akathisia, and dystonia have been reported. Management of these extrapyramidal symptoms depends upon the type and severity. Rapid relief of acute symptoms may require the use of an injectable antiparkinson agent. More slowly emerging symptoms may be managed by reducing the dosage of Navane and/or administering an oral antiparkinson agent.

Persistent Tardive Dyskinesia: As with all antipsychotic agents tardive dyskinesia may appear in some patients on long term therapy or may occur after drug therapy has been discontinued. The risk seems to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities.

There is no known effective treatment for tardive dyskinesia: antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear.

Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked.

It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time, the syndrome may not develop.

Hepatic effects: Elevations of serum transaminase and alkaline phosphatase, usually transient, have been infrequently observed in some patients. No clinically confirmed cases of jaundice attributable to Navane have been reported.

Hematologic effects: As is true with certain other psychotropic drugs, leukopenia and leukocytosis, which are usually transient, can occur occasionally with Navane. Other antipsychotic drugs have been associated with agranulocytosis, eosinophilia, hemolytic anemia, thrombocytopenia and pancytopenia.

Allergic reactions: Rash, pruritus, urticaria, photosensitivity and rare cases of anaphylaxis have been reported with Navane. Undue exposure to sunlight should be avoided. Although not experienced with Navane, exfoliative dermatitis and contact dermatitis (in nursing personnel) have been reported with certain phenothiazines.

Endocrine disorders: Lactation, moderate breast enlargement and amenorrhea have occurred in a small percentage of females receiving Navane. If persistent, this may necessitate a reduction in dosage or the discontinuation of therapy. Phenothiazines have been associated with false positive pregnancy tests, gynecomastia, hypoglycemia, hyperglycemia, and glycosuria.

Autonomic effects: Dry mouth, blurred vision, nasal congestion, constipation, increased sweating, increased salivation, and impotence have occurred infrequently with Navane therapy. Phenothiazines have been associated with miosis, mydriasis, and adynamic ileus.

Other adverse reactions: Hyperpyrexia, anorexia, nausea, vomiting, diarrhea, increase in appetite and weight, weakness or fatigue, polydipsia and peripheral edema.

Although not reported with Navane, evidence indicates there is a relationship between phenothiazine therapy and the occurrence of a systemic lupus erythematosus-like syndrome.

NOTE: Sudden deaths have occasionally been reported in patients who have received certain phenothiazine derivatives. In some cases the cause of death was apparently cardiac arrest or asphyxia due to failure of the cough reflex. In others, the cause could not be determined nor could it be established that death was due to phenothiazine administration.

Dosage and Administration. Dosage of Navane should be individually adjusted depending on the chronicity and severity of the condition. In general, small doses should be used initially and gradually increased to the optimal effective level, based on patient response.

Some patients have been successfully maintained on once-a-day Navane therapy.

Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

Navane Intramuscular Solution–For Intramuscular Use Only. Where more rapid control and treatment of acute behavior is desirable, the intramuscular form of Navane may be indicated. It is also of benefit where the very nature of the patient's symptomatology, whether acute or chronic, renders oral administration impractical or even impossible.

For treatment of acute symptomatology or in patients unable or unwilling to take oral medication, the usual dose is 4 mg of Navane Intramuscular administered 2 to 4 times daily. Dosage may be increased or decreased depending on response. Most patients are controlled on a total daily dosage of 16 to 20 mg. The maximum recommended dosage is 30 mg/day. An oral form should supplant the injectable form as soon as possible. It may be necessary to adjust the dosage when changing from the intramuscular to oral dosage forms. Dosage recommendations for Navane (thiothixene) Capsules and Concentrate appear in the following paragraphs.

Navane Capsules; Navane Concentrate– In milder conditions, an initial dose of 2 mg three times daily. If indicated, a subsequent increase to 15 mg/day total daily dose is often effective.

In more severe conditions, an initial dose of 5 mg twice daily.

The usual optimal dose is 20 to 30 mg daily. If indicated, an increase to 60 mg/day total daily dose is often effective. Exceeding a total daily dose of 60 mg rarely increases the beneficial response.

Overdosage. Manifestations include muscular twitching, drowsiness, and dizziness. Symptoms of gross overdosage may include CNS depression, rigidity, weakness, torticollis, tremor, salivation, dysphagia, hypotension, disturbances of gait, or coma.

Treatment: Essentially symptomatic and supportive. For Navane oral, early gastric lavage is helpful. For Navane oral and Intramuscular, keep patient under careful observation and maintain an open airway, since involvement of the extrapyramidal system may produce dysphagia and respiratory difficulty in severe overdosage. If hypotension occurs, the standard measures for managing circulatory shock should be used (I.V. fluids and/or vasoconstrictors).

If a vasoconstrictor is needed, levarterenol and phenylephrine are the most suitable drugs. Other pressor agents, including epinephrine, are not recommended, since phenothiazine derivatives may reverse the usual pressor action of these agents and cause further lowering of blood pressure.

If CNS depression is present, recommended stimulants include amphetamine, dextroamphetamine, or caffeine and sodium benzoate. Stimulants that may cause convulsions (e.g. picrotoxin or pentylenetetrazol) should be avoided. Extrapyramidal symptoms may be treated with antiparkinson drugs.

There are no data on the use of peritoneal or hemodialysis, but they are known to be of little value in phenothiazine intoxication.

How Supplied. Navane (thiothixene) is available as capsules containing 1 mg, 2 mg, 5 mg, and 10 mg of thiothixene in bottles of 100, 1,000, and unit-dose pack of 100 (10 x 10's). Navane is also available as capsules containing 20 mg of thiothixene in bottles of 100, 500, and unit-dose pack of 100 (10 x 10's).

Navane (thiothixene hydrochloride) Concentrate is available in 120 ml (4 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, 5 mg, 6 mg, 8 mg, and 10 mg, and in 30 ml (1 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, and 5 mg. Each ml contains thiothixene hydrochloride equivalent to 5 mg of thiothixene. Contains alcohol, U.S.P. 7.0% v/v (small loss unavoidable).

Navane (thiothixene hydrochloride) Intramuscular solution is available in a 2 ml amber glass vial in packages of 10 vials. Each ml contains thiothixene hydrochloride equivalent to 2 mg of thiothixene, dextrose 5% w/v, benzyl alcohol 0.9% w/v, and propyl gallate 0.02% w/v.

References: 1. Rickels K, Norstad N, Downing R: The acutely decompensated schizophrenic patient, presented as a scientific exhibit at the American Psychiatric Association 28th Institute on Hospital and Community Psychiatry, September 20-22, 1976, Atlanta, Ga. 2. Stotsky BA: Relative efficacy of parenteral haloperidol and thiothixene for the emergency treatment of acutely excited and agitated patients. *Dis Nerv Syst* 38:967-973, Dec 1977. 3. Sloan RB, Razani J, Maloney MP, et al: Premorbid adjustment, cognition and outcome in schizophrenia, presented as a scientific exhibit at the 128th Annual Meeting of the American Psychiatric Association, May 5-9, 1975, Anaheim, California. 4. Engelhard DM, Rudorfer L, Rosen B: Haloperidol and thiothixene in the long-term treatment of chronic schizophrenic outpatients in an urban community: Social and vocational adjustment. *J Clin Psychiatry* 39:834-840, Dec 1978. 5. Itil TM, Unverdi C, Wohlrabe J, et al: Drug therapy of psychosis associated with organic brain syndrome, presented as a scientific exhibit at the American Public Health Association Centennial, Atlantic City, New Jersey, Nov 12-16, 1972. 6. Brauzer B, Goldstein B: Comparative effects of intramuscular thiothixene and trifluoperazine in psychotic patients: *J Clin Pharmacol* 8:400-403, Nov-Dec 1968. 7. Dillenkoffer RL, Gallant DM, George RB, et al: Electrocardiographic evaluations of schizophrenic patients, presented as a scientific exhibit at the 125th Annual Meeting of the American Psychiatric Association, Dallas, Texas, May 1-4, 1972. 8. Data on file at Roerig. 9. Goldstein B, Weiner D, Banas I: Clinical evaluation of thiothixene in chronic ambulatory schizophrenic patients, in Lehmann HE, Ban TA (eds): *Modern Problems in Pharmacopsychiatry.* Basel, S Karger, 1969, vol 2, pp 45-52.

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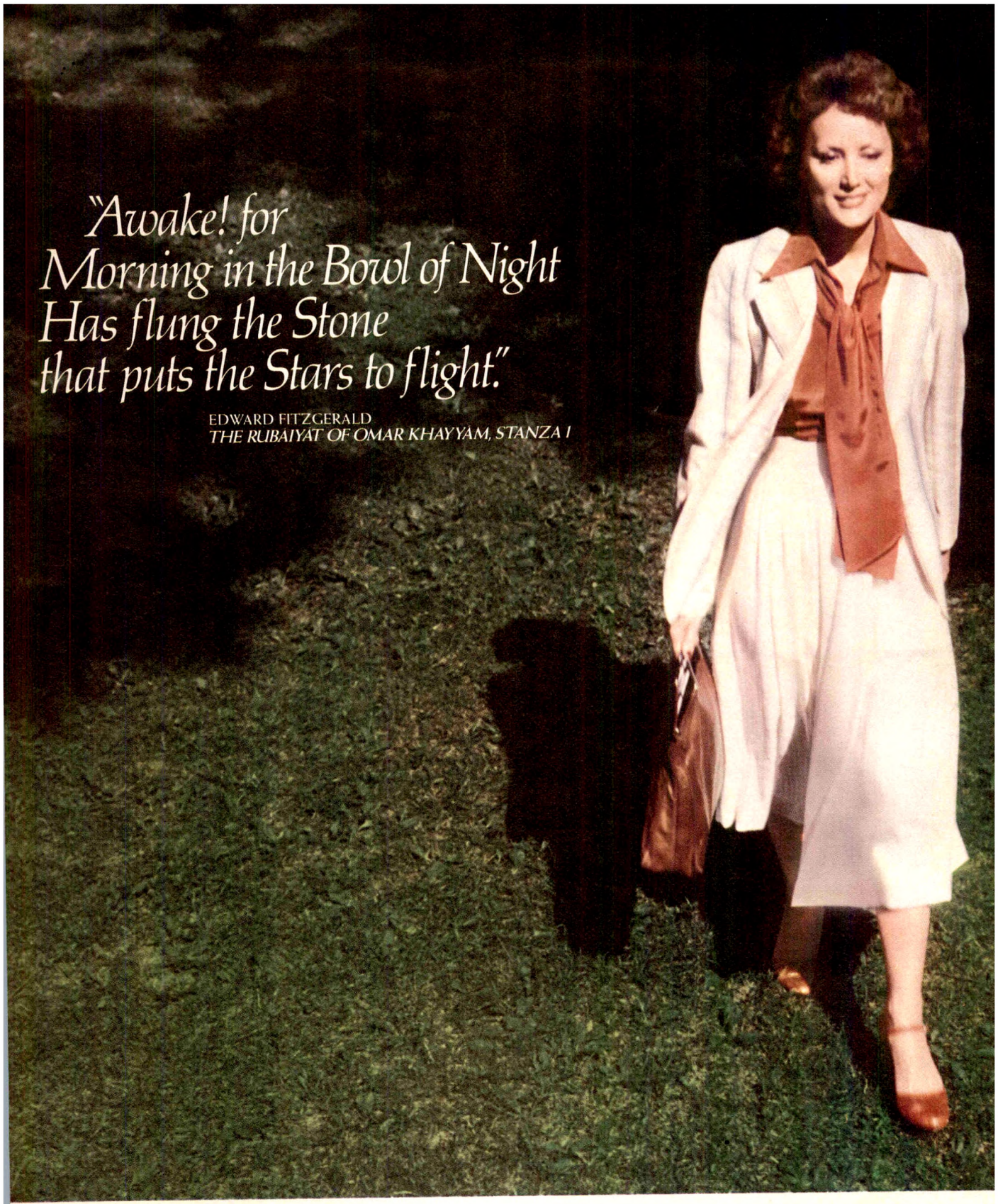
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Usage in children under 12 years of age is not recommended. MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with this drug, as serious side-effects and death have been reported with the concomitant use of certain drugs and MAO inhibitors.

In patients who may use alcohol excessively potentiation may increase the danger inherent in any suicide attempt or overdosage.

Precautions—Drowsiness may occur and patients should be cautioned against driving a motor vehicle or operating hazardous machinery. Since suicide is an inherent risk in depressed patients they should be closely supervised while receiving treatment. Although Adapin has shown effective tranquilizing activity, the possibility of activating or unmasking latent psychotic symptoms should be kept in mind.

Adverse Reactions—Dry mouth, blurred vision and constipation have been reported. Drowsiness has also been observed.

Adverse effects occurring infrequently include extrapyramidal symptoms, gastrointestinal reactions, secretory effects such as sweating, tachycardia and hypotension. Weakness, dizziness, fatigue, weight gain, edema, paresthesias, flushing, chills, tinnitus, photophobia, decreased libido, rash and pruritus may also occur.

Dosage and Administration—In mild to moderate anxiety and/or depression: 25 mg t.i.d. Increase or decrease the dosage according to individual response. Daily dosage, up to 150 mg may be taken at bedtime without loss of effectiveness. Usual optimum daily dosage is 75 mg to 150 mg per day not to exceed 300 mg per day.

Antianxiety effect usually precedes the antidepressant effect by two or three weeks.

How Supplied—Each capsule contains doxepin, as the hydrochloride: **10 mg, 25 mg, 50 mg, and 100 mg** capsules in bottles of 100 and 1000.

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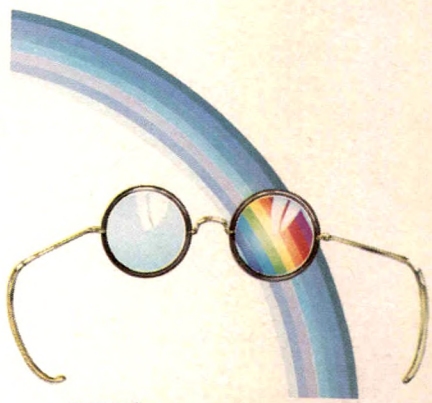
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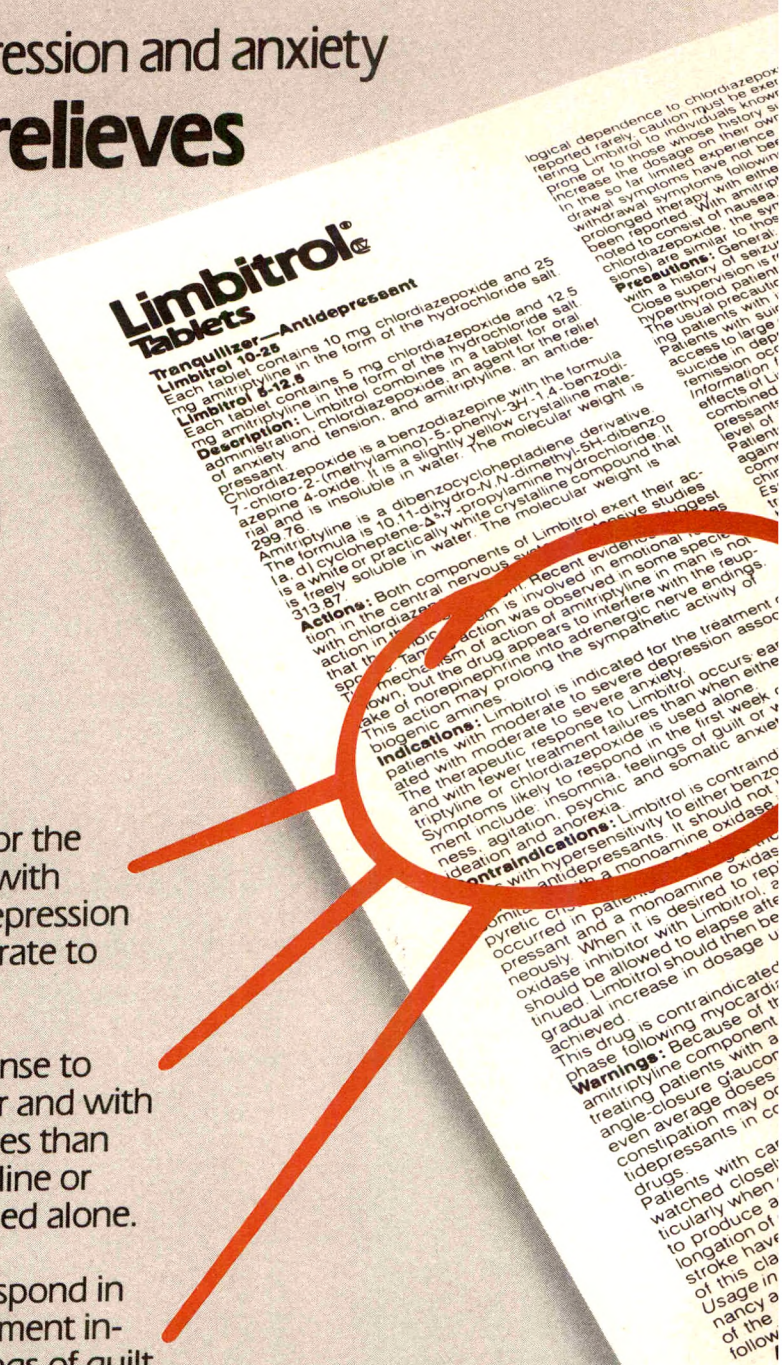
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convulsions, severe hypotension, stupor and coma.
Other symptoms may be agitation, hyperpyrexia or any of those
muscle rigidity, vomiting, hypertonicity.
listed under Adverse Reactions.
Treatment: Empty the stomach as quickly as possible by
emesis or lavage. In the comatose patient a cuff
endotracheal tube should be placed in position prior to
either of these measures. The stomach also should be emptied of activated
charcoal into the stomach but responds to stimuli only
the patient is stuporous but responds to stimuli only
close observation and nursing care may be required. It is
essential to maintain an adequate airway and fluid in-
take. Body temperature should be watched closely and
appropriate measures taken should deviations
occur.
The intramuscular or slow intravenous administration of
1 to 3 mg in adults (or 0.5 mg in children) of physo-
stigmine salicylate (Antilirium®) has been reported to
reverse the manifestations of amitriptyline overdosage
Because of its relatively short half-life, additional doses
may be needed at intervals of 30 minutes to 2 hours.
Because the manifestations of amitriptyline overdosage
may be treated by the use of barbiturates, cardiac
anesthetic rather than the use of barbiturates. Cardiac
monitoring is advisable, and the cautious use of digitalis
or other antiarrhythmic agents should be considered if
serious cardiovascular abnormalities occur. Serum
potassium levels should be monitored and kept within
normal limits by the use of appropriate I.V. fluids. Stan-
dard measures including oxygen, I.V. fluids, plasma ex-
panders and corticosteroids may be used to control
circulatory shock.
Dialysis is unlikely to be of value, as it has not proven
useful in overdosages of either amitriptyline or chlor-
diazepoxide. Since many suicidal attempts involve mul-
tiple drugs including barbiturates, the possibility of
dialysis being beneficial for removal of other drugs
should not be overlooked.
Treatment should be continued for at least 48 hours,
along with cardiac monitoring in patients who do not
respond to therapy promptly. Since relapses are fre-
quent, patients should be hospitalized until their condi-
tions remain stable without physostigmine for at least 24
hours.
Since overdosage is often deliberate, patients may
attempt suicide by other means during the recovery
phase.

References:
1. Granacher RP, Baldessarini RJ: Physostigmine: Its use
in acute anticholinergic syndrome with antidepressant
and antiparkinson drugs. Arch Gen Psychiatry 32:375-
380, Mar 1975
2. Burks JS, Walker JE, Rumack BH, Ott JE: Tricyclic
antidepressant poisoning: Reversal of coma,
choreoathetosis, and myoclonus by physostigmine.
JAMA 230:1405-1407, Dec 9, 1974
3. Snyder BD, Blonde L, McWhirter WR: Reversal of
amitriptyline intoxication by physostigmine. JAMA
230:1433-1434, Dec 9, 1974

Dosage and Administration: Optimum dosage varies with the severity of the symptoms and the response of the individual patient. When a satisfactory response is obtained, dosage should be reduced to the smallest amount needed to maintain the remission. The larger portion of the total daily dose may be taken at bedtime. In some patients, a single dose at bedtime may be sufficient. In general, lower dosages are recommended for elderly patients.
Limbitrol 10-25 is recommended in an initial dosage of three or four tablets daily in divided doses; this may be increased to six tablets daily as required. Some patients respond to smaller doses and can be maintained on two tablets daily.
Limbitrol 5-12.5 in an initial dosage of three or four tablets daily in divided doses may be satisfactory in patients who do not tolerate higher doses.

How Supplied: White, film-coated tablets, each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt) and blue, film-coated tablets, each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)—bottles of 100 and 500; Tel-E-Dose® packages of 100. Prescription Paks of 50.

THIS PRODUCT INFORMATION ISSUED OCTOBER 1978.

ROCHE LABORATORIES
Division of Hoffmann-La Roche Inc.
Nutley, New Jersey 07110

ROCHE

Limbitrol®

Tablets 10-25 each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt)

Tablets 5-12.5 each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)

CIV

Please see summary of product information on last page of this advertisement.

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Psychological Dependence: Experience
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LIMBITROL® TABLETS Tranquilizer—Antidepressant

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Relief of moderate to severe depression associated with moderate to severe anxiety.

Contraindications: Known hypersensitivity to benzodiazepines or tricyclic antidepressants: Do not use with monoamine oxidase (MAO) inhibitors or within 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use; then initiate cautiously, gradually increasing dosage until optimal response is achieved. Contraindicated during acute recovery phase following myocardial infarction.

Warnings: Use with great care in patients with history of urinary retention or angle-closure glaucoma. Severe constipation may occur in patients taking tricyclic antidepressants and anticholinergic-type drugs. Closely supervise cardiovascular patients. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, especially high doses. Myocardial infarction and stroke reported with use of this class of drugs.)

***Usage in Pregnancy:* Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Since physical and psychological dependence to chlordiazepoxide have been reported rarely, use caution in administering Limbitrol to addiction-prone individuals or those who might increase dosage; withdrawal symptoms following discontinuation of either component alone have been reported (nausea, headache and malaise for amitriptyline; symptoms [including convulsions] similar to those of barbiturate withdrawal for chlordiazepoxide).

Precautions: Use with caution in patients with a history of seizures, in hyperthyroid patients or those on thyroid medication, and in patients with impaired renal or hepatic function. Because of the possibility of suicide in depressed patients, do not permit easy access to large quantities in these patients. Caution patients about possible combined effects with alcohol and other CNS depressants and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Periodic liver function tests and blood counts are recommended during prolonged treatment. Amitriptyline component may block action of guanethidine or similar antihypertensives. Concomitant use with other psychotropic drugs has not been evaluated: sedative effects may be additive. Discontinue several days before surgery. Limit concomitant administration of ECT to essential treatment. See Warnings for precautions about pregnancy. Limbitrol should not be taken during the nursing period. Not recommended in children under 12.

In the elderly and debilitated, limit to smallest effective dosage to preclude ataxia, oversedation, confusion or anticholinergic effects.

Adverse Reactions: Most frequently reported are those associated with either component alone: drowsiness, dry mouth, constipation, blurred vision, dizziness and bloating. Less frequently occurring reactions include vivid dreams, impotence, tremor, confusion and nasal congestion. Many depressive symptoms including anorexia, fatigue, weakness, restlessness and lethargy have been reported as side effects of both Limbitrol and amitriptyline. Granulocytopenia, jaundice and hepatic dysfunction have been observed rarely.

This list includes adverse reactions not reported with Limbitrol but requiring consideration because they have been reported with one or both components or closely related drugs:

Cardiovascular: Hypotension, hypertension, tachycardia, palpitations, myocardial infarction, arrhythmias, heart block, stroke.

Psychiatric: Euphoria, apprehension, poor concentration, delusions, hallucinations, hypomania and increased or decreased libido.

Neurologic: Incoordination, ataxia, numbness, tingling and paresthesias of the extremities, extrapyramidal symptoms, syncope, changes in EEG patterns.

Anticholinergic: Disturbance of accommodation, paralytic ileus, urinary retention, dilatation of urinary tract.

Allergic: Skin rash, urticaria, photosensitization, edema of face and tongue, pruritus.

Hematologic: Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia.

Gastrointestinal: Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, black tongue.

Endocrine: Testicular swelling and gynecomastia in the male, breast enlargement, galactorrhea and minor menstrual irregularities in the female and elevation and lowering of blood sugar levels.

Other: Headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, jaundice, alopecia, parotid swelling.

Overdosage: Immediately hospitalize patient suspected of having taken an overdose. Treatment is symptomatic and supportive. I.V. administration of 1 to 3 mg physostigmine salicylate has been reported to reverse the symptoms of amitriptyline poisoning. See complete product information for manifestation and treatment.

Dosage: Individualize according to symptom severity and patient response. Reduce to smallest effective dosage when satisfactory response is obtained. Larger portion of daily dose may be taken at bedtime. Single *h.s.* dose may suffice for some patients. Lower dosages are recommended for the elderly.

Limbitrol 10-25, initial dosage of three to four tablets daily in divided doses, increased up to six tablets or decreased to two tablets daily as required. Limbitrol 5-12.5, initial dosage of three to four tablets daily in divided doses, for patients who do not tolerate higher doses.

Supplied: Limbitrol 10-25 tablets each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt) and Limbitrol 5-12.5 tablets each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)—bottles of 100 and 500; Tel-E-Dose* packages of 100; Prescription Paks of 50.

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Roche Laboratories
Division of Hoffmann-La Roche Inc.
Nutley, New Jersey 07110

How to initiate and maintain therapy

Select dosage strength appropriate for each patient

☐ Limbitrol 10-25 is recommended for most patients

☐ Limbitrol 5-12.5 may be indicated for those who do not tolerate higher doses and for elderly patients

Specify daily dosage based on symptom severity

☐ An initial dosage of three tablets is recommended.

☐ Dosage may be increased to six tablets or decreased to two tablets daily as necessary.

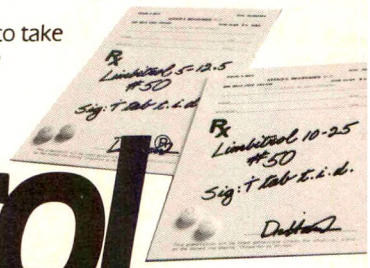
☐ Once a satisfactory response is obtained, evaluate response and, if necessary, reduce dosage to smallest amount needed.

Utilize dosage options to best accommodate individual patient needs

☐ T.I.D. or Q.I.D., familiar regimens most suited for patients who tolerate medication without undue drowsiness

☐ Two tablets one hour before bedtime and one tablet midday may minimize daytime drowsiness and help relieve a common target symptom—insomnia

☐ Entire dosage h.s. to take maximum advantage of the sedative effect



Limbitrol

Tablets 10-25 each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt)

Tablets 5-12.5 each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)

CIV

How to make each patient an informed patient

1. Discuss with patients the probability that they will experience drowsiness, especially during the first week.

2. Reassure your patients that drowsiness is one indication that the medication is working and that it may help alleviate their insomnia.

3. Encourage patients to report if drowsiness becomes troublesome so that, if necessary, dosage schedule can be adjusted or the symptom treated.

4. Caution patients about the combined effects with alcohol or other CNS depressants. Let them know that the additive effects may produce a harmful level of sedation and CNS depression.

5. Caution patients about activities requiring complete mental alertness, such as operating machinery or driving a car.

6. Warn pregnant patients and patients of childbearing age that the safety of Limbitrol in pregnancy has not yet been established.

Please see complete product disclosure for other pertinent information.

Limbitrol should not be used under the following circumstances:

1. Hypersensitivity to benzodiazepines or tricyclic antidepressants.
2. Should not be given with an MAO inhibitor. To replace an MAO inhibitor with Limbitrol, discontinue MAO inhibitor for a minimum of 14 days before cautiously initiating Limbitrol therapy.
3. During the acute recovery phase following myocardial infarction.

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Hydrochloride
Elixir USP

Prolixin® Tablets
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Hydrochloride
Tablets USP

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5 mg

2.5 mg

1 mg

0.5 mg per ml

See next page for brief summaries.

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CONTRAINDICATIONS: In presence of suspected or established subcortical brain damage. In patients who have a blood dyscrasia or liver damage, or who are receiving large doses of hypnotics, or who are comatose or severely depressed. In patients who have shown hypersensitivity to fluphenazine; cross-sensitivity to phenothiazine derivatives may occur.

WARNINGS: Mental and physical abilities required for driving a car or operating heavy machinery may be impaired by use of this drug. Potentiation of effects of alcohol may occur. Safety and efficacy in children have not been established because of inadequate experience in use in children.

Usage in Pregnancy: Safety for use during pregnancy has not been established; weigh possible hazards against potential benefits if administering this drug to pregnant patients.

PRECAUTIONS: Caution must be exercised if another phenothiazine compound caused cholestatic jaundice, dermatoses or other allergic reactions because of the possibility of cross-sensitivity. When psychotic patients on large doses of a phenothiazine drug are to undergo surgery, hypotensive phenomena should be watched for; less anesthetics or central nervous system depressants may be required. Because of added anticholinergic effects, fluphenazine may potentiate the effects of atropine.

Use fluphenazine cautiously in patients exposed to extreme heat or phosphorus insecticides; in patients with a history of convulsive disorders since grand mal convulsions have occurred; and in patients with special medical disorders such as mitral insufficiency or other cardiovascular diseases, and pheochromocytoma. Bear in mind that with prolonged therapy there is the possibility of liver damage, pigmentary retinopathy, lenticular and corneal deposits, and development of irreversible dyskinesia.

There is sufficient experimental evidence to conclude that chronic administration of antipsychotic drugs which increase prolactin secretion has the potential to induce mammary neoplasms in rodents under the appropriate conditions. There are recognized differences in the physiological role of prolactin between rodents and humans. Since there are, at present, no adequate epidemiological studies, the relevance to human mammary cancer risk from prolonged exposure to fluphenazine hydrochloride and other antipsychotic drugs is not known.

Periodic checking of hepatic and renal functions and blood picture should be done. Monitor renal function of patients on long-term therapy; if BUN becomes abnormal, discontinue fluphenazine. "Silent pneumonias" are possible.

Abrupt Withdrawal: In general, phenothiazines do not produce psychic dependence. However, gastritis, nausea and vomiting, dizziness, and tremulousness have been reported following abrupt cessation of high dose therapy; reports suggest that these symptoms can be reduced if concomitant antiparkinsonian agents are continued for several weeks after the phenothiazine is withdrawn.

ADVERSE REACTIONS: *Central Nervous System*—Extrapyramidal symptoms are most frequently reported. Most often these symptoms are reversible, but they may be persistent. They include pseudoparkinsonism, dystonia, dyskinesia, akathisia, oculogyric crises, opisthotonos, hyperreflexia. The incidence and severity of such reactions will depend more on individual patient sensitivity, but dosage level and patient age are also determinants. As these reactions may be alarming, the patient should be forewarned and reassured. These reactions can usually be controlled by administration of an anti-parkinsonian drug such as benztropine mesylate and by subsequent reduction in dosage.

Persistent Tardive Dyskinesia: As with all antipsychotic agents, persistent and sometimes irreversible tardive dyskinesia may appear in some patients on long-term therapy or may occur after discontinuation of drug. The risk seems greater in elderly patients, especially females, on high dosages. The syndrome is characterized by rhythmical involuntary movements of tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and may be accompanied by involuntary movements of extremities. There is no known effective therapy for tardive dyskinesia; usually the symptoms are not alleviated by antiparkinsonism agents. If the symptoms appear, discontinuation of all antipsychotic agents is suggested. The syndrome may be masked if treatment is reinstituted, or drug dosage increased, or a different antipsychotic agent used. Reports are that fine vermicular movements of the tongue may be an early sign of the syndrome which may not develop if medication is stopped at that time.

Phenothiazine derivatives have been known to cause restlessness, excitement, or bizarre dreams; reactivation or aggravation of psychotic processes may be encountered. If drowsiness or lethargy occur, the dosage may need to be reduced. Dosages, far in excess of the recommended amounts, may induce a catatonic-like state.

Autonomic Nervous System—Hypertension and fluctuations in blood pressure have been reported. Although hypotension is rarely a problem, patients with pheochromocytoma, cerebral vascular or renal insufficiency or severe cardiac reserve deficiency such as mitral insufficiency appear to be particularly prone to this reaction and should be observed carefully. Supportive measures including intravenous vasopressor drugs should be instituted immediately should severe hypotension occur; Levarterenol Bitartrate Injection is the most suitable drug; *epinephrine should not be used* since phenothiazine derivatives have been found to reverse its action. Nausea, loss of appetite, salivation, polyuria, perspiration, dry mouth, headache and constipation may occur. Reducing or temporarily discontinuing the dosage will usually control these effects. Blurred vision, glaucoma, bladder paralysis, fecal impaction, paralytic ileus, tachycardia, or nasal congestion have occurred in some patients on phenothiazine derivatives.

Metabolic and Endocrine—Weight change, peripheral edema, abnormal lactation, gynecomastia, menstrual irregularities, false results on pregnancy tests, impotency in men and increased libido in women have occurred in some patients on phenothiazine therapy.

Allergic Reactions—Itching, erythema, urticaria, seborrhea, photosensitivity, eczema and exfoliative dermatitis have been reported with phenothiazines. The possibility of anaphylactoid reactions should be borne in mind.

Hematologic—Blood dyscrasias including leukopenia, agranulocytosis, thrombocytopenic or nonthrombocytopenic purpura, eosinophilia, and pancytopenia have been observed with phenothiazines. If soreness of the mouth, gums or throat or any symptoms of upper respiratory infection occur and confirmatory leukocyte count indicates cellular depression, therapy should be discontinued and other appropriate measures instituted immediately.

Hepatic—Liver damage manifested by cholestatic jaundice, particularly during the first months of therapy, may occur; treatment should be discontinued. A cephalin flocculation increase, sometimes accompanied by alterations in other liver function tests, has been reported in patients who have had no clinical evidence of liver damage.

Others—Sudden deaths have been reported in hospitalized patients on phenothiazines. Previous brain damage or seizures may be predisposing factors. High doses should be avoided in known seizure patients. Shortly before death, several patients showed flare-ups of psychotic behavior patterns. Autopsy findings have usually revealed acute fulminating pneumonia or pneumonitis, aspiration of gastric contents, or intramyocardial lesions. Although not a general feature of fluphenazine, potentiation of central nervous system depressants such as opiates, analgesics, antihistamines, barbiturates, and alcohol may occur.

Systemic lupus erythematosus-like syndrome, hypotension severe enough to cause fatal cardiac arrest, altered electrocardiographic and electroencephalographic tracings, altered cerebrospinal fluid proteins, cerebral edema, asthma, laryngeal edema, and angioneurotic edema; with long-term use, skin pigmentation and lenticular and corneal opacities have occurred with phenothiazines.

For full prescribing information, consult package inserts.

HOW SUPPLIED: *Tablets*—1 mg in bottles of 50 and 500; 2.5 mg and 5 mg in bottles of 50 and 500 and in Unimatic® single-dose cartons of 100; 10 mg in bottles of 50 and 500. *Elixir*—in bottles of 473 ml (1 pint) and in 60 ml dropper-assembly bottles with dropper calibrated at 0.5 ml (0.25 mg), 1 ml (0.5 mg), 1.5 ml (0.75 mg), and 2 ml (1 mg). *Injection*—in multiple-dose vials of 10 ml.

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CONTRAINDICATIONS: In presence of suspected or established subcortical brain damage. patients who have a blood dyscrasia or liver damage, or who are receiving large doses hypnotics, or who are comatose or severely depressed. In patients who have shown hyperse sitivity to fluphenazine; cross-sensitivity to phenothiazine derivatives may occur.

Not intended for use in children under 12.

WARNINGS: Mental and physical abilities required for driving a car or operating heavy m chinery may be impaired by use of this drug. Physicians should be alert to the possibility th severe adverse reactions may occur which require immediate medical attention. Potentiation effects of alcohol may occur. Safety and efficacy in children have not been established becau of inadequate experience in use in children.

Usage in Pregnancy: Safety for use during pregnancy has not been established; wei possible hazards against potential benefits if administering this drug to pregnant patients.

PRECAUTIONS: Caution must be exercised if another phenothiazine compound caused chole tatic jaundice, dermatoses or other allergic reactions because of the possibility of cros sensitivity. When psychotic patients on large doses of a phenothiazine drug are to under surgery, hypotensive phenomena should be watched for; less anesthetics or central nervo system depressants may be required. Because of added anticholinergic effects, fluphenazi may potentiate the effects of atropine.

Use fluphenazine decanoate cautiously in patients exposed to extreme heat or phosphor insecticides; in patients with a history of convulsive disorders since grand mal convulsions ha occurred; and in patients with special medical disorders such as mitral insufficiency or ot cardiovascular diseases, and pheochromocytoma. Bear in mind that with prolonged thera there is the possibility of liver damage, pigmentary retinopathy, lenticular and corneal depos and development of irreversible dyskinesia.

Fluphenazine decanoate should be administered under the direction of a physician exp enced in the clinical use of psychotropic drugs. Periodic checking of hepatic and renal functi and blood picture should be done. Renal function of patients on long-term therapy should monitored; if BUN becomes abnormal, treatment should be discontinued. "Silent pneumoni are possible.

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ADVERSE REACTIONS: *Central Nervous System*—Extrapyramidal symptoms are most quently reported. Most often these symptoms are reversible, but they may be persistent. Th include pseudoparkinsonism, dystonia, dyskinesia, akathisia, oculogyric crises, opisthoton hyperreflexia. Muscle rigidity sometimes accompanied by hyperthermia has been repor following use of fluphenazine decanoate. One can expect a higher incidence of such reacti with fluphenazine decanoate than with less potent piperazine derivatives or straight-ch phenothiazines. The incidence and severity will depend more on individual patient sensitivity, dosage level and patient age are also determinants. As these reactions may be alarming, patient should be forewarned and reassured. These reactions can usually be controlled administration of an antiparkinsonian drug such as benztropine mesylate and by subsequ reduction in dosage.

Persistent Tardive Dyskinesia: As with all antipsychotic agents, persistent and sometir irreversible tardive dyskinesia may appear in some patients on long-term therapy or may oc after discontinuation of drug. The risk seems greater in elderly patients, especially females, high dosages. The syndrome is characterized by rhythmical involuntary movements of tong face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chew movements) and may be accompanied by involuntary movements of extremities. There is known effective therapy for tardive dyskinesia; usually the symptoms are not alleviated antiparkinsonism agents. If the symptoms appear, discontinuation of all antipsychotic agent suggested. The syndrome may be masked if treatment is reinstituted, or drug dosage increas or a different antipsychotic agent used. Reports are that fine vermicular movements of the ton may be an early sign of the syndrome which may not develop if medication is stopped at that ti

Phenothiazine derivatives have been known to cause restlessness, excitement, or biz dreams; reactivation or aggravation of psychotic processes may be encountered. If drowsin or lethargy occur, the dosage may need to be reduced. Dosages, far in excess of the rec mended amounts, may induce a catatonic-like state.

Autonomic Nervous System—Hypertension and fluctuations in blood pressure have b reported. Although hypotension is rarely a problem, patients with pheochromocytoma, cere vascular or renal insufficiency or severe cardiac reserve deficiency such as mitral insufficie appear to be particularly prone to this reaction and should be observed carefully. Suppo measures including intravenous vasopressor drugs should be instituted immediately sh severe hypotension occur; Levarterenol Bitartrate Injection is the most suitable drug; *epineph should not be used* since phenothiazine derivatives have been found to reverse its ac Nausea, loss of appetite, salivation, polyuria, perspiration, dry mouth, headache and const tion may occur. Reducing or temporarily discontinuing the dosage will usually control th effects. Blurred vision, glaucoma, bladder paralysis, fecal impaction, paralytic ileus, tachyca or nasal congestion have occurred in some patients on phenothiazine derivatives.

Metabolic and Endocrine—Weight change, peripheral edema, abnormal lactat gynecomastia, menstrual irregularities, false results on pregnancy tests, impotency in men increased libido in women have occurred in some patients on phenothiazine therapy.

Allergic Reactions—Itching, erythema, urticaria, seborrhea, photosensitivity, eczema exfoliative dermatitis have been reported with phenothiazines. The possibility of anaphylac reactions should be borne in mind.

Hematologic—Blood dyscrasias including leukopenia, agranulocytosis, thrombocytopen nonthrombocytopenic purpura, eosinophilia, and pancytopenia have been observed phenothiazines. If soreness of the mouth, gums or throat or any symptoms of upper respira infection occur and confirmatory leukocyte count indicates cellular depression, therapy sh be discontinued and other appropriate measures instituted immediately.

Hepatic—Liver damage manifested by cholestatic jaundice, particularly during the months of therapy, may occur; treatment should be discontinued. A cephalin flocculatio crease, sometimes accompanied by alterations in other liver function tests, has been reporte patients who have had no clinical evidence of liver damage.

Others—Sudden deaths have been reported in hospitalized patients on phenothiazi Previous brain damage or seizures may be predisposing factors. High doses should be avo in known seizure patients. Shortly before death, several patients showed flare-ups of psyc behavior patterns. Autopsy findings have usually revealed acute fulminating pneumoni pneumonitis, aspiration of gastric contents, or intramyocardial lesions. Although not a ge feature of fluphenazine, potentiation of central nervous system depressants such as opi analgesics, antihistamines, barbiturates, and alcohol may occur.

Systemic lupus erythematosus-like syndrome, hypotension severe enough to cause cardiac arrest, altered electrocardiographic and electroencephalographic tracings, alt cerebrospinal fluid proteins, cerebral edema, asthma, laryngeal edema, and angioneu edema; with long-term use, skin pigmentation and lenticular and corneal opacities have occu with phenothiazines. Local tissue reactions occur only rarely with injections of fluphena decanoate.

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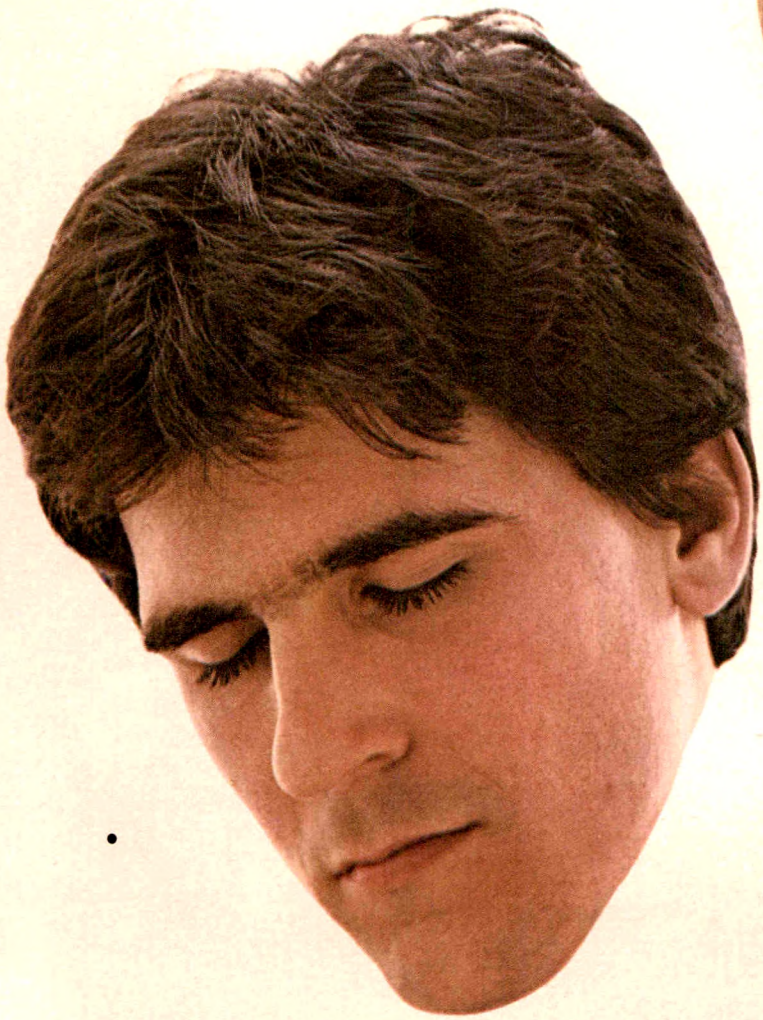
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Help for the apathetic/ withdrawn geriatric*

For the aged person suffering from withdrawn or apathetic senile behavior, Ritalin can often help relieve bothersome symptoms. In one double-blind study[1] of withdrawn, apathetic geriatric patients, Ritalin elicited notable improvement in competence, interest and retardation. While no side effects were reported in this study, blood pressure and pulse variations were noted in both groups but were not considered of exceptional magnitude.





Help for narcoleptic patients

The narcoleptic patient can fall asleep anytime... anyplace... and the consequences can be grave, or at the least, embarrassing. Although there is no known cure for narcolepsy, Ritalin acts promptly to counteract sleepiness and is the medication most frequently used by cited investigators.[2,3] Occasional problems of tolerance may be managed by complete withdrawal of Ritalin during vacation or holidays, then reinstating the drug at lower dosage.[3]

*Ritalin has been evaluated as possibly effective in these indications.

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(methylphenidate)



Help for patients with mild depression*

As Ritalin acts promptly to relieve symptoms of mild depression, mood and outlook usually improve, and the patient can cope again. Generally, one prescription is sufficient. Ritalin is contraindicated in severe depression.

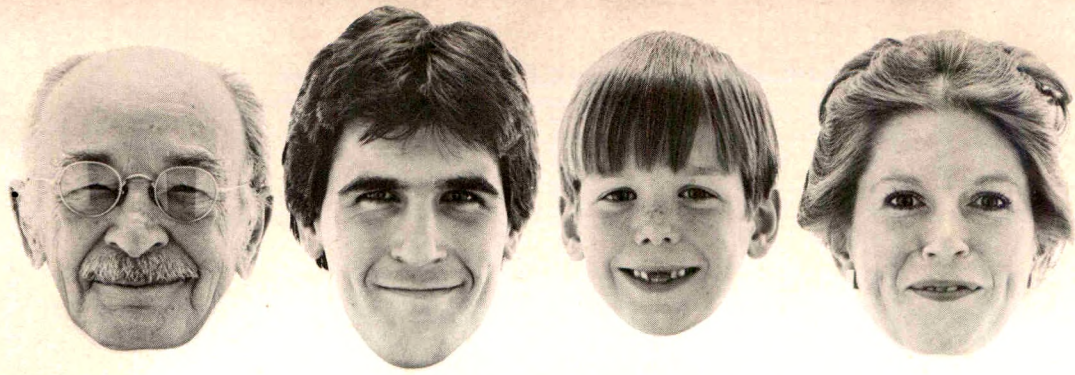


Help for the MBD child

For the child with Minimal Brain Dysfunction (MBD), adjunctive Ritalin is often highly effective when medication is needed to help reduce symptoms such as distractibility,[4] disorganized behavior,[4] and hyperactivity.[4] MBD children responding to Ritalin usually exhibit better classroom performance and improved interpersonal relationships. Moreover, as MBD symptoms are curbed, the affected child often responds more positively to nonpharmacologic modalities.[5] Ritalin should be periodically discontinued to assess the child's condition. Improvement may be sustained when the drug is temporarily or permanently discontinued.

Please turn page for prescribing information.

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RITALIN® C
(methylphenidate)
SO MUCH HELP FOR SO MANY PATIENTS

Ritalin® hydrochloride C
(methylphenidate hydrochloride USP)

TABLETS

INDICATIONS
Based on a review of this drug by the National Academy of Sciences-National Research Council and/or other information, FDA has classified the indications as follows:
Effective: Minimal Brain Dysfunction in Children—as adjunctive therapy to other remedial measures (psychological, educational, social)
Special Diagnostic Considerations
Specific etiology of Minimal Brain Dysfunction (MBD) is unknown, and there is no single diagnostic test. Adequate diagnosis requires the use not only of medical but of special psychological, educational, and social resources.
Characteristics commonly reported include: chronic history of short attention span, distractibility, emotional lability, impulsivity, and moderate to severe hyperactivity; minor neurological signs and abnormal EEG. Learning may or may not be impaired. The diagnosis of MBD must be based upon a complete history and evaluation of the child and not solely on the presence of one or more of these characteristics.
Drug treatment is not indicated for all children with MBD. Stimulants are not intended for use in the child who exhibits symptoms secondary to environmental factors and/or primary psychiatric disorders, including psychosis.
Appropriate educational placement is essential and psychosocial intervention is generally necessary. When remedial measures alone are insufficient, the decision to prescribe stimulant medication will depend upon the physician's assessment of the chronicity and severity of the child's symptoms.
Effective: Narcolepsy
"Possibly" effective: Mild Depression; Apathetic or Withdrawn Senile Behavior
Final classification of the less-than-effective indications requires further investigation.

CONTRAINDICATIONS
Marked anxiety, tension, and agitation are contraindications to Ritalin, since the drug may aggravate these symptoms. Ritalin is contraindicated also in patients known to be hypersensitive to the drug and in patients with glaucoma.

WARNINGS
Ritalin should not be used in children under six years, since safety and efficacy in this age group have not been established.
Sufficient data on safety and efficacy of long-term use of Ritalin in children with minimal brain dysfunction are not yet available. Although a causal relationship has not been established, suppression of growth (*ie*, weight gain, and/or height) has been reported with the long-term use of stimulants in children. Therefore, patients requiring long-term therapy should be carefully monitored.
Ritalin should not be used for severe depression of either exogenous or endogenous origin.
Ritalin should not be used for the prevention or treatment of normal fatigue states.
There is some clinical evidence that Ritalin may lower the convulsive threshold in patients with prior history of seizures, with prior EEG abnormalities in absence of seizures, and, very rarely, in absence of history of seizures and no prior EEG evidence of seizures. Safe concomitant use of anticonvulsants and Ritalin has not been established. In the presence of seizures, the drug should be discontinued.
Use cautiously in patients with hypertension. Blood pressure should be monitored at appropriate intervals in all patients taking Ritalin, especially those with hypertension.
Symptoms of visual disturbances have been encountered in rare cases. Difficulties with accommodation and blurring of vision have been reported.

Drug Interactions
Ritalin may decrease the hypotensive effect of guanethidine. Use cautiously with pressor agents and MAO inhibitors.
Human pharmacologic studies have shown that Ritalin may inhibit the metabolism of coumarin anticoagulants, anticonvulsants (phenobarbital, diphenylhydantoin, primidone), phenylbutazone, and tricyclic antidepressants (imipramine, desipramine). Downward dosage adjustments of these drugs may be required when given concomitantly with Ritalin.

Usage in Pregnancy
Adequate animal reproduction studies to establish safe use of Ritalin during pregnancy have not been conducted. Therefore, until more information is available, Ritalin should not be prescribed for women of childbearing age unless, in the opinion of the physician, the potential benefits outweigh the possible risks.

Drug Dependence
Ritalin should be given cautiously to emotionally unstable patients, such as those with a history of drug dependence or alcoholism, because such patients may increase dosage on their own initiative.
Chronically abusive use can lead to marked tolerance and psychic dependence with varying degrees of abnormal behavior. Frank psychotic episodes can occur, especially with parenteral abuse. Careful supervision is required during drug withdrawal, since severe depression as well as the effects of chronic overactivity can be unmasked. Long-term follow-up may be required because of the patient's basic personality disturbances.

PRECAUTIONS
Patients with an element of agitation may react adversely; discontinue therapy if necessary.
Periodic CBC, differential, and platelet counts are advised during prolonged therapy.

ADVERSE REACTIONS
Nervousness and insomnia are the most common adverse reactions but are usually controlled by reducing dosage and omitting the drug in the afternoon or evening. Other reactions include hypersensitivity (including skin rash, urticaria, fever, arthralgia, exfoliative dermatitis, erythema multiforme with histopathological findings of necrotizing vasculitis, and thrombocytopenic purpura); anorexia; nausea; dizziness; palpitations; headache; dyskinesia; drowsiness; blood pressure and pulse changes, both up and down; tachycardia; angina; cardiac arrhythmia; abdominal pain; weight loss during prolonged therapy. Toxic psychosis has been report
Although a definite causal relationship has not be
established, the following have been reported in
tients taking this drug: leukopenia and/or anemia
few instances of scalp hair loss.
In children, loss of appetite, abdominal pain, wei
loss during prolonged therapy, insomnia, and
tachycardia may occur more frequently; howeve
any of the other adverse reactions listed above r
also occur.

DOSAGE AND ADMINISTRATION
Adults
Administer orally in divided doses 2 or 3 times da
preferably 30 to 45 minutes before meals. Dosag
will depend upon indication and individual response.
Average dosage is 20 to 30 mg daily. Some patie
may require 40 to 60 mg daily. In others, 10 to 15
daily will be adequate. Patients who are unable t
sleep if medication is taken late in the day should
take the last dose before 6 p.m.

Children with Minimal Brain Dysfunction
(6 years and over)
Start with small doses (*eg*, 5 mg before breakfas
and lunch) with gradual increments of 5 to 10 mg
weekly. Daily dosage above 60 mg is not recommended. If improvement is not observed after
appropriate dosage adjustment over a one-mon
period, the drug should be discontinued.
If paradoxical aggravation of symptoms or other
verse effects occur, reduce dosage, or, if neces
discontinue the drug.
Ritalin should be periodically discontinued to as
the child's condition. Improvement may be sustained when the drug is either temporarily or per
nently discontinued.
Drug treatment should not and need not be inde
and usually may be discontinued after puberty.

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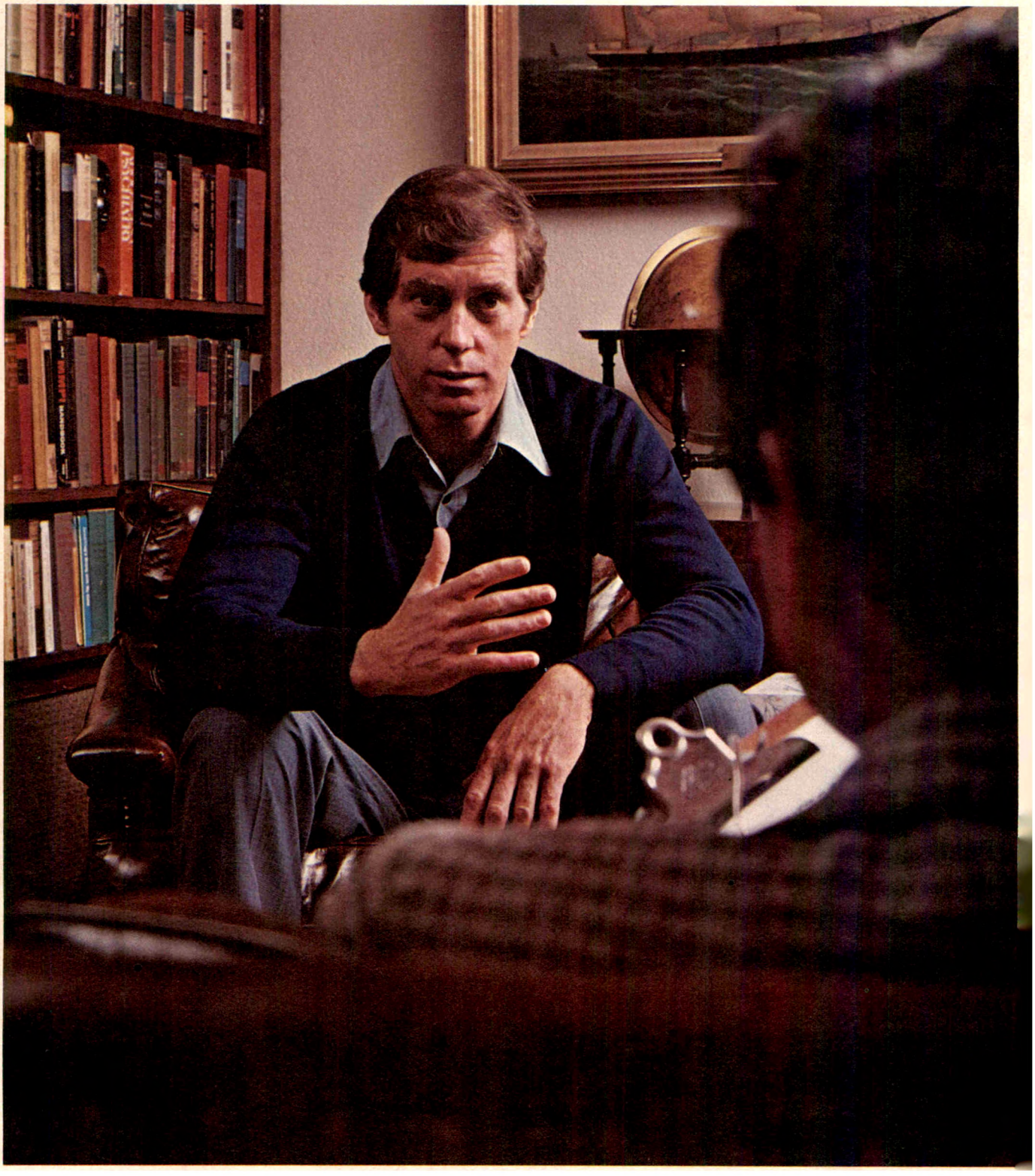
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Contraindications: Known hypersensitivity. Should not be given concomitantly with a monoamine oxidase inhibitor since hyperpyretic crises, severe convulsions, and deaths have occurred. When used to replace a monoamine oxidase inhibitor, allow a minimum of 14 days to elapse before initiating therapy with amitriptyline HCl. Initiate dosage of amitriptyline HCl cautiously with gradual increase in dosage until optimum response is achieved. Not recommended during the acute recovery phase following myocardial infarction.

Warnings: May block the antihypertensive action of guanethidine or similarly acting compounds. Should be used with caution in patients with a history of seizures or a history of urinary retention, or with angle-closure glaucoma or increased intraocular pressure; in patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely; arrhythmias, sinus tachycardia and prolongation of the conduction time have been reported, particularly with high doses; myocardial infarction and stroke have been reported with drugs of this class. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. May impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle. In patients who use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage. Safe use during pregnancy and lactation has not been established; in pregnant patients, nursing mothers, or women who may become pregnant, weigh possible benefits against possible hazards to mother and child. Not recommended for patients under 12 years of age.

Precautions: Schizophrenic patients may develop increased symptoms of psychosis; patients with paranoid symptomatology may have an exaggeration of such symptoms; manic depressive patients may experience a shift to the manic phase. In these circumstances, the dose of amitriptyline HCl may be reduced or a major tranquilizer, such as perphenazine, may be administered concurrently.

When given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required; paralytic ileus may occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs. Use cautiously in patients receiving large doses of ethchlorvynol, since transient delirium has been reported on concurrent administration. May enhance the response to alcohol and the effects of barbiturates and other CNS depressants. The possibility of suicide in depressed patients remains until significant remission occurs. Potentially suicidal patients should not have access to large quantities of this drug. Prescriptions should be written for the smallest amount feasible. Concurrent electroshock therapy may increase the hazards associated with such therapy; such treatment should be limited to patients for whom it is essential. When possible, discontinue the drug several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported. Use with caution in patients with impaired liver function.

Adverse Reactions: *Note:* Included in this listing are a few adverse reactions not reported with this specific drug. However, pharmacological similarities among the tricyclic antidepressant drugs require that each reaction be considered when amitriptyline is administered. *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, increased intraocular pressure, constipation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, leukopenia, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, black tongue, rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache, and malaise; these are not indicative of addiction.

Overdosage: Hospitalize as soon as possible all patients suspected of having taken an overdose. Treatment is symptomatic and supportive. In addition, the intravenous administration of 1 to 3 mg physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage should be repeated as required, particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine.

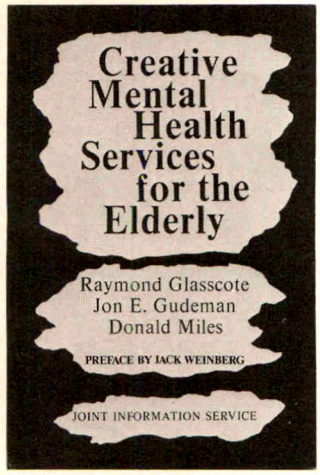
How Supplied: Tablets containing 10 mg and 25 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100, 1000, and 5000; tablets containing 50 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100 and 1000; tablets containing 75 mg and 100 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100; tablets containing 150 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 30 and 100; for intramuscular use, in 10-ml vials containing per ml: 10 mg amitriptyline HCl, 44 mg dextrose, 1.5 mg methylparaben and 0.2 mg propylparaben as preservatives, and water for injection q.s. 1 ml.

For more detailed information, consult your MSD representative or see full prescribing information. Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa. 19486

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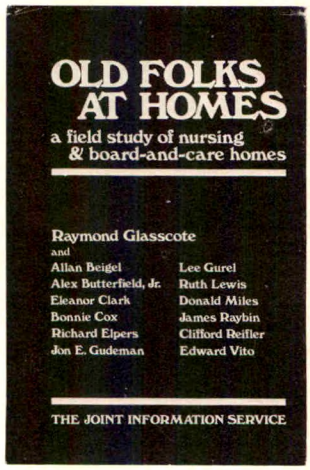
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THE AMERICAN JOURNAL OF PSYCHIATRY

Research on the Psychosocial Treatment of Schizophrenia: A Summary Report

BY LOREN R. MOSHER, M.D., AND SAMUEL J. KEITH, M.D.

The authors present an overview of research on psychosocial treatments for schizophrenia. Findings from studies of five therapeutic approaches—individual psychotherapy, group psychotherapy, family therapy, milieu therapy, and community support systems—are discussed in detail. The usefulness of each type of therapy is critically assessed on the basis of available data from controlled outcome studies. The authors make recommendations regarding high-priority areas to be addressed in future studies of psychosocial treatment.

ALTHOUGH WE have learned a great deal about the design of research on psychosocial treatment, the data now available are sufficiently flawed to lead us to eschew categorical "yes, it works" or "no, it does not work" statements about a given therapeutic approach. The most common failings of the currently available studies are as follows:

1. Insufficient exposure to the particular intervention. The treatment is not given a reasonable chance to demonstrate its ability to produce change. By their very nature, psychosocial treatments cannot be expected to produce rapid, dramatic changes. For example, Hogarty and associates (1) did not see significant beneficial effects from major role therapy until patients had completed 18 months of treatment, and these effects were even more pronounced at 24 months.

2. Failure to define adequately the characteristics of the psychosocial intervention, the therapists, and the patients. Some studies report results from undefined individual therapy delivered by inexperienced therapists, fail to assess the fit between patient and therapist, and make no attempt to study whether therapy was in fact taking place. The mere juxtaposition of patients and therapists in a room for an hour does not ensure that therapy is being given.

3. Use of inappropriate outcome measures. Unfortunately, rehospitalization has retained its preeminent position as the most widely cited outcome variable, despite the fact that myriad non-patient-related factors are known to affect rehospitalization (e.g., administrative policies, availability of nonhospital resources for care, type of living arrangements, family emotional climate). In addition, psychosocial treatments address their major efforts to enhancing social adjustment, which has only a modest relationship to recidivism. Despite this, many psychosocial treatment studies have not assessed systematically the variables that are particularly relevant to this type of treatment, e.g., ability to work and social and interpersonal skills.

Evaluation of research on patients labeled "schizophrenic" is almost always plagued by questions about the type of patients included in this heterogeneous category. Although differences in results might be accounted for by subtle differences in patient characteristics, in most cases only the categories "acute" or "first admission" as contrasted with "chronic" or "long-term" have much influence on results. Insofar as is possible, we will include in this review a description of research samples as acute or chronic and inpatient or outpatient.

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The opinions expressed in this paper are those of the authors and do not necessarily reflect the official position of the National Institute of Mental Health.

INDIVIDUAL PSYCHOTHERAPY

The use of individual therapy with schizophrenic patients grew out of the psychoanalytic movement. Although psychoanalysis had European origins, the work of Meyer (2) and Sullivan (3) in this country provided a fertile ground for its transfer and use with schizophrenic patients here. Interest in individual psychoanalytic therapy with schizophrenics grew dramatically in the 1950s and early 1960s. This burgeoning interest was in part responsible for research conducted to assess the efficacy of such therapy during those years. It should be noted that most of the patients who received this treatment did so in a very small number of private treatment facilities (e.g., Chestnut Lodge and the Menninger Clinic). However, because of the influence of psychoanalysis in university departments of psychiatry and the prolific writings of some analysts (e.g., Arieti and Searles), the impact of the treatment of these relatively few patients was far greater than their numbers.

Controlled Research

Current doubts about the usefulness of individual psychotherapy as a treatment for schizophrenic patients stem from the negative results of controlled outcome studies in the 1960s, the development of clearly effective drug treatment, and the waning influence of psychoanalysis in departments of psychiatry. In the 1960s, there were four major studies in which schizophrenic inpatients treated with psychotherapy were compared to those who received other forms of treatment. The sample selection and individual psychotherapy techniques are summarized below:

1. Grinspoon and associates (4) studied single, male, chronic schizophrenics who had been hospitalized for more than 3 years in a large state hospital and treated with an analytically oriented approach by experienced therapists.
2. May (5) reported on first-admission "middle prognosis" schizophrenics treated with an ego-supportive approach, primarily by state hospital residents.
3. Karon and Vandenbos (6) studied acute, lower class, inner-city first-admission schizophrenics who were given either direct analysis or ego analysis by both experienced and inexperienced psychologists.
4. Rogers and associates (7) studied a mixed group of acute and chronic state hospital schizophrenics treated with client-centered therapy, primarily by experienced psychologists.

Several extensive critical reviews of these studies are available (8–10).

The fall from favor of individual psychotherapy as a treatment for schizophrenia is dramatically illustrated by the following: when one of the four studies was being designed in 1960, it was regarded as unethical for a control group not to receive psychotherapy, but by 1968 it was considered unethical to give a control group in another study psychotherapy alone.

Of the four controlled psychotherapy studies conducted during this period, those by Grinspoon and associates (4) and May (5), both of which had largely negative results, became the most widely known and influential. The two other studies (6, 7) had more positive results. Each of the four studies, viewed in retrospect, had critical design problems that led many scientists to warn against the premature conclusion that individual psychotherapy is of no value with schizophrenics. For example, Grinspoon and associates (4) focused on a small group of very chronic "back ward" patients who were transferred from a large state hospital into a highly staffed, nurturing, but undemanding milieu. We now know that for the chronic patient the dependency-reinforcing effects of such a milieu are likely to outweigh any possible benefit derived from 4 or 5 hours of individual therapy, even when it is provided by highly experienced therapists such as those in the Grinspoon and associates study.

The influential study by May (5) had two major flaws as a test of individual therapy: inexperienced state hospital residents served as therapists, and the subjects (middle-prognosis first-admission schizophrenic patients) received relatively few hours of therapy (an average of 46). It should be remembered that despite the prevailing negative zeitgeist about individual psychotherapy for inpatient schizophrenics, Karon and Vandenbos (6) reported positive results from their controlled study and Rogers and associates (7) obtained equivocally positive results. However, the more positive studies also had enough design problems to make it unwise to draw firm conclusions at this time about the value of individual psychotherapy for inpatient schizophrenics.

Fortunately, there is now a collaborative study under way at McLean Hospital and Boston University, under the direction of Alfred Stanton and Peter Knapp, that addresses may of the problems of previous research on inpatients treated by intensive psychotherapy. The patients are relatively acute, the therapists are experienced, the treatment will continue over a prolonged period and into the outpatient phase (a year or more), the outcome measures are appropriate to this treatment, whether therapy is in fact going on will be assessed, and the therapists and their fit with the patients will be studied.

Since the publication of the four inpatient studies in the 1960s, four posthospitalization comparisons of group and individual therapy with heterogeneous populations of schizophrenic patients have been published (11–14). In terms of readmission, three showed individual therapy and group therapy to be similar and one (14) found an advantage for group therapy. The two studies that measured social adjustment found opposite results, one in favor of group therapy (13) and the other in favor of individual therapy (12).

In general, these studies also suffer from design problems, especially with regard to control of neuroleptic drugs, assessments of whether therapy was actually taking place, and use of appropriate outcome

measures. However, the data seem to indicate that group therapy has some advantages, in terms of patient and therapist satisfaction, over individual therapy for the more chronic patients. There are no data on strictly acute patients treated with individual therapy after discharge. Nor are there data on whether group and individual therapy might be synergistic for some patients. Both of these deficiencies should be remedied by a new generation of studies.

GROUP PSYCHOTHERAPY

As far as its history is determinable, group psychotherapy appears to have grown out of the observation that patients with similar medical or psychological problems can receive benefit from each other as well as from the doctor. From its origins in lectures for mental reeducation of hospitalized patients (15, 16), group treatment of schizophrenia has diversified into many forms, including supportive therapy, psychodynamic therapy, and activity groups. Neither the early didactic techniques designed to instill understanding of the psychological mechanisms underlying behavior through classroom presentations and lectures (17–19) nor the strictly psychoanalytic approaches introduced during the same period (20–23) remain in wide use with schizophrenic patients today.

Although the primary aim in most traditional group therapies is facilitation of insight into personal and interpersonal problems, much group work with schizophrenics is oriented toward providing support, an environment in which patients can develop social skills, or a format that allows friendships to begin and be sustained.

There are many reviews of the group therapy outcome literature extending up to 1968 (24–27). Considering the data available to them, these reviewers generally concluded that most controlled evaluations of group therapy with schizophrenics (especially long-term inpatients) showed meager, if any, therapeutic benefit. There were individual instances of special benefits from group treatment, but design problems, such as lack of follow-up, inappropriate control groups, and insufficient duration of treatment, made it difficult to draw firm conclusions. This review will focus on the 1966–1976 decade of research.

Comparison with the Usual Hospital Treatment Program

Hospital settings almost always provide a wide range of treatment modalities, e.g., recreational and occupational therapy, music therapy, and medication. Therefore, group psychotherapy offered in inpatient settings is an "add-on" rather than an exclusive treatment form. In each of six studies of group therapy versus no group therapy reviewed by Parloff and Dies (28), patients selected for their predicted suitability for group therapy were randomly assigned to either group therapy or a no-group-therapy hospital treatment control group. Unfortunately, as is so often the case, diverse measures of outcome were used, with little overlap between studies, making direct comparisons difficult.

Of six comparisons of group therapy of schizophrenics against a no-group-therapy hospital condition, four failed to reveal any unique or impressive contribution assignable to group psychotherapy (28). It should be noted, however, that the studies with positive results (29, 30) were better designed in terms of length of treatment and outcome measures that addressed the investigators' hypotheses directly.

Group Therapy Combined with Other Psychosocial Interventions

Five of the six studies that tested group therapy in combination with another treatment approach reported that the combination—be it neuroleptic drugs, videotape feedback, ward activity, or dyadic social interaction—was superior to the treatment forms offered singly (28).

Integrating the results of these two types of in-hospital studies is difficult for a number of reasons: the patient groups are often not comparable, the outcome measures vary widely and frequently do not address the specific effects sought by the treatment, and many of the studies involve short treatment periods. This problem is especially relevant when the subjects are long-stay patients (as was the case in all but two of the studies). However, positive results were found by those investigators who carefully defined the behaviors or attitudes they wished to change and used outcome measures that tapped them. In addition, it appears that the more structured the activity—Olson and Greenberg's incentive therapy (29), Corder and associates' required dyadic interaction (31), and Robinson's videotape feedback (32)—the more likely a positive result (33). Although this interpretation is somewhat speculative, it is consistent with the types of milieu therapy found to be most useful for chronic patients.

Aftercare Treatment Studies

The treatment of schizophrenics has been revolutionized during the past 15 years. Extended hospitalization has given way to early release, and treatment is increasingly provided in general hospitals and outpatient settings rather than in large mental hospitals. Patients are discharged into the community, to their families, to halfway houses, or to foster homes with greater frequency. As this emphasis on community treatment has grown, it has become apparent that psychosocial interventions are required to aid the patient in maintaining community adjustment—drugs do not teach social and interpersonal skills. Group psychotherapy has been used as a treatment modality aimed at maintaining the discharged patient in the community by enhancing social and interpersonal skills.

During the past decade six major studies (11–14, 33, 34) have tested the relative efficacy of some form of group therapy contrasted with an alternative treatment

or a no-therapy control group in posthospitalization patients.

These recent studies of group psychotherapy given to heterogeneous groups of posthospital schizophrenics have not yielded strong or consistent evidence that this form of treatment reduced rates of rehospitalization, improved vocational adjustment, diminished psychopathology, or enhanced social effectiveness when compared with individual therapy or no-group-therapy control groups. However, because the evidence on each of these variables is contradictory and the therapy was not well defined, it cannot be concluded that outpatient group therapy is not useful for schizophrenics. In fact, a treatment whose principal aim is to promote socialization and enhance interpersonal skills probably should not be expected to have a striking effect on rehospitalization or vocational adjustment. Two of the three studies that addressed the socialization and interpersonal skills outcome variables (13, 34) showed a positive effect from group treatment. The negative findings of Levene and associates (12) were based on relatives' ratings, whereas the positive findings of the other two studies (13, 34) came from research raters and the patients themselves. This raises a methodologic issue that was also present in the inpatient group therapy studies, i.e., the influence of the rater on the assessment of outcome. It is also noteworthy that O'Brien and associates (13) and Herz and associates (11) reported that both group patients and group therapists retrospectively reported more favorable feelings about the treatment experience than did individually treated patients and their therapists. These morale-enhancing, affiliative effects are probably very important and certainly need further research, because they are particularly difficult issues with long-term, community-based patients.

What is badly needed now are comparative studies in which group or individual therapy is used to treat relatively acute patients; treatment should be started after the patients' psychoses have subsided but while they are still in residential care, and it should be continued into their posthospital period. The design should include patients maintained on and patients withdrawn from neuroleptic drugs. In addition, the therapy should be for a prolonged period (at least 6 months), the outcome measures should be focused primarily on social and interpersonal variables rather than rehospitalization rates, and a 2-year follow-up assessment period should be required. Finally, the samples should be sufficiently large to allow subgroup analysis in order to distinguish the types of patients who respond especially well, or poorly, to each form of treatment. The last condition will probably necessitate large-scale collaborative research.

FAMILY THERAPY

Although family therapy has been in widespread use for more than 15 years, there have been only three controlled comparative studies of its effectiveness with schizophrenics (35–38). All of these studies were conducted with relatively acute patients and the therapy was short-term (6–10 sessions). Ro-Trock and associates (38) compared 10 sessions of inpatient family therapy (focused on promoting clear communication and shifting the balance of maladaptive relationships within the family so that new, more adaptive forms of relating would be possible) and individual therapy (focused on helping the patient develop solutions and problem solving skills in dealing with life conflicts). Langsley and associates (37) compared hospitalization and "usual" treatment with family therapy aimed at quickly reducing the level of tension so that family resources could be garnered to cope more effectively in the crisis situation. Goldstein and associates (35, 36) compared 6 sessions of family therapy begun immediately after discharge with usual immediate post-discharge treatment, including maintenance neuroleptic drugs. The family therapy had four major goals: 1) promoting acceptance of the occurrence of the psychosis, 2) identifying precipitating stresses, 3) looking at possible future similar stressful events, and 4) developing strategies for avoiding, minimizing, and coping with these stresses.

Each study found family therapy to have advantages over the treatment with which it was being compared. Langsley and associates (37) concluded that family crisis intervention could be effective in preventing hospitalization for schizophrenics, but at 18-month follow-up, the patients' crisis management skills had not been significantly enhanced by the treatment.

Ro-Trock and associates (38) found that family therapy was associated with fewer readmissions and more rapid return to functioning in the community than individual therapy. They could not, however, identify any clear and consistent differences between the two treatments on their interaction and self-report measures. Goldstein and associates (36) found significant differences favoring family therapy at the end of the treatment in terms of reduced symptomatology and enhanced psychosocial adjustment. However, at 6-month follow-up only the "good premorbid" subsample of the family therapy subjects was functioning significantly better than the control subjects.

In addition to these controlled studies, there are a number of uncontrolled studies of longer term family therapy that included a substantial percentage of chronic patients (39-42). In each instance the investigators concluded that family therapy had resulted in greater improvement than would have been expected in its absence.

Given the lack of clearly negative results from family therapy, it is difficult to understand why so few studies of this type of treatment are available. Because family therapy is in widespread use in both inpatient and outpatient settings, research on its efficacy should be stimulated and supported. In particular, this research should compare longer-term family therapy (6–12 months) with another psychosocial treatment; the

sophisticated approach of Goldstein and associates (36) to the neuroleptic drug variable and subgroup analysis should be combined with the family interaction methodology of Ro-Trock and associates (38). In addition, the design should include 1- to 2-year posttreatment assessment of community adjustment and family interaction. Because of the origins and theoretical underpinnings of the family approach, this research might best be focused primarily on late adolescent and early adult schizophrenics living with their families of origin.

MILIEU TREATMENT

Over the past decade, marked progress has been made in defining, clarifying, and doing research on what constitutes a therapeutic milieu (43). Specific milieu typologies have been developed from new instruments designed to assess milieu characteristics systematically (44). The differing characteristics of inpatient milieus most useful for acute as compared with chronic patients have been described clinically. Acute patients have been found to recover best in highly staffed, accepting, supportive, stimulus-decreasing (at least early in treatment), relatively long-stay (3–5 month) environments in which psychosocial interventions are viewed positively (45–47).

In addition, four milieu characteristics associated with better posthospitalization psychosocial adjustment among relatively acute patients have been identified through the use of recently developed systematic assessment instruments (44, 48): 1) small size, 2) positive staff expectations, 3) line staff's active involvement and investment in the treatment process and decision-making (resulting in high morale), and 4) a practical, down-to-earth, problem-solving orientation (49–52). Intriguingly, these important milieu characteristics have been demonstrated only recently, after a long history of research that found little relationship between in-hospital milieu characteristics and posthospitalization adjustment among acute patients. Although derived completely independently, these characteristics are quite consistent with the clinical analysis of "good" milieus for acute patients described above.

Three recent clinical studies are noteworthy in having demonstrated that outcome (in terms of rehospitalization and posthospitalization social adjustment) was as good (45) or better (47, 53) in unmedicated patients as in patients treated with neuroleptic drugs. These findings are contrary to previous reports (8) in which milieu without neuroleptic drug treatment was reported to be relatively ineffective with acute patients. In effecting change without using neuroleptics, these newer studies would seem to have provided a specifically therapeutic milieu. This research is also important because of the emerging evidence of severe long-term toxicities of neuroleptics (e.g., tardive dyskinesia, breast cancer). That is, it is now possible to design milieu programs in which neuroleptic drug use can be minimized. However, a great deal of research is necessary before the fits between patient and milieu characteristics and need for drug treatment can be defined precisely. Given the emerging picture of neuroleptic toxicities, these studies warrant immediate stimulation and support.

In contrast to the clinical descriptions of good milieus for acute patients, chronic patients seem to respond best to a very structured, highly organized, high-expectation milieu oriented toward specific behavior change (54). In addition, chronic patients do best when the changes sought and the procedures for bringing them about are very clearly and specifically defined. There is also evidence that neuroleptic drug treatment interferes with the ability of some very chronic patients to learn new, more adaptive, socially appropriate behaviors in task-focused milieu programs (55).

For chronic patients, posthospital adjustment seems to be related to the degree to which behaviorally focused inpatient milieus have taught them adaptive behaviors and skills and have suppressed symptomatic and maladaptive behaviors. In addition, however, it appears important that the in-hospital program be continued for some time in the community and that a variety of supportive arrangements be available indefinitely (55, 56). Given a behavior-therapy-oriented program with continued aftercare and an adequate community residential program, more than 90% of very chronic patients can be discharged into the community, with a 2-year rehospitalization rate of less than 5% (54). Although only 10% of these patients will be able to live completely independently, they are able to function well enough to fulfill their basic needs for food, shelter, socialization, and recreation.

The recent findings in milieu research are striking in their consistency, replicability, and clinical relevance. Moreover, they can be implemented, with appropriate modifications, in settings where acute, chronic, or both types of patients are treated.

REHABILITATION

Psychosocial rehabilitation services, which began when a group of former state mental hospital patients banded together in New York in the late 1940s, have come to represent a major force in responding to the needs of the chronic schizophrenic population. The active collaboration of patients in the programs has been maintained, and the services have developed with professional leadership. The general goal is reintegration of the psychiatrically disabled into the community by maintaining and augmenting whatever level of functional independence a patient has been able to achieve. In general, rehabilitation has focused on programs to develop social support and independent living capabilities, emphasizing vocational components.

Results of research on the chronic patient popu-

lation have underlined the need for rehabilitation programs for this high-recidivism and low-employment group. An extensive review of research findings by Anthony and associates (57) showed baseline readmission figures of 30%–40% after 6 months, 35%–50% after 1 year, and 60%–75% after 3–5 years. In terms of employment, most studies indicate a 10%–30% rate of independent employment at follow-up, regardless of the time period studied. Unfortunately, little controlled comparative research has been carried out on the impact of comprehensive psychosocial rehabilitation centers on recidivism and employment.

Beard and associates (58) have recently reported on a 5-year follow-up study which showed that with an active outreach program for the initial 2 years of participation, Fountain House clients had significantly lower rehospitalization rates at 1, 2, and 5 years than control subjects. Those who were rehospitalized spent 40% fewer days in the hospital than rehospitalized control subjects. Their data also indicate that the more contact patients had with the program, the less likely they were to be rehospitalized.

Other aspects of comprehensive rehabilitation programs that have received research attention are aftercare clinics and transitional living arrangements. Aftercare clinics, which usually include some form of therapeutic or casework contact in addition to medication, have been shown to reduce readmission rates (59). Evidence indicates that this effect results from a combination of factors—drugs, additional services, and the type of patients who attend. Hogarty and associates (60) reported that patients who received major role therapy (casework with an experienced social worker) plus drugs had significantly better social adjustment than those who received drugs alone. This effect first appeared at 18 months and was somewhat stronger at 24 months. An interesting patient variable, chronicity (a composite rating based on the number of previous hospitalizations, length of last hospitalization, psychotic versus nonpsychotic diagnoses, and previous employment status), has also been shown to predict outcome (61). Kirk (61) reported that the recidivism rate of aftercare clinic attenders who had a high level of chronicity decreased as the number of visits increased to above six. In contrast, the recidivism rate of aftercare clinic attenders who had a low level of chronicity was not affected by an increased number of visits. Based on the results of this and another recent study (62), as well as the Fountain House results, it is tempting to conclude that aftercare treatment is most effective with those chronic patients who maintain a continuing relationship with the program.

Transitional facilities (halfway houses, day centers) are a major component of psychosocial rehabilitation and have been shown to be effective in reducing recidivism as long as the patients remain in contact with the facility (59). In their review, Rog and Raush (63) found data to suggest lower recidivism rates for residents of halfway houses, although they noted the almost complete absence of controlled studies. Successful programs have also been developed using a transitional person rather than a transitional facility to reduce readmission (64–66).

The field of psychosocial rehabilitation is experiencing a rapid expansion of its activities, as indicated by the establishment of the International Association of Psychosocial Rehabilitative Services and NIMH's new Community Support Program (a major component of which is psychosocial rehabilitative services). High priority should be given in this field to studies obtaining basic recidivism and employment data from several centers in an attempt to replicate the Fountain House results. Following this initial research effort more complex studies of patients' skill gains, quality of life, and satisfaction with services, as well as patient/program "fit" and patient/society benefits, should be implemented.

COMMUNITY SUPPORT SYSTEMS

The past decade has seen a gradual emergence of interest in a variety of community-based resources that, taken together, provide important support to schizophrenic patients. At least 10 different potential elements of community support programs have been identified (67). These resources include cooperative apartments, halfway houses, and psychosocial rehabilitation centers such as Fountain House and Thresholds (68). Such community support systems are in part a response to the progressive nuclearization of the family and are intended to provide the support that has been lost because of the dissolution of extended kinship networks or discharge from the hospital.

Unfortunately, community resources are still relatively scarce, they can accommodate only a small portion of potential clients, and they remain largely isolated and little known because most result from the efforts of one or two concerned, enthusiastic persons. Generally, they are a response of the private sector to failures of the medical and social welfare systems to provide adequately for the needs of patients. However, NIMH has recently funded 18 pilot demonstration community support programs.

The efficacy of these programs, beyond their humanitarian importance, remains largely untested. Although the efficacy of particular community supports (e.g., psychosocial rehabilitation centers) is not well established, there are data that suggest community support systems are effective and will therefore receive even greater emphasis in the next decade. Data from the International Pilot Study of Schizophrenia (69) are relevant to the issue of community support. This careful longitudinal research involved more than 1,000 patients in eight countries on admission to a hospital and 2 and 5 years later. They found, to everyone's surprise, that the outcomes of schizophrenic patients in developing countries (Nigeria, India, Colombia) were significantly better than those of patients in developed countries (United States, Russia, England,

Denmark, Czechoslovakia). Although this finding is open to a variety of interpretations, an explanation positing the presence of supportive extended kinship networks is consistent with the family organizations in these developing countries. A related explanation—that these relatively uncomplicated societies are less stressful to individuals—also fits the community support model. It has been shown for medical illness (70, 71) that social support protects against the development of illness and aids recovery when illness is related to life stress. Thus, one parsimonious explanation is that in developing countries, for whatever levels of stress occur there is greater protection available to individuals from their naturally occurring social support systems. This case is even more persuasive when taken in conjunction with two bodies of data from the United Kingdom which indicate that 1) schizophrenics experience greater life stress than others in the 3-week period before the onset of overt symptomatology (72), and 2) having more than 35 hours a week of face-to-face contact with hostile, critical, overinvolved parents or spouses is highly predictive of relapse among discharged schizophrenics, even when they are maintained on neuroleptic drugs (73).

In sum, there is a substantial body of data derived from several different perspectives indicating that social support systems, especially relationships outside the nuclear family unit, can serve as protective buffers for individuals exposed to stresses that can result in the development or exacerbation of schizophrenia.

Although they have not generally been conceptualized as community support systems, a variety of alternatives to hospitalization have been studied; these can be seen as providing support systems for individuals undergoing the severe personal distress and crisis that comes to be labeled schizophrenia. Stein and Test's training in community living (74), Langsley and associates' family crisis intervention (37), Pasamanick and associates' visiting nurse program (75), Sanders and associates' enablers program (76), Polak and Kirby's family foster care (77), Fairweather and associates' lodges (56), Mosher and Menn's Soteria program (78), and day-care centers (79, 80) are examples of the intentional provision of a support program in lieu of the support inherent in hospital care. The results of these divergent programs, used with a wide variety of patients, have been similar. Each study found that the vast majority of acutely disturbed schizophrenics, who would otherwise have been hospitalized, can be treated effectively and at lower cost in nonhospital settings if sufficient interpersonal support and protection are made available to them and to their caretakers (33).

What remains to be learned from these studies is a more refined definition of the specific and nonspecific ingredients in each program that result in optimal recovery. However, the uniformity of available results seems to warrant the more widespread implementation of such programs—especially in view of rapidly escalating hospital costs. Yet, with the exception of Fairweather and associates' lodges (56), this has not happened. The barriers to wider use are both conceptual, in that treating severely disturbed persons outside of hospitals is contrary to long-standing practice, and fiscal, in that third-party payers, including Medicaid, as a rule will not reimburse for nonhospital residential care. Thus, in addition to the further research needed, there is also a need for incentives to provide cost-effective alternatives to hospitalization and fiscal mechanisms that will support such alternatives adequately.

CONCLUSIONS

Perhaps the most important statement that can be made on the basis of this review of recent research on psychosocial treatments for schizophrenia is that we know more about their effectiveness than is generally acknowledged. Although critical, experimentally oriented researchers might disagree with this conclusion on methodologic grounds, we believe the consistency of the data about the effectiveness of psychosocial treatments supports this interpretation. It is noteworthy that the positive findings from controlled studies are most consistent for those treatments that involve extensive attention to the individual patient's social environment (i.e., non-hospital-based family therapy and various residential milieu therapies, including alternatives to hospitalization).

If these findings are considered in conjunction with the less methodologically rigorous evidence for the positive effects of support systems on outcome, it is tempting to conclude that the most effective types of psychosocial treatments for schizophrenia are those that provide the most comprehensive, corrective sustaining social support systems.

Although a great deal of research on these types of treatments is recommended, it does seem that based on this review, family therapy and a particular type of milieu treatment can be recommended for newly diagnosed schizophrenics. In addition, the more widespread implementation of various alternatives to hospitalization is supported by the available evidence. Finally, a specific behavior-change-oriented milieu approach to very chronic patients seems warranted by the studies we have reviewed.

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Awareness and Disengagement in Cancer Patients

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The authors found that cancer patients (N=44) did not differ from patients with other chronic illnesses (N=27) with respect to awareness of their condition or ratings on an unobtrusive, nonverbal measure of disengagement. Cancer patients who were aware were more engaged than those who were not, but this was equally true for the noncancer patients. Awareness, for the cancer patients, was not associated with frequency of visiting or with living for a longer or shorter period than expected. There was a significant interaction, however, in that patients who were aware lived longer if they were engaged, whereas patients who were unaware lived longer if they were disengaged. Caution and judgment should be exercised in sharing information with terminally ill patients because awareness may be beneficial for some but not for all.

IN RECENT DECADES, many have come to believe that access to the information that one has a terminal condition, which approximates knowledge of one's diagnosis in people with cancer (1, 2), facilitates an acceptance of dying, improves the quality of life that still remains, and leads to a more peaceful and dignified death (1-8). Others believe that denial of one's condition, including denial of apparent physical deterioration, preserves hope and life. They suggest than an inability to maintain denial results in hopelessness, depression, and premature mortality (9-15).

Lacking a body of relevant experimental data, we are faced with a conflict in values. Is there or should there be a right to the truth about one's impending death? Is there or should there also be a right for an individual to refuse consciously or unconsciously to face such information or to avoid the question entirely? Should one provide information that has not been requested? Should one tell the truth if the truth may be harmful? Physicians must make these decisions daily. The immediate question is whether sharing such information will prove beneficial to patients, and in this regard there are few reliable guidelines to help physicians. Merely preserving life is no longer considered a satisfactory and sufficient criterion, and variables such as hope, the will to live, and the quality of life have not been readily amenable to objective investigation. Nevertheless, in a previous paper, we suggested that meaningful and measurable criteria could be developed to evaluate the consequences of awareness, such as the maintenance of communication channels, degree of disengagement, and actual length of life compared with expected length of life (16).

A continuing involvement in the world of people and things would appear to represent a more desirable attitude toward living than depression, "turning to the wall," and disengagement. Responding to external stimuli, retaining an interest in one's possessions and appearance, and keeping in touch with events are observable and quantifiable behaviors. An index of disengagement made up of such items would seem to be related to important aspects of the quality of life. Maintaining communications with family and friends is also generally considered to be desirable and beneficial. Although there are differing opinions about whether awareness or lack of awareness is more likely to encourage visiting, visiting patterns are observable, and few would disagree that visiting provides the patient with support and helps to preserve hope. Finally, there are many reports about patients who have given up and died sooner than expected or fought and lived longer than expected. To the extent that one's motivation and attitudes contribute to survival, an index of living longer than medically expected would seem to reflect not only extended existence but also hope and a will to continue living.

The main purpose of the present study was to compare cancer patients who were aware of their condition with those who were not with respect to 1) disengagement, 2) visiting patterns, and 3) living for a longer or shorter period than expected. Another purpose was to compare cancer and noncancer patients with respect to awareness and disengagement. A critical constraint on the design of the study was the neces-

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sity to develop instruments and methods that were not intrusive so that the procedures would not alter the patients' state of awareness.

METHOD

Subjects

Two general medical floors serving patients of similar socioeconomic status were selected for the study. The majority of patients on these floors had diagnoses of cancer or of cardiovascular, renal, or neurologic disease. At the time our observations were made, there were 44 patients with malignancies and 27 with other diseases who met our criteria of being between the ages of 45 and 75, lucid, mobile, and able to move the head and all extremities. There was no significant difference in age between the cancer and noncancer patients.

Indices

Awareness. Judgments regarding awareness of diagnosis were categorized as follows:

1. Both patient and family know the diagnosis.
2. The patient but not the family knows the diagnosis.
3. The family but not the patient knows the diagnosis.
4. Neither the family nor the patient knows the diagnosis.

Although we could not question patients or family members without affecting their awareness, information could be obtained from attending physicians, house staff officers, or nurses. While physicians know what they have told the patient and/or the family, they might not know what has been retained or what the patient and family have told each other. House staff officers should be able to assess the "working knowledge" of the patient but often have little contact with the family. We expected the floor nurses, who spend more time with both patient and family, to provide the most useful judgments. In a pilot study, 41 patients were judged as to their state of awareness by their attending physicians, house staff officer, and two nurses. The judgments of the nurses did not differ significantly from those of the attending physicians or house staff officers, but the attending physicians and house staff did differ significantly from each other ($\chi^2=6.01$, $p<.05$). Furthermore, the judgments of the two nurses who had rated each of the 41 patients were highly consistent (90% agreement). In the current study, therefore, nurses' judgments were used to categorize the patients' state of awareness.

Actual/expected survival time. In "End Results in Cancer" (17), life expectancies after diagnosis are provided with reference to the patient's age and the type and site of the malignancy. The patients were compared in terms of actual versus expected length of life after diagnosis.

Disengagement. The Nonverbal Disengagement Index (appendix 1) was developed to assess an individual's involvement in the world of people and things without the need for interview data. The index includes the following 10 items, which are rated on 5-point scales from engagement to disengagement after a brief observation of the patient: orientation, posture, grooming, dress, personal effects, reaction 1, reaction 2, modesty, expression, and activity.

The reliability of the instrument was evaluated in several ways. In one pilot study, 15 observations were made of each of 63 patients over a 5-day period. Stability over observations was investigated by comparing scores obtained during even-numbered observations with those obtained during odd-numbered observation periods. The correlation was significant ($r=.69$, $p<.01$). Internal consistency was evaluated by comparing scores on each item with the total scores of the other 9 items. The correlations ranged from .34 to .74 (average=.52) and were all significant ($p<.01$). In addition, interrater agreement was evaluated in another pilot study in which two observers independently rated a sample of 40 patients. The ratings of the two observers did not differ in mean scores and were highly correlated ($r=.88$, $p<.01$).

Visiting. During a trial period it was found that nurses' judgments were consistent in categorizing visiting frequency as either none, rare, few, or many. Furthermore, the nurses appeared to have no difficulty in responding to the question.

Procedure

Observations for the Nonverbal Disengagement Index were made by one of the authors (W.C.M.) in the course of changing water carafes at the usual times: morning, afternoon, and evening. The procedure took approximately 30 seconds and scores were recorded on the answer sheets after the observer left the patient's room. Scoring required approximately 10 seconds and was unobtrusive because the large cart used to transport the carafes held much other paraphernalia. Three observations were made of each patient on 5 consecutive days, for a total of 15 observations. Patients who were not present for at least 8 observations (e.g., were out of the room for X rays or were discharged during the study) were not included in the analyses. Individual scores were averaged and converted to a distribution with a mean of 100 and a standard deviation of 10. The range of scores was from 80.7 to 127.6, and people with scores over 100 were considered engaged. For this group, disengagement scores were not significantly related to the ingestion of tranquilizers, antidepressants, sedatives, or analgesics.

Following the observation period, awareness and visiting ratings were obtained from the charge nurses. Information about the type and site of malignancy and the date of diagnosis was obtained from the charts or by contacting the patients' attending physicians.

TABLE 1
State of Awareness of Patients with Malignant and Nonmalignant Conditions

Awareness Category	Malignancy (N=44)		No Malignancy (N=27)	
	N	%	N	%
1 (patient and family)	27	61.4	20	74.1
2 (patient only)	0		0	
3 (family only)	12	27.3	2	7.4
4 (neither)	5	11.4	5	18.5

TABLE 2
Awareness and Disengagement of Patients with Malignant and Nonmalignant Conditions

State	Malignancy		No Malignancy	
	N	%	N	%
Aware				
Engaged	17	63	14	70
Disengaged	10	37	6	30
Unaware				
Engaged	5	29	2	29
Disengaged	12	71	5	71

TABLE 3
Relationship of Visit Frequency to Awareness and Disengagement in Cancer Patients

Visitors	Aware		Unaware	
	Engaged	Disengaged	Engaged	Disengaged
Rare or none	1	6	1	3
Few	5	0	1	6
Many	4	1	2	0
Total	10	7	4	9

TABLE 4
Relationship of Awareness and Disengagement to Survival Time After Diagnosis

State	Lived Longer		Died Sooner	
	N	%	N	%
Aware				
Engaged	7	85.7	10	52.6
Disengaged	1	14.3	9	47.4
Unaware				
Engaged	1	12.5	4	50.0
Disengaged	8	87.5	4	50.0

RESULTS

The numbers of patients with malignant and nonmalignant conditions who were rated as falling into the four categories of awareness are given in table 1. In most instances, both patients and families were aware of the diagnosis. There were no cases in which the patient was aware and the family was not. Subsequent analyses were based on patients' awareness (categories 1 and 2) or unawareness (categories 3 and 4). While cancer patients tended to be more highly represented than noncancer patients in category 3 (family aware and patient unaware), the overall distributions by state of awareness for patients with malignant and nonmalignant conditions were not significantly different. Similarly, there was no significant difference between the proportions of cancer and noncancer patients who were disengaged (50% and 41%, respectively, see table 2).

A significant difference was found when individuals with malignant and nonmalignant conditions were compared with respect to both awareness and engagement ($\chi^2=9.40$, $p<.05$). As indicated in table 2, awareness was associated with engagement in both cancer and noncancer patients, but there was little difference in the extent of this relationship attributable to diagnosis.

Data on frequency of visiting by families and friends were available for only 30 of the patients with malignant conditions because this variable was introduced later in the study (see table 3). Visiting frequency was not found to be associated with state of awareness but was associated with engagement ($\chi^2=7.75$, $p<.05$). Engaged individuals were visited more frequently than those who were disengaged.

The patients with malignancies were followed by examining the records or contacting their attending physicians for 3 years, at which time 40 of the 44 had died. Seventeen had lived longer than expected and 27 died sooner than expected. The group lived an average of 1.2 years less than expected. This is probably attributable to 1) four patients who were still living and could contribute to increasing the average, and 2) the sample being composed of patients who were already in trouble and had been referred to a university hospital. The actual length of life after diagnosis correlated significantly with the actuarial expectancies ($r=.38$, $p<.05$). Of the 27 patients who were aware of their diagnosis, 19 (70.4%) died sooner than expected as compared to 8 of the 17 (47.1%) who were unaware (table 4), but this difference was not statistically significant. Similarly, 14 of the 22 patients (63.6%) who were engaged died sooner than expected as compared to 13 of the 22 (59.1%) who were disengaged, a nonsignificant difference.

A significant relationship was found when patients who lived longer or died sooner than expected were compared with respect to both awareness and disengagement ($\chi^2=9.74$, $p<.05$). The major contribution to this finding apparently was made by the interaction of awareness and disengagement in the "lived longer" category. Patients who were aware lived longer if they were engaged, while those who were unaware lived longer if disengaged. Interestingly, dying sooner than expected was not significantly associated with any of the combinations of awareness and engagement.

DISCUSSION

While there are strong opinions about the effects of telling terminally ill patients about their condition, there have been few experimental studies to support these positions because of the difficulty involved in operationalizing criteria such as hope, will to live, and quality of life. In developing the measures of awareness, disengagement, visiting, and length of life versus expected length of life for use in this study, another difficulty was the requirement that the methods be unobtrusive so that they would not affect the state of awareness of the patients and family members.

We first compared our group of cancer patients with a matched group of noncancer patients from the same medical floors and found no significant differences in awareness or disengagement. Of course, the noncancer patients also had chronic illnesses and some were terminal. Nevertheless, in this hospital for these patients the diagnosis of cancer did not affect disengagement or awareness significantly more than did other diagnoses.

Cancer patients who were aware were more engaged than those who were unaware, but this was also true for the noncancer patients. Awareness of a diagnosis of cancer appeared to have no greater impact on disengagement than awareness of other diagnoses. It may be that instead of assessing the effects of awareness on disengagement we were really observing the effects of engagement on awareness. Engaged individuals are more interested in the world and their surroundings and may therefore ask more questions and receive more information. To resolve this question we would need to conduct a longitudinal study to learn whether engaged people are told their diagnoses more often than disengaged patients and then to note how a change in awareness affects the state of engagement.

Awareness was not related to frequency of visits by family and friends, whereas engagement was significantly related to visiting. Engaged people were visited more often than those who were disengaged.

With respect to the longevity of cancer patients, neither awareness nor disengagement was significantly related to actual versus expected length of life after diagnosis. However, there was a significant interaction. Patients who were aware lived longer if they were engaged, whereas patients who were unaware lived longer if they were disengaged. It may be that awareness is more beneficial for those patients who are engaged and interested and possibly ask more questions, whereas disengaged individuals may do better if they are not given more information than they request. We did find that the actual time lived past diagnosis correlated significantly with statistically derived life expectancies.

Our study does not provide answers as to who should be told, when they should be told, what they should be told, how they should be told, or by whom. More research is required. We have demonstrated that questions such as these can be experimentally investigated and evaluated in relation to meaningful and measurable criteria. Until this research is done, caution and judgment should be exercised in sharing information with terminally ill patients. Cancer patients who were aware of their diagnoses did not have more visitors and did not live longer than those who were unaware. With respect to engagement, awareness of cancer did not appear to have a greater effect than awareness of other diagnoses. Since awareness may be beneficial for some patients but not for all, each patient should be evaluated as carefully as possible and treated with judgment, respect, and sensitivity to his or her individual needs, fears, strengths, and anxieties.

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APPENDIX 1
Nonverbal Disengagement Index[1]

I. *Dimensions*
A. *Orientation:* the direction in which the patient is facing

[1]Instructions for completing the Index are as follows: "Choose the description under each heading that best fits the patient. Check the box on the score sheet that corresponds with the description you choose."

1. To observer or other person (including talking on the phone)
2. To object requiring attention or activity (tray, bathing, reading, etc.)
3. Neutral
4. To object with no activity occurring, blank space, or wall
5. Seems turned away from any potential stimulus to activity or attention (such as turning back on person talking, pretending sleep, or having curtains pulled around the bed)

B. *Posture:* the patient's position in space
1. Movement of person or extremities in or out of bed
2. In bed, nonreclining
3. In bed, reclining
4. Propped in chair or bed—inactive
5. Lying in bed in fetal, closed, or rigid position

C. *Grooming*
1. Well groomed
2. —
3. Clean but not neat (disheveled, unshaven, or clothes rumpled)
4. —
5. Not groomed

D. *Dress*
1. Wears his own clothing
2. —
3. Wears a combination of his own and hospital clothing
4. —
5. Does not use any personal clothing

E. *Personal effects in the room:* includes everything from having the TV service to grooming articles, radios, food, or pictures brought from home and gifts, flowers, or cards sent by others.[2]
1. Family pictures, favorite pillows, or work brought from home
2. Hobby objects (including nonfiction books, food requested from home)
3. Light reading materials, radio, or TV service
4. Flowers, cards, or gifts and grooming objects
5. No personal objects

F. *Reaction 1:* patient reactions when you do not initiate interaction
1. Initiates personal interaction
2. Makes a request of a functional nature or a comment related to something about his care
3. Reacts with social amenities ("please," "thanks," etc.); relatively noninteractive
4. Glances at you—reactions tend to be noncommittal
5. Turns away or ignores you

G. *Reaction 2:* patient's reactions when you have smiled at him or initiated interaction verbally
1. Interacts with you verbally
2. Reacts with a bare minimum of what is socially acceptable
3. Smiles, carries out social amenities, but does not converse
4. Glances, makes noncommittal responses
5. Gives no acknowledgment of you or turns away

H. *Modesty*
1. Dressed as though he were in a hotel—very modest
2. More casual but appropriately dressed
3. Mixed impression
4. Partially exposed through light clothing, etc.
5. Exposes breasts, genitals, or buttocks

I. *Expression*
1. Emotionally expressive
2. —
3. Nondefinitive expression
4. —
5. Turned inward, preoccupied, or blank

J. *Activity*
1. Interacting with people (verbally or nonverbally, including telephoning)
2. Active use of objects (those requiring active manipulation)
3. "Passive" type activity
4. Pretense of activity or token activity (becomes active with persuasion but quickly stops)
5. Nonrelated to environment through activity

[2]Marking item number 1 under personal effects means that the patient has the things listed in that item plus all the rest. This indicates the highest level of personal effects.

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Psychiatric Combat Reactions During the Yom Kippur War

BY ITZHAK LEVAV, M.D., HAIM GREENFELD, AND ELI BARUCH, M.D.

The authors explore the stress of war by reporting some selected epidemiological aspects of the psychiatric combat reactions among Israeli troops during a period of intensive hostilities in the Yom Kippur War of 1973. They examined the distribution of these casualties according to intensity of fire, age, rank, sociopsychological grading, and military assignment and measured treatment outcome at the end of the war and 18 months later.

WARS HAVE BEEN and, regrettably, still are laboratories in which the effect of major stressors can be investigated (1). Glass (2) stated that "wars provide unusual opportunities for learning, firsthand, the problem of man's adaptation under difficult and changing circumstances."

The stress of war is the theme of this report. We will deal with selected psychiatric epidemiological aspects of the casualties during an active fighting period in the Yom Kippur War of 1973. We feel that the results of this inquiry contribute to the growing body of literature on stress, which encompasses natural and man-made disasters (3) at one extreme and the general spectrum of less exceptional life events (4) at the other.

We have made use of the concept of the epidemiological triangle of agent (intensity of fire), host (age, rank, and sociopsychological grading), and environment (Army corps and military assignment) to guide the presentation of our data. We feel that this model helps coalesce findings that have developed somewhat apart in the literature (5).

The different labels given to the psychopathology related to war stress epitomize the opposing stands regarding its etiology and treatment. In a review of the experience accrued during World War II and the Korean conflict (5), Glass stated that the early label of "psychoneurosis" has "implications of unresolved intrapsychic conflict from which symptoms are unsuccessfully derived" but that war studies have "caused awareness that social and situational determinants of behavior are more important than the assets and liabilities of individuals involved in coping with war stress and strain." The literature has, in part, adopted opposing positions, addressing itself either to the premorbid personality of the soldier who suffers psychiatric combat reaction (host) or to the military factors related to the rate of casualties (agent/environment). We believe that the data presented here will lead to the adoption of an eclectic stand.

METHOD

The time span covered by the study was a period of active warfare characterized by a large number of casualties in all of the troops involved in the fighting. Case identification and time of onset of the combat reaction were based on the review of the record of each soldier evacuated to treatment centers away from the firing line. The group studied was composed of soldiers suffering from acute combat reactions who were treated away from the front line by specialized Army personnel. The study group did not include soldiers whose combat reactions were treated in their units or at the battalion level by Army medical teams or those whose emotional reaction accompanied a physical injury.

The diagnoses were independently made by two of us (I.L. and H.G.). In only a few of the cases (less than 2%) were there discrepant diagnoses; these were settled by agreement.

Additional information on each soldier was obtained from his personal military file. This information included educational level, ethnic affiliation, and previous military assignments as well as a sociopsychological grading that provided an indication of the individual's intelligence and personality features. The sociopsychological grading, which represents the measurements of one element of the epidemiological triangle (host), consists of education, command of the language, general abilities, and intelligence. All these are regarded as measures of the intellectual function of the individual. Personality features were determined by such measures as sociability, punctuality, and identification with the Army (6). Each file also included a mili-

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tary-psychiatric grading—an overall clinical evaluation of the type of psychopathology present and an appraisal of the disability level of the individual relative to his role in the Army.

Data on changes that took place in the military-psychiatric grading after intervention by mental health personnel were also available. These gradings were compared with those made at follow-up 18 months after the cessation of hostilities. Information was also obtained on the few cases in which changes had taken place at a time other than at follow-up but after 18 months.

The study group was compared with soldiers who had been in the theater of war and who had suffered nonpsychiatric injuries. The latter group, which was used for comparison rather than as a true control group, resembled the study group in type of military assignment—active participation in battle. The essential difference between the groups was in the type of pathology that prompted removal from the firing line.

CASE REPORTS

Case 1. A soldier was brought to the field hospital in a state of clouding of consciousness. During the course of abreactive treatment the soldier described having experienced the following traumatic events: after a number of days of continuous fighting, with almost no food or sleep, his unit suffered heavy bombing from the air. After losing consciousness he awakened in a ditch, where another unit found him. He was then transferred to the medical station for treatment. However, this station was also bombed. He again passed out and when he awakened found himself in a field hospital. The hospital personnel gave him a diagnosis of gross stress reaction, conversion type, and recommended that he be kept for a short rest and then returned to his original unit.

Case 2. A soldier was transferred to a general hospital in the rear, where he was interviewed by a psychologist. He had been evacuated from his unit because he lost consciousness after a shell hit his tank. A fellow tankman with whom he had served for about 10 years was killed in that action. The hospital record indicated that while in the hospital he still suffered from dizziness, pain and pressure in his head, insomnia, tension, and poor concentration and was totally inactive, lying in bed most of the time.

FINDINGS

Agent Data

(Because of security considerations the data presented do not include rates or the number of soldiers in each group used as bases for percentages. Figure 1 has a schematic format for the same reason. The tests of significance were based on the original numbers, which are classified information.)

The data on the agent (intensity of warfare or fire) in the epidemiological triangle were measured by the number of soldiers wounded in action, a criterion suggested by Glass (7) on the basis of his studies during World War II. Figure 1 shows that the shape of the curve depicting the frequency distribution of psychiatric combat reactions is quite similar to that of the physical casualties except for a short time lag between the two.

FIGURE 1
Percent of Subjects Involved in Physical (Wounded) and Psychiatric Casualties by Day of Combat

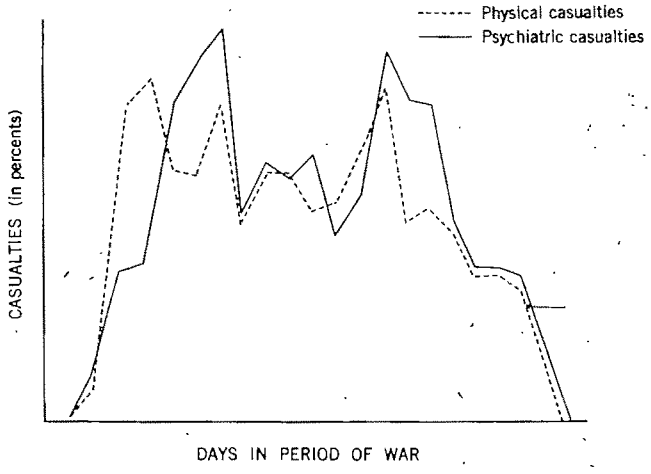


TABLE 1
Number of Physical (Dead and Wounded) and Psychiatric Casualties

Army Unit	Deaths	Wounded	Psychiatric Casualties
A	5	30	17
B	2	15	13
C	0	7	4
D	0	1	1
E	0	2	0

A closer look at the casualties of five Army units provides a further corroboration of this relationship. Table 1 shows that the higher the number of dead and wounded the higher the number of psychiatric combat reactions. In other words, the more intense the fire the higher the number of psychiatric casualties.

Environmental Data

Military assignment essentially determines such factors as whether the soldier is in a passive or active role in the face of danger, whether he is alone or in a group, and whether he strongly or loosely identifies with his unit. As an illustration, table 2 shows a comparison of three major military assignments. The fighting group includes soldiers whose primary duty was to actively participate in combat. The logistic group includes soldiers whose primary task was to provide logistic support for the fighting group. The services group includes soldiers in the service units posted either close to or relatively far away from the firing line. A comparison of the distribution of the psychiatric casualties and the physically injured in combat or, rather, the examina-

TABLE 2
Distribution of Psychiatric and Physical (Wounded) Casualties by Military Assignment

Assignment	Percent of Psychiatric Casualties	Percent of Physical Casualties	Ratio of Psychiatric to Physical Casualties
All troops			
Fighting	69.8	89.5	0.8
Logistic	25.5	8.5	3.0
Services	4.7	2.0	2.4
Two selected units			
Fighting	3.4	10.9	0.3
Logistic	4.7	2.5	1.9
Other	91.9	86.6	1.1

TABLE 3
Distribution of Psychiatric and Physical (Wounded) Casualties by Rank, Age, and Sociopsychological Grading

Characteristic	Percent of Psychiatric Casualties	Percent of Physical Casualties	Ratio of Psychiatric to Physical Casualties
Rank[a]			
Nonofficers with low or intermediate rank	91.4	79.4	1.2
Nonofficers with high rank	3.4	4.6	0.7
Officers	3.0	16.0	0.2
Age[b]			
Up to 18[c]	4.8	0.02	240.0
19-24	31.3	50.6	0.6
25-29	38.9	28.7	1.4
30 and older	23.2	20.8	1.1
Sociopsychological grading[d]			
Low	15.5	8.4	1.9
Middle	32.8	16.8	2.0
High	50.9	75.0	0.7

[a]The rank of 2.2% of the soldiers was unknown.
[b]The age of 1.8% of the soldiers was unknown.
[c]The number of soldiers in this age group was so small that I was advised to drop this category altogether.
[d]The grading of .8% of the soldiers was unknown.

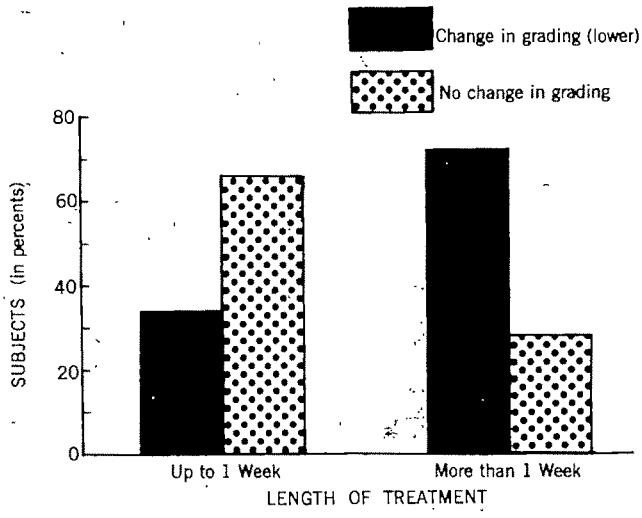
tion of the ratios observed by dividing the former by the latter shows that a soldier in a nonfighting assignment was in a relatively high risk group for war stress. This difference was statistically significant (χ^2=256.85, df=2, p<.001).

Table 2 also shows a comparison of two Army units with different assignments. We built a control for age and, to a limited extent, personality factors into this comparison, and the results are identical to those for the whole Army. Soldiers in the nonfighting groups had significantly more psychiatric casualties (χ^2=48.62, df=2, p<.001).

Host Data

Military rank is a complex concept that encompasses personality features, abilities, degree of responsibility, and relationships with other men. To be an officer usually implies, among other things, that one is intelligent and well educated, has a higher set of norms, and is able to provide leadership. In the Israeli Army, the officer usually leads his unit into battle; thus, he places himself in a most vulnerable position. The comparison of physical casualties and psychiatric combat reactions is of interest for the elucidation of the role played by the host in the epidemiological triangle. The data in table 3 show that officers were five times less vulnerable to war stress; the inverse was true in the lowest rank. This difference was significant (χ^2=134.20, df=2, p<.001).

FIGURE 2
Percent of Subjects Receiving Changes in Military-Psychiatric Grading Before and After 1 Week of Treatment



A comparison of both groups of casualties by age shows that the youngest soldiers and those 25 to 29 years old were particularly vulnerable to war stress (see table 3). This difference was significant (χ^2=248.42, df=2, p<.002). Table 3 also shows that men with lower sociopsychological gradings seemed to have been at significantly higher risk to develop war stress (χ^2=29.32, df=2, p<.001).

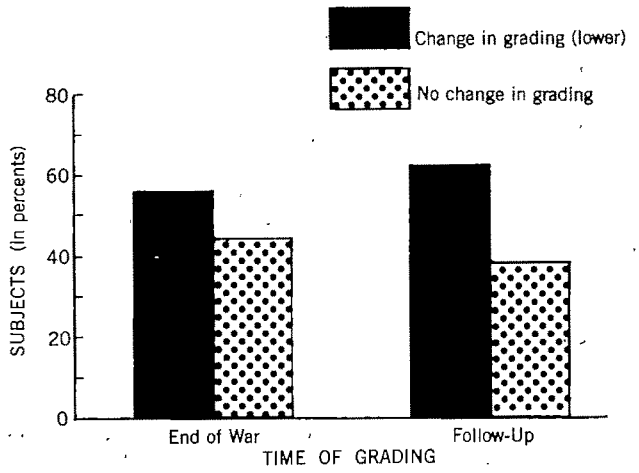
Other things being equal, the ratios of psychiatric to physical casualties show the greater role of host and environmental factors in contrast to the agent as a factor, as shown in figure 1 and table 1.

Treatment Outcome

Treatment outcome was measured in terms of the change that took place in the military-psychiatric grading of the soldier. Since guidelines for treatment have been well established in the literature and there is universal agreement as to their benefit (7), we examined the data to see whether further insights might be gained. We found that treatment lasting more than 1 week brought about changes in the military-psychiatric grading in more than 50% of the studied population: the longer the treatment the higher the likelihood the soldier was given a lower grading (see figure 2).

Of the variables investigated, the sociopsychologi-

FIGURE 3
Percent of Subjects Receiving Changes in Military-Psychiatric Grading by the End of the War and at 18-Month Follow-Up



cal grading was the only one found to be highly correlated with the military-psychiatric grading (Kendall's Tau B=.240, p=.002). The lower the sociopsychological grading the lower the military-psychiatric grading, indicating that personality factors acted in the rate of recovery.

By the end of the war, 55.1% of the psychiatric casualties had received gradings lower than the ones they had had at the outset of hostilities. Of these, 17.4% were regarded as temporarily or definitely (1.2%) unfit for further military duties. The status of the group as a whole did not tend to improve at 18-month follow-up. On the contrary, new cases (6%) were eventually added to the group whose profiles were lowered (see figure 3). Again, the sociopsychological grading was correlated with the military-psychiatric grading at follow-up (Kendall's Tau B=.197, p=.01).

DISCUSSION

The concept of combat psychiatric reactions as used in this study comprises all behavioral manifestations related to the stress of war, including psychomotor and sensorial manifestions. However, no claim for completeness can be made because it is likely that such allied psychiatric problems as self-inflicted wounds, illegal avoidance of combat, and passive participation in battle (7) were not covered because of the sources of data used.

The psychiatric casualties included in this report were only those treated at the rear. However, in view of administrative directives in force at the time, it is reasonable to assume that the study population comprises almost all of the casualties because soldiers who required any intervention by a physician were evacuated. (Interviews held with physicians who staffed the medical outfits of some of the battalions most actively engaged in combat confirmed the fact that only a small minority of soldiers were treated at the front.)

In a study of Swedish soldiers who were involved in combat in the Congo (Zaire) in 1961 as members of the United Nation Forces (8), Kettner compared 35 men who broke down from "combat exhaustion" with 35 control subjects who were "serving at the same time and under comparable conditions in the Congo but did not break down." Despite the neatness of his investigation, however, Kettner's number of cases was small. Furthermore, since these soldiers belonged to a selective force, their situation rendered the study less comparable to those of other wars, e.g., World War II (9), or to most armies that use different criteria for recruitment than does the United Nations. Therefore, despite the progress made as a result of Kettner's study the problem of control groups in studies of war stress remains open.

The group of physical casualties used for comparison in our study does not constitute a control group in the strict sense of the term (10) because of the interrelationship between the physical and psychiatric casualties. However, constraints arising out of the realities of war demand a softer stand that allows for the argument that the group selected is potentially useful for the comparison of such variables as rank because men serving in the same units were subjected to identical fire conditions.

The epidemiological triangle shows the factors related to the agent, the host, and the environment were involved in whether or not a soldier had psychiatric difficulties. In fact, a composite profile of the man at risk could be built: given a day of intense fire, the youngest soldier or the one older than 25, of low military rank and with a low sociopsychological grading, participating in combat in logistic or service units, was relatively more vulnerable to stress.

We have not exhausted all of the factors likely to be linked to combat psychopathology (11). In fact, we have restricted ourselves to only a few of them. It is of interest to consider that vulnerability increases in an inverse relationship to the scores obtained on tests of certain personality features (e.g., punctuality and sociability) and measures of intelligence. These results are comparable to those suggested by Glass and associates (12) in studies of prediction of performance in the military and, to some extent, to Lazarus' on coping behavior according to personality style (13). It is worth noting that the factors cited above operate even when unfit men have already been screened out at recruitment. These factors were not found in Kettner's very selective troops (8), but it was possible to find these factors in our study because of the wider base of the Israeli Army.

Another factor worth emphasizing is the protective effect of the soldier's role: adaptation to danger through activity seems to diminish psychiatric risk. However, it should be noted that the way we measured role—by military assignment—includes a consideration of the availability of support through imme-

diate interaction with other soldiers. A relatively low proportion of officers broke down, but personality factors and intelligence also played a part in this finding. Not all men are equally vulnerable to stress (fire, in this case). Both predisposition and environmental factors account for breakdown in combat. (Unfortunately, we did not analyze their relative weight.)

Our data might seem to indicate that a critical period of up to 1 week for the treatment of combat reactions establishes a dividing line between good and poor outcome. Of course, the point could be made that the length of treatment was primarily a function of the type and severity of the psychopathology present and not a result of the use of a criterion based on management procedures. We tend to give a greater weight to the latter argument, at least as far as the treatment of the psychiatric casualties in the Yom Kippur War is concerned. A number of our case records showed that although a recommendation was made that a soldier be returned to his unit, the soldier was admitted to a second treatment setting. Therefore, conditions other than those based on psychopathology might have led to a lower military-psychiatric grading. We feel that it would be advisable to add another criterion, brevity, to the three already well established in the literature—immediacy, proximity, and expectancy (14)—to assure satisfactory outcome.

Intense stress leaves psychological scars that cause impaired functioning (15–17). On the basis of a 1-year follow-up study of 17 soldiers (of a total group of 24), Merbaum (18) observed "a chilling picture of the profoundly debilitating effects of [war] stress on future social and emotional adjustment."

The data presented here show that a year and a half after the end of hostilities the majority of the psychiatric casualties were judged unable to function in the military at their prewar level. Admittedly, these data were based on a follow-up that was not designed with the rigor of a research undertaking. Thus, spurious factors—such as the wish of the examining psychiatrist to spare the soldier return to a highly stressing situation, a likelihood to be reckoned with in a country that is still at war with its neighbors, or even the knowledge that the soldier broke down—might have limited the objectivity of the assessment. On the other hand, each soldier was seen at follow-up by more than one psychiatrist, which could have reduced the bias. The high correlation found between personality—measured in terms of sociopsychological grading—and psychiatric difficulties might suggest that not all of the results were in the eyes of the beholder but indicated the true status of the soldier. We could then assume that war stress produces not only acute but also chronic psychiatric disability.

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The Myth of the Alliance with Borderline Patients

BY GERALD ADLER, M.D.

The author questions the concept of a therapeutic alliance in the psychotherapy of patients with borderline and narcissistic personality disorders. The motivating elements in their treatment are the emerging, sustaining transferences, which also allow the patients to appreciate the real qualities of their therapist. As they internalize aspects of their interactions with the therapist, including the therapist's real characteristics that are lacking in themselves, the patients gradually develop the capacity to work collaboratively with him or her. When a solid therapeutic alliance is established, the patients have reached a neurotic level of functioning.

BORDERLINE PATIENTS present serious difficulties to the therapist who wishes to engage them in collaborative psychotherapeutic work. Intrinsic to the problems of these patients is an inability to establish and maintain a therapeutic alliance. The nature of their often overwhelming feelings that emerge during psychotherapy can rapidly disrupt a tentative alliance as well as the continuation of therapy itself. This paper will address the issues of difficulties in alliance formation with borderline patients, present an understanding of the developmental, structural, and defensive aspects of these patients relevant to alliance problems, and stress the aspects of the real and transference relationship between patient and therapist that facilitate or hinder the continuance of the developing fragile alliance with borderline patients.

CORE BORDERLINE ISSUES

The state of the borderline patient's ego development, the quality of his or her object relationships, and the instability of a cohesive self set the stage for complex problems in psychotherapeutic work with them. When these patients are distressed enough to seek or accept treatment, they have regressed to a position in which life and death issues feel, and often are, paramount (1, 2). The usual precipitants involve the loss of a relationship that has offered a feeling of holding or togetherness; this loss has often occurred through the patient's escalating demands for support and sustenance, which are part of the borderline regression. This regression, with its intensified feelings of aloneness and abandonment, is at the core of the borderline problem. The patients' vulnerability stems from a failure to internalize a soothing, dependable, anxiety-mediating relationship (3) that usually develops from adequate experiences of "good-enough mothering" in childhood (4). Often these feelings can also be traced back to the maternal failures occurring between ages one and two, especially at the rapprochement subphase of Mahler's schemata (5) of separation-individuation.

Although it is an obvious oversimplification to ascribe an adult disorder to a specific failure of a childhood developmental phase, the behavior of the adult borderline patient in therapy can be more readily understood by cautious formulation and use of such models. The end result in the adult borderline is an inability to depend on internal resources for soothing and self-esteem and a concomitant inability to trust the external environment to provide what is missing within the person. When the primitive defenses of borderline persons become manifest through splitting, projective identification, denial, primitive idealization, and devaluation, as defined by Kernberg (6), it is extremely difficult for them to make appropriate use of people in their lives. Their allies may take on repudiated aspects of themselves and lose their sustaining or supporting roles.

As part of this emerging regressed state, borderline patients become increasingly in touch with their envy and rage (6), intensifying their sense of aloneness and abandonment. Their anger is particularly devastating to them, since it is experienced as destructive and often annihilatory in its most extreme form. This murderously felt anger propels the borderlines' regression to painful aloneness by triggering a specific aspect of their vulnerability: a loss of evocative memory capacity (3). As defined by Piaget (7) and Fraiberg (8), the capacity for object permanence (evocative memory

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capacity) follows a developmental line that gradually allows the child to have a sustained memory of an object even when the object is absent. This capacity is not attained before 18 months of age and at first is vulnerable to regression to only recognition memory, i.e., the need to have the object repeatedly and frequently present in order to remember it. Once evocative memory capacity is solidly attained, the child has a concept of object permanence. This concept of object permanence gives the child the increasing ability to use internalized aspects of important people as part of himself or herself.

Borderline patients have not achieved a stable evocative memory capacity. When stressed by their mounting rage, they can readily regress to recognition memory capacity or even earlier, i.e., they lose the ability to have sustaining memories and fantasies of important people, intensifying their sense of painful aloneness and abandonment. In psychotherapy the patient's loss of evocative memory capacity is accompanied by his or her feeling that the therapist does not exist or has been destroyed.

THE NARCISSISTIC PERSONALITY-BORDERLINE CONTINUUM

Patients with narcissistic personality disorders, in contrast to borderline patients, do not have the latter's evocative memory vulnerability. In fact, the stability of the narcissistic or self-object transferences that narcissistic personalities establish is a major way of distinguishing them from borderlines. If the mirror and idealizing transferences that Kohut (9, 10) defined as diagnostic of narcissistic personality disorders are allowed to emerge without the therapist's interference in the countertransference, they are a manifestation of the patient's capacity to use the therapist in a new and useful way—as a self-object. The self-object concept defines the patient as requiring a relationship with the therapist of differing degrees of merger on a mirroring and idealization pathway. In this self-object relationship, the therapist performs certain fantasied and/or real functions that the patient feels are missing in himself or herself. When these self-object transferences emerge and are allowed to flourish, there is a sense of stability in the treatment that only momentarily breaks down in a fragmentation manifested by, e.g., transient hypochondriasis or aloof arrogance. This regression may appear when unbearable present or earlier feelings emerge or because of empathic failures of the therapist.

In contrast to this relative stability, the appearance of intense feelings in the borderline patient, for example, rage and envy (6), leads to a much more extreme fragmentation process. This process can result in the borderline patient's desperate emptiness and aloneness as part of his or her regression from evocative memory achievement and the self-object transference cohesiveness.

THE NATURE OF THE ALLIANCE WITH PRIMITIVE PATIENTS

The narcissistic personality-borderline continuum is important in understanding the nature of the motivating forces in the treatment and in defining issues of transference, therapeutic alliance, and real relationship with the therapist. In clinical work with borderline patients, we are frequently impressed with the rapid breakdown of what seems to be a tenuous, or sometimes even more solid, alliance. The "alliance" can disappear, and desperate borderline aloneness can emerge when unbearable affects appear in the therapy or when the therapist makes a response that is unempathic or perhaps incorrect. When we examine the narcissistic personality disorders with their stable self-object transferences, we can ask whether a therapeutic alliance exists or whether these primitive transferences themselves allow the patient to be sustained in the treatment. Although we invoke concepts of alliance and make statements about building alliance, it seems probable that the empathic optimal support and frustration offered by the therapist provide the empathic framework that the patient needs in order to sustain himself or herself with a self-object transference; the therapist can mistake this stable transference for an alliance.

It is often through the failure of our therapeutic work that we can clarify distinctions that were previously obscure. A case report can help to illustrate the absence of an alliance with a patient based on the failure to establish a sustaining self-object transference.

CASE REPORT

A 35-year-old single woman who sought therapy for chronic depression and inability to maintain relationships with men was regarded by both her therapist and his supervisor as someone with a hysterical character problem. After nearly 1 year of twice-weekly psychotherapy, the patient remained essentially unchanged and felt that she was making little progress. The therapist focused his work on her disappointment in her relationship with her father and competitive feelings toward her mother. He also stressed the collaborative nature of their work and emphasized frequently that the two of them were looking at or could look at certain issues and feelings together.

Following one of these exhortations about collaboration after approximately 1 year of therapy, the patient looked her therapist squarely in the eye and said, "Don't give me any more of that 'we' crap!" Although the therapist was momentarily stunned, he had no adequate response or explanation. It was only after careful review of his work with the patient that he concluded that he had been treating someone with a narcissistic personality disorder as a person with a neurotic character problem. His lack of understanding of the nature of the patient's despair and developmental difficulties was perceived by the patient as the therapist's empathic failure. Under those circumstances there was little to sustain the patient except for her perception that the therapist was occa-

sionally emphatically correct and struggled to understand her; however, nothing suggesting a therapeutic alliance was ever present with her.

TRANSFERENCE OR ALLIANCE

My review of the treatment of primitive patients, studied both through supervision of trainees and through conducting intensive psychotherapy, supports the hypothesis that the "we" invoked by the therapist often makes the therapist more comfortable but is effective only when it coincides with the patient's feeling sustained through a self-object transference. The patient usually does not experience the working collaboration; instead, he or she is held in the therapy by feeling supported, soothed, and understood.

At the beginning of the therapeutic process, when the patient's difficulties are clearly borderline with painful aloneness and loss of evocative memory capacity as primary problems, the therapist's main focus is on understanding the regression and the ultimate reestablishment of a holding environment and sustaining self-object transferences. Understanding the reasons for the patient's regression includes assessing whether unbearable affects, especially rage, envy, and disappointments, arose in the therapy related to the therapist, past figures, or people in the patient's current life. Based on this understanding and the awareness of the borderline patient's increased vulnerability when he or she transiently loses evocative memory capacity, the therapist takes appropriate steps to help the patient. These may include clarification and interpretation of the issues leading to the regression from evocative memory capacity, confrontation of the patient's avoidance mechanisms related to his or her ignoring real dangers to self-survival, and the therapist's availability and existence. As the therapist confronts the patient with his or her existence, which the patient cannot experience during this time, the therapist may offer extra appointments and opportunities for telephone contact to reestablish that the therapist does indeed exist, care, and is available for the patient (11). The therapist also balances his or her availability with the realistic limits of his or her own capacity to give and sustain. Thus hospitalization becomes an alternative when the therapist's appropriate help is insufficient.

When successful, these therapist activities demonstrate the therapist's existence, caring, commitment, and willingness to work hard with the patient. They also reestablish the holding, sustaining self-object transferences. However, they do not establish a therapeutic alliance, only the self-object precursors, which ultimately can be internalized slowly as the primitive transferences are resolved and neurotic transferences become more solidly established. At the point that the patient is capable of a solid therapeutic alliance, that patient no longer has a borderline or narcissistic personality disorder; in fact, he or she is well within the neurotic spectrum and approaching the end of therapy.

While borderline issues remain paramount, the tenuous and fluctuating therapeutic alliance with these patients can be monitored by the therapist's constant evaluation of the patient's needs for holding and support on the one hand, and capacity to bear frustration and face his or her disappointments and rage on the other hand. The empathic therapist titrates his or her responses based on an assessment of the patient's current capacities and vulnerabilities. The awareness that the inevitable empathic failures of all therapists will lead to episodes of the patient's feeling intense aloneness and abandonment provides the therapist with opportunities to clarify with the patient the nature of his or her vulnerability. Through this activity and support, the regressions that occur during psychotherpy with borderlines can most often be confined to the therapy session.

Over time, the patient learns that the therapist is a dependable person who can understand and bear the patient's transference distortions and exquisite vulnerability to regress whenever intense feelings emerge in his or her life and in therapy. With repeated episodes, the patient's regression becomes less intense because the repeated regressive episodes and their therapeutic utilization lead to the internalization of the self-object function and to structure formation. The consequent developing capacity to feel soothed and held while apart from the therapist, which includes the capacity to evoke the memory of the therapist between sessions, goes hand in hand with the development of higher level defenses, including the healing of splitting and the capacity to tolerate anger, closeness, and aloneness.

TRANSFERENCE, REAL RELATIONSHIP, AND ALLIANCE

In discussions of psychotherapeutic work with primitive patients, we often hear that the real relationship between patient and therapist is particularly important. The real relationship is rarely defined but is sometimes equated with the therapist's revealing much about himself or herself and his or her life and background (12). In the context of the present paper, we have the opportunity to explore the nature of the patient's real relationship with the therapist and its relevance to the discussion of the therapeutic alliance and sustaining primitive transferences.

Clinically, we have examples of therapists who reveal much about themselves to their patients yet find that their patients are stuck in a regressive impasse (12). It is also not unusual for primitive patients who know many facts about their therapist to say that they really do not *know* their therapist. Exploration of these statements often clarifies that these patients do not see others as separate from themselves; when they are not in a relationship in which they are partially fused to or merged with another person, they feel empty, fragmented, alone, and uncertain about who they are and

who the other person is. However, when they feel sustained in a relationship that offers them the degree and kind of merger or idealization experience they require, they feel whole and do not raise questions about not knowing the other person. Thus, when sustained in a self-object kind of relationship, they feel complete and do not question the reality of the other person when that person is part of themselves.

Paradoxically, when these patients are sustained in the stability of a self-object transference, they begin to develop the capacity to observe and appreciate the real qualities of the person they simultaneously use as a self-object. This capacity to see the real or objective qualities of people increases as they gradually internalize functions from the self-object that are missing aspects of their own ego and self. Finally, when they are sufficiently separate and able to appreciate the real aspects of their therapist, they can work increasingly collaboratively with him or her, i.e., they can establish a therapeutic alliance. To summarize, I belive that a sequence occurs in the successful therapy of primitive patients: 1) the establishment of stable self-object transferences that sustain them, 2) the increasing capacity to appreciate the therapist as a real and separate person, and 3) their gradual ability to ally themselves with their therapist in the service of accomplishing work.

Using these formulations, the therapist has as a major task the clarification of where the patient lies in this continuum, what causes the patient's fluctuations within it, and what responses by the therapist will solidify the patient's achievements as he or she advances along this developmental sequence. Thus the primitive patient's dissatisfaction that the therapist is not real to him or her may be viewed as the patient's failure to establish a sustaining self-object transference at that moment. The therapist's formulation and empathic understanding determines his or her responses at different times and is specifically related to a clarification or interpretation that addresses the appropriate point of the developmental sequence described earlier.

The therapist's comfort with himself or herself and the countertransference feelings that emerge with these primitive patients is an obvious but important factor in the ultimate outcome of the psychotherapy (13). This personal comfort can allow the therapist to be flexible and use his or her human qualities and warmth appropriately in the best interests of the patient. The combination of a theoretical model and an empathic flexibility can provide a framework in which the therapist can use his or her intellect and empathy for optimal psychotherapeutic work with this challenging group of patients and gradually increase our growing understanding of them.

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A Psychoanalyst Reports at Mid-Career

BY JOHN E. GEDO, M.D.

The author states that the current pessimistic impression of the potential of psychoanalysis as a therapy is largely due to the inclusion of a disproportionate number of inexperienced analysts in reports on groups of practitioners. To correct this bias, he summarizes his own experience of two decades as a full-time psychoanalyst, describing his successful analyses as well as his unsuccessful attempts. He concludes that the satisfaction of commitment to a psychoanalytic career comes from the fact that it confirms the usefulness of the analytic method as a means to assist others to achieve personal growth and to illuminate human mental life.

REPORTS BASED on the work of groups of practitioners have created a misleadingly pessimistic impression of the potential of psychoanalysis as a therapy because such surveys have included a disproportionate number of inexperienced analysts. As a corrective for this source of bias, I propose to summarize my own experience as a clinician who has taken the unusual course of total immersion in psychoanalytic practice. The price one has to pay for such a choice is that of abandoning the hope of rendering service to significant numbers of people. Its rewards lie in the direction of perfecting oneself as a technician so that one can apply the psychoanalytic method in the area of its greatest utility, that of a unique research tool for the illumination of human mental life. The vast majority of psychoanalysts devote themselves primarily to patient care, spending most of their professional life performing treatments less time-consuming than the psychoanalytic procedure itself (1).

By contrast, over the past 20 years I have treated a total of 36 people (exclusive of those currently working with me). Among the patients who completed treatment, 28 terminated analysis on the basis of a consensus about the satisfactory outcome of the enterprise. The others were about evenly divided among those who interrupted treatment to resume later with me or elsewhere, therapeutic failures in spite of prolonged efforts, and patients who fled treatment against advice.

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THE PATIENTS

My group of 36 patients, who were exactly divided between men and women, represented a relatively narrow segment of the professional and academic elite. All were white and were born in the United States. Nominally, more than half were Protestants, about one-third were Jewish, and there was a scattering of other faiths. By profession, one-third were in academic life, either as faculty or as graduate students, about a fourth were mental health professionals, and another fourth were evenly divided among journalism, the arts, business, and law. The women who were not employed were married to men of professional status. At the start of treatment, the patients were evenly distributed in the 22–46-year-old age range. Half of the patients had had some form of previous psychotherapy (some with me many years ago), and one-sixth more had been in analysis elsewhere.

Without exception, analysis was undertaken to alleviate the consequences of a complex and severe character disturbance. Most of these patients had decided on analysis only after a serious adaptive failure at work, in family life, or both, resulting in severe loss of self-esteem. The presenting clinical picture was often alarming, with depression, suicidal preoccupations, frantic perverse activities, panic, or psychosomatic decompensation. Whatever the symptoms, I adhered to an unvarying policy of accepting anyone with a serious commitment to seeking self-understanding if there was no clear risk of psychotic fragmentation or a likely need for hospitalization.

A fourth of the patients confronted the necessity for major character change following the dissolution of a marriage, and more than another fourth were currently experiencing severe marital strains. In fact, of the latter group, about half eventually dissolved their marriage. A third quartile consisted of people who had never married and who realized that their characteristic mode of relating to the opposite sex absolutely precluded having a satisfactory family life. Thus, fewer than one-fourth of my patients had reasonably gratifying intimate relationships. In the sphere of work, at least 1 in 4 had reached a dead end in their career and a like number were seriously dissatisfied with their professional progress. Overt adaptive difficulties account-

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ed for the decision to seek analysis on the part of all but 4 patients. These 4, most of whom were mental health professionals, sought help for subjective discomforts alone.

THE ANALYSIS

The analytic procedure was invariably carried out in the so-called classical manner (2, 3), one hopes in the spirit of humane common sense recommended by the best clinicians (4, 5). Actually, my schedule permits about 160 sessions per year if my patients and I meet 4 times a week (or 200 if we can manage 5 weekly appointments). With negligible exceptions, analyses that reach a successful conclusion require more than 600 sessions, sometimes as many as 1,000, spread over 3 to 7 years. People who interrupt treatment generally do so relatively early, usually after about a year of work. In only one instance did a patient decide to leave prematurely at a later stage. Conversely, in the cases that ended in failure, we did not reach that reluctant conclusion until the analytic process had been given a chance to unfold in the usual manner over a number of years.

Regardless of outcome, the decision actually to dispense with my services was left entirely to the patient. The analyst should only state an expert opinion about what remains to be accomplished and what the chances of reaching those goals might be. The duration of treatment did not turn out to be directly related to the severity of psychopathology; it seemed to be more closely tied to the specific issue of how rapidly I was able to develop a meaningful way of communicating with the analysand. That task is much more difficult with people who suffer from disturbances with a genesis in early childhood. Such developmental problems may impair the patient's language functions or even the capacity for rational thought. Cultural differences may also contribute to difficulties in communication.

The work of two decades was scarcely uniform in quality; significant changes are bound to occur over such a span. Increased experience may lead to greater skill, increased age brings older patients, and general progress in the field induces changes in one's approach. The factor of experience is easiest to pinpoint. By far the greatest proportion of my analytic failures occurred immediately after my graduation from a psychoanalytic institute, i.e., among the first patients I tried to treat without supervision. Perhaps the most meaningful change in my therapeutic philosophy has involved the gradual development of methods for dealing with the effects of earlier maturational vicissitudes on the typical intrapsychic conflicts discovered by Freud (6, pp. 153–168).

I cannot report on the results of my analytic efforts with complete confidence because of the lack of systematic follow-up study; perhaps only with incomplete cases can the ultimate outcome be gauged when the work is stopped. However, without my making any deliberate effort to learn about my former patients, relevant information has come to my attention about a substantial proportion of them. Such intelligence becomes available more or less automatically about mental health professionals who reside in the same community and about people with intimate friends or relatives in the analytic world. Occasionally, patients return for a consultation about some crisis in their life or that of a family member; others may write to communicate some piece of news, usually about a major accomplishment. As a result, I have learned something about the later outcome of most of the analyses that have successfully terminated. By contrast, I have seldom had news about patients who did not reach a successful analytic termination.

SATISFACTORY TERMINATIONS

Clearly, a satisfactory termination implies that the major defects in adaptive capacity which prompted the patients to come for assistance are no longer in evidence. Favorable results were particularly striking with regard to career problems. Patients whose analysis ended favorably have invariably done well in the sphere of work; often they have achieved impressive public success. In crude financial terms, analysis proved to be a sound investment for them. A number of patients, including some of the married women who had not been employed previously, launched new careers. Several women devoted themselves to rearing their children with enthusiasm and satisfaction. To my knowledge, only 1 of these patients has had subsequent problems with work; excessive ambition led this person to attempt more than he was qualified to achieve.

It is more difficult to assess outcome in the sphere of intimacy. An external criterion, such as the establishment of gratifying family life, cannot be used because it depends in part on circumstances beyond the control of the individual. Women older than 35 are less likely to marry than are younger women; among my patients, only one woman older than 35 married. Among men and among women under 35, about three-quarters entered permanent relationships after successful analysis, as far as I know. More importantly, all of the desperately strained, masochistic relationships that brought my successful patients into treatment had come to an end as a result of the work, either through separation or because of improvements, often facilitated by psychoanalytic treatment of the affected spouse.

In summary, the successfully terminated analyses were characterized, as a group, by extensive favorable adaptive changes in the capacity for productive work as well as in the capacity for gratifying personal intimacy. It may well be possible to obtain comparable results through other psychotherapeutic methods, but I have never been able to do so (7). Nor would I assume that the permanent disappearance of the more

threatening symptoms (perverse activities, hypochondriasis, psychosomatic crises, mood disorders) could have been accomplished only by psychoanalytic means. The therapeutic results should be regarded as specifically psychoanalytic only if they reflect actual maturation in those mental functions which subserve adaptation, i.e., if the results are sustained because the analysand achieved effective self-regulation in the areas of former limitations. Whether improved adaptation has really come about in these patients because of such learning or simply because they received continuous assistance through the relationship to the analyst is quite difficult to determine. This issue generally occupies the forefront in the termination phase of analysis; actual termination should be decided on only if there is reason to believe that improvements will be sustained without the continuing availability of the analyst.

The above description of successful terminations represents the estimate my patients and I made jointly in these cases. Obviously, this is a matter of judgment; only a formal follow-up study of long-term performance after termination can substantiate its validity. The analyst is naturally tempted to believe that no news from an ex-patient is good news, but this assumption is untested. I can claim only that the freedom of the great majority of my ex-patients to contact me when something newsworthy occurs in their lives (or about crisis situations, for that matter) argues for the probability that those who have not been heard from are not simply withholding unfavorable tidings.

The most significant negative information I have gathered about my successfully terminated patients highlights the limitations of my results. In two instances, the difficulties experienced by the ex-patient have required further analysis; a few others involved mental health professionals interested in psychoanalytic training who resumed analytic treatment in connection with their career requirements. One of the people who needed further assistance was the person who got into deep water by setting unrealistic vocational goals. Actually, this person had decided to resume analysis less than 6 months after termination, having returned to tell me with some bitterness about the intention to consult someone else. I have reported the specific dynamics of this disaster in another context (8). At the time of this announcement, the patient's discomfort was purely subjective; the adaptive breakdown that actually led to resumption of treatment occurred some years later.

The second case that required re-analysis was even more startling: the patient went from success to success in every sphere of life and eventually returned for help because of severe anxiety about this unprecedented state of affairs. The second analysis had to be performed by another analyst in another city, but I was informed that the issues which had arisen in the new situation were quite different from the ones this patient and I had dealt with. It would seem, then, that the analytic work had been incomplete in both instances, in spite of the fact that the procedure had been brought to a satisfactory termination in terms of the usual process criteria. An operation may look successful without truly benefiting the patient.

UNSUCCESSFUL ANALYSES

It is always easier to detect one's therapeutic limitations in retrospect. I believe mine have coincided, more or less, with those of the discipline as a whole in the past decade. I started the analyses I was unable to bring to a successful conclusion with some awareness of their potential difficulties. Most of these patients would have been turned away as unpromising by anyone interested in a perfect record as a therapeutic virtuoso. These individuals may not have been unanalyzable, but success could be expected only in unusually fortunate circumstances. For example, the one person who has interrupted treatment in recent years was a woman who had been seriously suicidal when we began. She went through a series of episodes of disorganization in the early stages of analysis, but the availability of assistance gradually calmed this turbulence. The patient was able to rationalize her flight from analysis on the ground that the resulting improvement in her life had more than satisfied her goals; in a literal sense, this was probably true. The therapeutic alliance was ruptured, however, by the emergence through free association of the patient's conviction that she was an evil heretic, unable to live by the rules of the religious sect that defined her entire adult existence. It was my unwillingness to concur in this delusional view that precipitated the crisis: I became the source of evil by telling her she was not a heretic—that the set of private religious convictions she lived by was just as worthy as the sect's official dogma. In trying to treat a prophetess through the psychoanalytic method, I was consciously trying to extend the range of its applicability.

The most obvious factor distinguishing the patients with whom I did not succeed, as a group, was the fact that the effort made was inadequate in the face of overwhelming difficulties. Poor prognostic signs were not always apparent early in the treatment. For example, one patient who eventually proved to be psychotic presented herself in a manner that misled me into seeing her as a hysterical personality. Months later, it emerged that she had been given that diagnosis by a previous consultant. Thoroughly familiar with the phenomenology implied by this term, she became the victim of her pathological suggestibility and unconsciously enacted the part of a model hysteric.

In terms of their ultimate diagnoses, the patients who continued in treatment for many years without appreciable progress had unresolved problems related to the most archaic developmental levels, i.e., they might fit the category of borderline personality organization. None of the successfully terminated analyses dealt with problems of that magnitude. Patients who

interrupted work with me even though they wanted to continue in psychoanalysis did not differ markedly in their personality organization from those I succeeded in analyzing. In these cases, it was impossible to establish a therapeutic alliance for complex reasons involving unfavorable reality circumstances relating to such specific character traits in these individuals as suspiciousness bordering on the paranoid (e.g., complicated third-party arrangements for payment and uncomfortably proximate professional and/or social contacts).

With regard to demographic criteria, a disproportionate number of patients in the unsuccessful group were in their 50s. The group also contained several individuals from cultural backgrounds relatively unfamiliar to me: the religious fanatic I have mentioned, people with a quasi-rural background, and a student radical descended from Italian peasants. I might add that the majority of these patients happened to be in some branch of academic psychology professionally. I wonder if it was more difficult for these institutional "rivals" to "submit" to psychoanalysis than it was for others. There is also the possibility that I have some unconscious prejudice toward them. At any rate, it is probably significant that 3 of these psychologists sought assistance for purely subjective reasons, i.e., without having sustained any demonstrable adaptive failure. I infer that the desire to seek analysis in the absence of any acute life crisis may indicate a sense of deep disturbance with unfavorable prognostic significance.

Failure of an analytic effort does not end the therapist's medical responsibility; on the contrary, such an outcome constitutes a potential emergency because patients are likely to react to it with a sense of catastrophe. There may be panic, depression, and paranoid accusations of maltreatment. It is imperative to deal with these contingencies in an appropriate therapeutic fashion. Treatment of these complications may require much time and effort. (In one case, my responsibility has actually turned out to be interminable.) If the therapist succeeds in demonstrating his or her continuing availability, the acute symptoms may subside; eventually, the therapeutic contact may then be tapered off. In one instance, the patient tried to disprove the unfavorable verdict by making strenuous conscious efforts to show improved adaptation. These attempts were somewhat beneficial, so that from an uninformed vantage point this "analysis" might have appeared to be successful. The patient finally left me with some bitterness because of my continuing insistence on the judgment that *analytic* treatment was not feasible.

The most difficult potential hazard after an analytic effort has failed is the emergence of a regressive state requiring hospitalization. This was the outcome with the "borderline" person whom I had misdiagnosed as having a hysterical personality; under the circumstances, I was obliged to transfer this patient into the care of others. Her subsequent treatment, however, was no more successful.

CONCLUSIONS

In describing my analytic experience over two decades, I have concentrated on observations about people and events. This is relatively easy, but it is also the less significant part of what needs to be said. The more meaningful side of these experiences is subjective, and this is almost impossible to describe adequately. Eighty years ago Sigmund Freud wrote that he found it difficult to convey the intellectual beauty of his work. Indeed, psychoanalysis continues to offer the kind of excitement, complexity, novelty, and rigor that characterize great art, scientific discovery, or other intellectual problem-solving on the highest levels. The evolution of our understanding of the complexities of psychic organization has entailed a corresponding increase in the therapeutic effectiveness of the analytic method. In principle, the analyst should not approach his or her clinical work with the personal need to be a healer; to require patients to improve is an illegitimate infringement on their autonomy. Nevertheless, much of the satisfaction of commitment to a psychoanalytic career comes from the continuous reconfirmation of the matchless usefulness of the analytic method as a means for personal growth.

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Psychiatric Malpractice: The California Experience

BY PAUL FREDRIC SLAWSON, M.D.

The cost of psychiatric malpractice insurance has increased dramatically in recent years. To shed light on the extent to which this increase is justified the author polled 133 California psychiatrists who had indicated experience with some form of claims activity over the past 5 years. The 107 respondents reported 105 cases, of which 57 (54%) resulted in no more than a notification of a possible claim, 22 (21%) did not proceed beyond legal deposition, 20 (19%) were settled by dollar payment before trial, and 6 (6%) were left to the courts. The author recommends tort reform and refinement of reported claim and loss data to contain the cost of malpractice insurance.

IN THE PAST FEW YEARS an unprecedented number of patients have initiated lawsuits against doctors, asserting negligent practice of medicine. A "malpractice crisis" developed, and one result was that doctors were threatened with large increases in liability insurance premiums. This expense would be passed on to patients in the form of higher professional fees. The higher premiums reflected the insurance industry's response to an increasing number of liberal awards to patients whose complaints would have found modest, if any, satisfaction in the courts only a few years before. The largest number of suits were in the surgical specialties, particularly those associated with an immediate threat to life. In this area, awards based on estimates of the cost of long-term care, coupled with an allowance for inflation and compensation for pain and suffering, led to indemnity costs grossly beyond reasonable expectation. The insurance companies reacted by either sharply increasing their premiums or withdrawing from the market.

Until recently all of this had little impact on the practice of psychiatry. Professional liability premiums for psychiatrists have traditionally been low, reflecting an insurance industry belief that psychiatric practice involved infrequent occurrences of malpractice suits and a modest exposure to loss. However, in the last 3 years the cost of malpractice insurance for psychiatrists has increased substantially in almost all metropolitan areas. What was a minor office expense has become a major factor in the cost of practicing psychiatry. There is a need to justify this change.

The literature on psychiatric malpractice is scant. Studies have shown psychiatry to be a lower-risk form of medical practice but have provided more qualitative than quantitative insight into the nature of the problem. Bellamy (1) reviewed psychiatric malpractice cases that reached the level of the appellate court during a 15-year period and found that the number of cases increased in each of the successive 5-year periods studied. Rothblatt and Leroy (2) cited psychiatric malpractice figures for the years 1931 through 1971. Between 1931 and 1940 they noted only 3 cases. However, between 1941 and 1950 they found 7, and between 1951 and 1960 there were 9. During the last period of their study, 1961 to 1971, they found 25 cases. These data would suggest the probability of a significant change in claims experience during the period under study.

In an effort to gain a better understanding of a problem that was clearly going to be of concern to California psychiatrists, APA's Area Six conducted a survey of its membership to determine the frequency and extent of claims of malpractice. This was accomplished through a questionnaire sent to more than 3,100 psychiatrists in the state. The response rate was over 50%, and the initial results of this survey were reported in an earlier study (3).

A summary of the initial findings is provided below:

1. Of the 1,537 responding psychiatrists, just over 50% were involved in full-time private practice; 95% purchased malpractice insurance with average limits of liability in excess of $200,000–$600,000.
2. Almost all of the responding psychiatrists prescribed drugs, but less than 10% gave ECT.
3. Over 50% of the respondents had minimal hospital practice, and only 20% carried an average hospital caseload of more than 3 patients.
4. The responding psychiatrists made a total of 166 notifications of a claim or potential claim to their insurance company in the 5-year period from 1971 to 1976.
5. In a given year, about 2% of the insured psychiatrists had some form of claims activity.
6. Interpolation of claims frequencies of the doctors who reported more than 1 claim in the 5-year period revealed an estimated rate of claims or potential claim notification of 3 claims per 100 psychiatrists per year.

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Although this information may give some idea of the frequency with which patient dissatisfaction occurs in general psychiatric practice, it does not provide insight into the nature of the problem or its impact on the quality of care, nor does it estimate the cost of this problem as manifested in subsequent litigation and indemnities paid.

It is my intent in this article to review the claims files obtained in the earlier study. For this purpose, 133 psychiatrists who indicated claims activity and who also supplied identification data sufficient to allow more detailed follow-up were asked to complete a supplementary form requesting additional information about the incident reported. This form included the age, sex, diagnosis, and site of treatment of the patient involved, the exact nature of the allegation, and the current status of the complaint (i.e., whether or not it had been resolved or was still pending). If the circumstance was resolved, information was requested with respect to the manner of settlement, the amount of indemnity paid, and whether the resolution was accomplished by settlement before trial, during trial, or in the form of a judgment subsequent to verdict. Inquiry was also made as to the psychiatrist's satisfaction with the manner in which the circumstance was handled by the insurance company and the defense attorney provided by the insurance company.

FINDINGS

Requests for more detailed information concerning malpractice experience were sent to 133 California psychiatrists who had indicated some form of claims activity during the years 1971 to 1976. This yielded 107 responses (80%) and a total of 105 cases or individual complaints suitable for review. The number of cases is smaller than the number of responses because some of the cases supplied by respondents were initiated and often settled before the 5-year period under study. Although a small number of respondents supplied more than 1 case, this was more than offset by the number of cases excluded for reason of a claim filed before 1971. The following data are derived from the 105 cases originating in the established time frame.

The Patients

Almost all malpractice actions are initiated by a patient or someone acting for a patient. Most of the patients involved in the 105 cases under study were middle-aged. Their age range was 12–74 years, and 60% of the patients were within the 25–55-year-old group. There were 4 patients under age 18 and 2 older than 65. Forty-seven of the patients were men, 55 were women, and the sex of 3 was not specified. Thirty-seven of the patients were diagnosed as having schizophrenia (including the borderline states), and 28 were considered primarily depressed. Manic-depressive disease, neuroses, character disorders, and a group of miscellaneous conditions, including organic brain syndrome, each made up less than 10% of the sample. Most of the patients were inpatients. Only 95 of the 105 patients could be classified. Of these, 73 were or had been hospitalized and 22 were exclusively outpatients.

The Incidents

Dissatisfaction with treatment is the major factor in most malpractice actions. In the 105 cases studied there were 114 allegations because a few cases contained more than 1 complaint. Twenty-nine of the cases involved suicide; this amounted to just over 25% of the cases reported. Mistake in diagnosis and/or improper treatment was claimed in 22 (19%). Drug reactions accounted for 19 claims (16%), and 15 cases (13%) involved the assertion of unlawful detention. There were 3 claims of improper sexual involvement and 2 alleged breaches of confidentiality. The range of complaints was broad; 24 cases (21%) could not be classified in a meaningful way. These included potential claims from such diverse activities as rendering first aid at an accident site to an assertion of libel.

The Outcome

Complaints of malpractice are resolved in different ways. Some are simply threats and become nothing more than a filed notice of a potential claim, but others involve a certain amount of legal and investigative work that may result in settlement or trial. Fifty-seven (54%) of the 105 reported cases have, to date, resulted in no more than a notification of a possible claim. Twenty-two cases (21%) have been subject to legal process up to and including deposition but have not proceeded beyond that point. A total of 20 cases (19%) were settled by dollar payment before trial. That leaves 6 cases (6%) for the courts. Of these, 3 have been tried and won by the defendant psychiatrists and 1 was abandoned in midtrial by a plaintiff acting as her own attorney. The remaining 2 cases have not yet come to trial.

Malpractice premiums reflect an effort on the part of the insurance company to anticipate losses. Threats of suit and legal-investigative procedures do not usually entail great expense. However, settlements and court judgments can be very costly. For this reason, the 26 cases that were settled or tried warrant closer study.

The patients involved in the 20 settlements ranged in age from 12 to 70 years. A majority were middle-aged. Thirteen of the patients were women and 7 were men. Seventy-five percent were or had been inpatients. Most were diagnosed as depressed or schizophrenic. The complaints settled were almost equally divided among suicide, claims of improper treatment, and adverse reactions to drugs. Only 2 patients had received ECT; in neither was ECT the issue. The average cost of settlement was $22,400, with a range of $120,000 to $500. Data on these 20 cases are summarized in table 1.

Five of the patients involved in the 6 cases that reached the court level were men and 1 was a woman.

TABLE 1
Summary of 20 Psychiatric Malpractice Actions Resolved by Settlement

Patient Number	Age	Sex	Hospital Status	Diagnosis	Allegation	Settlement (dollars)	Remarks
1	32	F	Inpatient	Depression	Failure to restrain	80,000	Patient jumped from window before transfer from general to neuropsychiatric hospital
2	42	F	Inpatient	Schizo-affective	Suicide in hospital	—	Settled by hospital; patient had received both medication and ECT
3	46	F	Inpatient	Schizophrenia, paranoid type	Improper filing of commitment paper	1,000	Alleged negligence of hospital clerical staff
4	30	F	Inpatient, outpatient	Depressive neurosis	Sexual advance while patient was under hypnosis	5,000	Lawsuit probably triggered by billing dispute
5	61	F	Outpatient	Organic brain syndrome	Improper release of clinical information	1,500	Letter containing clinical information picked up by union representative; patient did not sign release
6	23	M	Inpatient	Mental retardation	Failure to diagnose brain tumor	—	State hospital patient; tumor found 7 years after examination; settled by state
7	70	F	Inpatient	Acute manic episode	Improper restraint, fractured hip	1,000	Doctor held not at fault; insurance company paid nuisance settlement
8	23	F	Inpatient	Depressive neurosis	Wrongful death	25,000	Primary doctor on vacation; patient alleged she took tricyclic overdose; hospitalized; experienced convulsion, asphyxiation; patient died
9	34	M	Outpatient	Manic-depressive	Improper medication, lithium versus chlorpromazine	70,000	Case initially considered defensible; doctor destroyed records; insurance company settled
10	56	F	Inpatient	Psychotic depression	Failure to restrain, suicide	10,000	Private hospital, no seclusion room available; patient checked every 10 minutes; hanged herself with drapery cord
11	38	M	Outpatient	Phobic neurosis	Failure to diagnose	120,000	Driving phobia treated for 2½ years with improvement; progressive ataxia; neurologist diagnosed vitamin B12 deficiency without anemia; medical group settled
12	48	M	Inpatient	Manic-depressive	Improper medication	15,000	Patient alleged lithium caused neurological deficits
13	40	F	Inpatient, outpatient	Depression, schizophrenia	Improper use of medication	2,500	Patient alleged addiction and failure to warn of possible addiction; suit followed collection effort
14	65	F	Outpatient	Manic-depressive	Improper medication	2,990	Patient developed paralysis agitans; claimed symptoms were caused by amitriptyline
15	43	F	Inpatient, outpatient	Neurotic depression	Improper medication and treatment	60,000	Patient took chloral hydrate overdose, with subsequent aspiration, hypoxia, and brain damage
16	—	M	Outpatient	—	Failure to certify patient as air controller	2,000	Single visit, doctor refused request, attorneys settled
17	—	F	Outpatient	—	Bill dispute	2,500	Bill placed for collection, patient sued, attorneys settled
18	43	M	Inpatient	—	Failure to diagnose, failure to restrain	500	Suicide; patient eloped, jumped from bridge; doctor denied liability; insurance company settled
19	50	F	Inpatient	Schizophrenia, paranoid type	Improper treatment, faulty commitment	2,800	Patient alleged overmedication with sedatives, resulting in fall, and hospital failure to file proper form with district attorney
20	12	M	Inpatient	Neurotic depression	Failure to properly diagnose and refer for treatment	2,000	Youth referred to long-term treatment center after inpatient evaluation; while there he hanged himself

Their age range was 22–74 years. Five of the 6 were hospitalized, and 4 were depressed. Three of the 6 cases involved ECT. One patient objected to that form of treatment, 1 died as a result of cardiac arrest more than 2 hours after treatment, and the third committed suicide. The 3 other cases concerned an assault on a third party, a claim of harrassment by the psychiatrist, and an elopement followed by suicide. None of the court cases that have been tried was lost by defendant psychiatrists. Three doctors were absolved of all liability, 1 case was dropped during the trial, and 2 are pending (see table 2).

DISCUSSION

The survey from which these malpractice claims were derived provides no information concerning the

TABLE 2
Summary of 6 Psychiatric Malpractice Actions Moved to Trial

Patient Number	Age	Sex	Hospital Status	Diagnosis	Allegation	Judgment	Remarks
1	74	Male	Inpatient	Involutional depression	No informed consent, fraud, battery	For defendant	ECT case; patient claimed inadequate preparation for treatment, asserted alternative modes were not tried first
2	25	Male	Inpatient, outpatient	Schizophrenia, paranoid type	Failure to restrain	Pending trial	Patient in board and care facility; assaulted and killed passerby in street; suit by family of decedent
3	33	Female	Outpatient	Schizophrenia, paranoid type	Harrassment	Suit dropped in midtrial; for defendant	Patient claimed she was being followed by her doctor and his associates; patient acted as her own attorney
4	22	Male	Inpatient	Organic brain syndrome	Failure to restrain	Pending trial	Patient eloped from hospital, ran in front of a truck and was killed; hospital settled; doctor refused settlement
5	31	Male	Inpatient	Psychotic depression	Failure to restrain	For defendant	Patient drowned while on authorized hospital outing; patient had received ECT
6	62	Male	Inpatient	Involutional melancholia	Wrongful death	For defendant	ECT case, patient with known heart disease had cardiac arrest 2½ hours after ECT

claims experience of nonrespondents. This is a limitation of the technique. It is possible that doctors who have had more unfavorable malpractice experience are reluctant to talk about it. However, the reverse may be true. Many psychiatrists confronted with the threat of suit react with an indignation they are quick to share with both colleagues and their professional society. A personal follow-up of a random sample of members would be required to show that the respondents were not atypical.

Given this reservation, and in the absence of other data, these findings support a contention that psychiatric practice presents a low risk to professional liability insurers. The initial survey showed a rate of 3 claims or notifications of potential claim per 100 psychiatrists per year over a 5-year period. This part of the study shows that more than 54% of these claims were associated with no subsequent legal involvement and an additional 21% stopped short of any settlement or trial. This means that 75% of the claims involved no settlement, judgment, or court costs. The 20 cases that were settled cost the insurance companies a total of about $440,000 over a 5-year period, or about $88,000 per year. The 4 cases that went to trial resulted in no paid judgments.

If the total number of claims is reduced by those which are only threats of suit and legal-investigative procedures, leaving just the cases that were settled or involved the courts, a frequency of "significant" claims, i.e., those which could produce a substantial loss, can be calculated. For the respondent population this rate is .02 for the 5-year period, or .4 claims per 100 psychiatrists per year. This means that in a given year there would appear to be less than 1 chance in 200 for a psychiatrist to subject his or her insurance company to a significant loss.

The patients associated with these potentially more expensive claims had few characteristics that would set them apart from patients encountered in routine practice. They tended to be middle-aged and were diagnosed as depressed or psychotic. Suicide was the most frequent complaint. It was the issue in 6 of the 20 settlements and in 2 of the 6 court cases.

Problems with ECT were not a source of loss for the insurance companies. Although 3 of the 6 court cases involved patients who had received ECT, none resulted in judgments. Only 2 of the patients who received settlements had been treated with ECT, and ECT was not at issue in either case. Of the total of 105 cases of complaint, 9 patients had received ECT, but in only 3 was that form of treatment, as such, in question. In 2 of these cases the defendant psychiatrists were absolved by the courts, and the other case was dropped before any legal involvement.

Given the limitations of the survey, these data support an inference that the actual cost of insuring psychiatric practice in California (a populous state considered to have a high-risk legal climate) was modest during a recent 5-year period. Using a few reasonable assumptions, e.g., an average of $50,000 for legal and court cost per tried case and an average legal-investigative expense of $500 for each unlitigated claim, an estimated mean yearly loss per insured doctor can be calculated. For the psychiatrists who responded to this study, that loss amounted to $108 per year. That figure would cover all paid losses and the estimated claims expense but not the reserves set aside for the 2 untried cases, insurance administrative expense, or profit.

Most psychiatrists have been subject to a sharp increase in the cost of malpractice insurance. The extent to which such increases are justified is not known. Trent (4) reported loss data from APA's program, which show an increasing frequency of claims and larger amounts paid out as that loss experience matures. In the past few months there have been a number of large settlements and judgments against California psychiatrists. They will affect future premiums.

Efforts to respond to increased malpractice costs are already under way. The California legislature has enacted a law that provides a sliding scale of attorney contingent fees and requires periodic payment of larger awards and disclosure of collateral sources of payment in malpractice actions. The National Association of Insurance Commissioners (NAIC) has initiated a nationwide study of closed malpractice claims. Effective this year, that study will specifically identify psychiatric cases and report the nature of the maloccurrence as well as the cost of settlement. It will be some time before the data derived from this study can achieve sufficient credibility to influence the rate structure of psychiatric professional liability insurance. The actual dollar savings from these efforts will remain uncertain for at least a few years. All of the cost of malpractice insurance is eventually borne by the patient. Adequate tort reform and mandated refinement of reported claim and loss data offer the best chance for containment.

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Characteristics and Disposition of Persons Found Not Guilty by Reason of Insanity in New York State, 1971–1976

BY RICHARD A. PASEWARK, PH.D., MARK L. PANTLE, M.A., AND HENRY J. STEADMAN, PH.D.

In order to develop an empirical profile of persons acquitted by reason of insanity from which policy could be developed, the authors studied all acquittals in New York State from 1971–1976. During this period 196 men and 29 women were found not guilty by reason of insanity. In contrast to adults admitted to state correctional facilities, the study group contained proportionately more persons who were white and older. The most frequent crimes for which subjects were tried were homicide, assault, and robbery. Most victims were acquaintances of the defendants. By 1976, 67 persons had been discharged after hospitalization ranging from 1 to 1,235 days. These defendants are still being followed after their return to the community.

PHILOSOPHICAL DISCOURSES on the insanity plea abound in the legal and psychiatric literature. Yet few empirical studies have dealt with the frequency with which the defense is employed, its success rate (1–3), and the characteristics and dispositions of the defendants involved (4–8). Despite the limited data available on the plea of not guilty by reason of insanity, legislators, prosecutors, and defense attorneys express chronic dissatisfaction with whatever statute happens to be contemporary law and exert periodic efforts to alter the law in some fashion. Considering the limited information available on this plea, such efforts must be based on subjective impressions of the manner in which the law operates and its consequences.

The present investigation concerns the characteristics and disposition of persons found not guilty by reason of insanity in New York State. The time frame used was from the effective date of a revised criminal law procedure (9) governing hospitalization of persons acquitted for criminal acts by reason of insanity, September 1, 1971, to June 30, 1976. This report deals with a comprehensive depiction of the defendants' backgrounds, offenses associated with acquittal by reason of insanity, and courses of hospitalization after acquittal. Future reports will focus on how these patients compare both with those acquitted during the last 6 years of the prior statute and with a group of felons convicted of similar offenses from 1971 to 1973.

The insanity defense in New York is basically governed by a modified American Law Institute rule with the irresistible impulse component of that rule deleted. According to statute, "A person is not criminally responsible for conduct if at the time of such conduct, as a result of mental disease or defect, he lacks substantial capacity to know or appreciate either: (a) The nature and consequences of such conduct; or (b) That such conduct was wrong" (10). After acquittal the defendant is automatically committed to an appropriate mental institution until such time as his or her mental state has been restored and he or she no longer poses a threat to self or others.

METHOD

Patient files of the New York State Department of Mental Hygiene were searched to identify all persons found not guilty by reason of insanity during the study period. The files of the Departments of Mental Hygiene, Correctional Services, and Criminal Justice Services were then abstracted to secure the basic data required for the subjects.

All of the inmates admitted to the state's adult correctional institutions during the study period were used as a comparison group. Originally, it had been intended that the comparison groups would comprise those populations of the state who, during the study period and as the result of alleged felonies, had been arrested, indicted, acquitted, and had indictments dismissed. Unfortunately, New York's criminal justice data are not organized to permit the drawing of such comparison populations. Despite some obvious limitations, it was therefore necessary to use adult inmates admitted to the state's correctional facilities as the comparison group. This inmate group included only those persons admitted from 1971 to 1975, since 1976 data were not available.

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RESULTS

Frequency of Successful Pleas

During the nearly 5-year study period 225 individuals were adjudicated as not guilty by reason of insanity. In the 4-month period of 1971 there were 15 cases; in 1972, 25 cases; in 1973, 37 cases; in 1974, 55 cases; in 1975, 61 cases; and in the 6-month period of 1976, 32 cases. This number is considerably more than the 11 successful cases during the 1960s reported by Foster (11) and later by Stone (12). However, Foster's information was found to be incorrect, apparently due to faulty records that were corrected during our investigation. There were, in fact, 53 successful pleas in New York from March 1, 1965, to September 1, 1971.

Because in New York the insanity defense can be raised at any stage during a criminal trial, it was not possible to establish the frequency with which the plea was actually made and thus its success rate. Such a determination would necessitate an examination of the transcripts of all felony trials in the state, a task beyond the scope of our work.

Demographic Characteristics

Of the 225 cases in our study, 196 (87.1%) involved men and 29 (12.9%) women. In contrast, during the period September 1, 1971, to December 31, 1975, 96.3% of the admissions to the state adult correctional facilities were men and 3.7% women, suggesting a possible overrepresentation of women in our study population.

Blacks comprised 60 (26.7%) of the 225 cases; whites, 146 (64.9%); Puerto Ricans, 12 (5.3%); and persons from other ethnic backgrounds, 1 (0.4%). In 6 cases (2.7%), ethnicity was unknown. Of the comparison group, 56.5% were black; 26.9%, white; 16.4%, Puerto Rican; and 0.4%, other. These figures indicate an overrepresentation of whites and an underrepresentation of blacks and Puerto Ricans in our study.

Study subjects ranged in age from 16–77 years, with a mean of 36 years (average age for men was 36 years and for women, 33 years). Distribution of age did not differ significantly between the sexes. The average age of the prison group was 23 years (mean age of 27 years for men and 33 years for women). It should be kept in mind that the prison group represents only those persons admitted to adult correctional facilities in New York. Therefore the ages of the entire population imprisoned in New York, both juvenile and adult, would be substantially lower.

In general, our study group was much older than a general criminal population. Whereas criminal activity is typically regarded as "a young person's game" and individuals in the 15–30 age bracket represent a large proportion of all persons arrested and convicted, 62% of our study group were more than 30 years old and 28% were over age 40.

Hospitalization and Criminal Histories

Among the 196 male patients, 109 (56%) had had no psychiatric hospitalizations in a New York facility before the arrest date for the crime of concern. Eighty-seven had been hospitalized and accounted for a total of 227 separate hospitalizations; type of hospitalization was known in 80 cases. Fifty had prearrest hospitalizations that were exclusively civil. These 126 hospitalizations ranged from 19 subjects with 1 prior civil hospitalization to 1 individual with 14 such hospitalizations. Twelve individuals had been hospitalized under solely criminal code procedures for a total of 19 hospitalizations. The remaining 18 subjects has been hospitalized under both civil and criminal procedures. They totaled 44 civil and 38 criminal hospitalizations.

Of the 29 female subjects, 21 (72%) had no hospitalizations before their arrest. Seven had been hospitalized previously under civil statutes for a total of 15 hospitalizations. One had been hospitalized under civil statutes on 5 occasions and twice under criminal provisions.

One hundred twenty-five (56%) of the 225 study subjects had no prior arrest record. Those with prior records included 5 women (17% of the women) and 95 men (48% of the men). The entire male group, including those without previous arrests, averaged 2.4 arrests while the comparable mean for women was 0.4. For the 95 men with previous arrests, the mean number of arrests was 5.0 and ranged from 18 men with 1 arrest to 1 defendant with 21 arrests. The five women with prior arrests averaged 2.4 arrests.

The 100 persons previously arrested produced 492 arrests. Of these, 151 (31%) were against persons, including 9 for murder, 1 for negligent homicide, 69 for assault, 31 for robbery, 19 for sex offenses, and 22 others. Property offenses accounted for 152 previous arrests (31%). Various minor offenses such as disorderly conduct, criminal mischief, and promoting gambling resulted in 116 (23%) of the 492 total arrests. The remainder were drug related (N=40, or 8%) and weapon or burglar tool possession (N=33, or 7%).

Acquitted Offenses and Victims

Table 1 provides the distribution for the most serious crime for which the study subjects were tried. It is apparent that crimes of violence or potential violence against persons and, most specifically, some form of homicide represented the most frequent charge. Murder and manslaughter accounted for 133 (59%) of all criminal charges, while assault, rape, and robbery accounted for another 53 (24%) of the cases. It is striking that 90% of the women were charged with some type of homicide.

A comparison with the criminal charges against the prison admission group is relatively meaningless, since the study population was almost uniformly tried for the crime for which they were arrested, but crimes for

[1]A table listing the frequency of successful insanity pleas and the rates of the plea, by county, is available from Dr. Steadman on request.

TABLE 1
The Most Serious Crime for Which the Study Subjects Were Tried

Crime	Men (N=196) N	Men (N=196) %	Women (N=29) N	Women (N=29) %	Total (N=225) N	Total (N=225) %
Murder	96	49.0	24	82.7	120	53.3
Manslaughter	11	5.6	2	6.9	13	5.8
Robbery	15	7.6	1	3.4	16	7.1
Assault	32	16.3	0	0.0	32	14.2
Kidnapping	1	0.5	0	0.0	1	0.4
Reckless endangerment	4	2.0	0	0.0	4	1.8
Menacing	1	0.5	0	0.0	1	0.4
Rape	5	2.6	0	0.0	5	2.2
Sexual, other	5	2.6	0	0.0	5	2.2
Endangerment of a child's welfare	1	0.5	0	0.0	1	0.4
Arson	12	6.1	2	6.9	14	6.2
Possession of a weapon	1	0.5	0	0.0	1	0.4
Burglary	4	2.0	0	0.0	4	1.8
Escape, absconding	3	1.5	0	0.0	3	1.3
Possessing forged papers	1	0.5	0	0.0	1	0.4
Criminal mischief	1	0.5	0	0.0	1	0.4
Motor vehicle violation	1	0.5	0	0.0	1	0.4
Criminal sale of controlled substances	2	1.0	0	0.0	2	0.9

which the inmate group were tried and convicted typically represented the termination of a plea-bargaining process in which the initial charge was often drastically reduced.

The vast majority of cases for both sexes (83.5%) involved but a single victim; 6.7% involved 2 victims; 1.8%, 3 victims; 0.4%, 5 victims; in 7.6% of the cases there was no victim. Of the 163 cases in which hospital records noted whether there was a victim, 17 (10%) involved crimes having no victim, such as drug sales and arson involving an abandoned building. In 103 cases (63%) the victim was known to the patient before the crime. In this category, members of the patient's family constituted the largest target group. Twenty-one (13%) involved their children; 19 (12%), their parents; 16 (10%), their spouses; 5 (3%), other relatives; and 5 (3%), in-laws. By far, the most frequent victims of female subjects were their children (16, or 57%); most of these cases involved infanticide. Other victims known to the patients included well-known acquaintances (20, or 12%), boyfriends, girlfriends, and/or members of their family (4, or 2%), coworkers (3, or 2%), employers (2, or 1%), and casual acquaintances (8, or 5%). Thirty-two of the victims (19.6%) were strangers. Law enforcement officers represented an identifiable target group (11, or 7%). In all but one case the officer became a victim while pursuing his official duties. However, one case involved the slaying of an officer by a fellow patrolman.

County of Trial

The rate of successful pleas of not guilty by reason of insanity varied widely among counties. In 25 counties there were no successful pleas. A 3 × 2 tabulation of counties by 1970 populations of 1) 0–100,000 persons, 2) 100,000–500,000, or 3) over 500,000 and by rates of successful pleas of 1) .00–.20 or 2) above .20 per 10,000 population revealed no significant association between county population and rate of successful pleas. In this regard, for example, the highest rates were found in Albany (.63; population of 286,742), Steuben (.40; 99,546), Erie (.35; 1,113,491), and Genesee (.34; 58,722). In like manner, New York and Kings counties, both heavily populated areas of New York City, had rates of .25 and .05, respectively.[1] The vast difference between Albany County and the rest of New York should be noted in the context of the study of the increased frequency of acquittals due to insanity reported by Grunberg and colleagues (13). Albany County is so atypical that any inferences drawn from these data, particularly when they are as global as these investigators', should not be generalized to the rest of New York.

Unfortunately, the manner in which New York justice data are compiled does not permit the computation of rates of pleas of not guilty by reason of insanity based on arrest, indictment, acquittal, and dismissal data for each county. If, however, it can be assumed that the typical pattern of urban, suburban, and rural crime prevails in New York, a reasonable inference can be drawn that high rates of the plea do not necessarily occur in counties with high rates of arrest and indictment. Again, unfortunately, the mode of compilation of criminal justice data in New York does not permit a direct analysis in a manner comparable to that of Pasewark and Lanthorn (4), who found that the frequency of the insanity plea in Wyoming was unrelated to population magnitude or density but was inversely related to the rate of arrest and indictment within a county. Thus the more "brisk" the criminal justice business in a county, the less likely it was that the plea of not guilty by reason of insanity would be entered. In addition, Pasewark and Lanthorn found the plea to be more powerful than commonly supposed in that a larger proportion of defendants entering this plea had their indictments dismissed than did persons not entering the plea.

The variable rate of successful pleas among New York counties suggests the possibility that the appropriate legal statutes concerning the plea are not applied uniformly throughout the state. Although current data do not permit the determination of reasons for the differential rate observed, a number of considerations warranting investigation include differences in arrest and indictment rates; the presence of judges, prosecutors, and/or defense counselors from given counties who have a strong orientation toward a psychodynamic explanation of behavior; and the presence of articulate groups of psychiatrists who serve as expert witnesses in particular counties. In any event, the differential rate of successful pleas among counties should raise questions as to the wisdom of altering the statutory language governing the plea if the goal of such change is to ensure uniform and consistent application of the law.

TABLE 2
Primary Diagnosis of the Study Subjects on Hospitalization

Diagnosis	Men (N=196)		Women (N=29)		Total (N=225)	
	N	%	N	%	N	%
Psychosis						
Alcohol	4	2.0	0	0.0	4	1.8
Chronic brain syndrome	5	2.6	0	0.0	5	2.2
Drug	2	1.0	1	3.4	3	1.3
Schizophrenia, paranoid type	73	37.2	7	24.1	80	35.6
Schizophrenia, undifferentiated type	19	9.7	7	24.1	26	11.6
Schizophrenia, other types	18	9.2	4	13.8	22	9.8
Affective	5	2.6	0	0.0	5	2.2
Other	7	3.6	3	10.3	10	4.4
Total	133	67.9	22	75.9	155	68.9
Neurosis	6	3.1	1	3.4	7	3.1
Personality disorder						
Antisocial	12	6.1	0	0.0	12	5.3
Explosive	3	1.5	0	0.0	3	1.3
Inadequate	1	0.5	1	3.4	2	0.9
Other	4	2.0	3	10.3	7	3.1
Total	20	10.2	4	13.7	24	10.7
Mental retardation	5	2.6	0	0.0	5	2.2
Other						
Alcoholism	4	2.0	0	0.0	4	1.8
Drug dependency	2	1.0	0	0.0	2	0.9
Transient personality disorder	1	0.5	0	0.0	1	0.4
Adolescent disorder	3	1.5	1	3.4	4	1.8
Total	10	5.1	1	3.4	11	4.9
Nonpsychotic or no disorder	8	4.1	1	3.4	9	4.0
Unknown or deferred	14	6.2	0	0.0	14	6.2

TABLE 3
Length of Hospitalization of Persons Discharged (N=67)

Crime	Men (N=58)				Women (N=9)			
		Days Hospitalized				Days Hospitalized		
	N	Range	Mean	Median	N	Range	Mean	Median
Murder	23	1-1235	278.48	218.00	8	56-621	245.62	304.00
Manslaughter	2	143-160	151.50	151.00	0			
Robbery	3	14-160	104.67	140.00	1		62.00	62.00
Assault	14	33-639	332.29	371.00	0			
Reckless endangerment	2	78-91	84.50	84.00	0			
Burglary	2	154-201	177.50	177.00	0			
Arson	3	45-141	93.33	94.00	0			
Sexual abuse	3	36-614	256.33	199.00	0			
Possession of a weapon	1		863.00	863.00	0			
Driving while intoxicated	1		7.00	7.00	0			
Endangerment of a child's welfare	1		322.00	322.00	0			
Criminal mischief	1		71.00	71.00	0			
Escape	1		39.00	39.00	0			
Absconding	1		94.00	94.00	0			

Hospitalization After Acquittal

Diagnoses associated with the 330.20 hospitalizations were available in all but 14 of the 225 cases (see table 2). It should be remembered that a substantial period of time typically intervened between the patient's arrest and his or her enforced hospitalization after acquittal. Hence it is quite probable that in some cases, overt symptoms present at the time of the criminal act had abated.

Of these 211 patients whose diagnosis was known, 155 (69%) were given the diagnosis of psychosis; 7 (3%), neurosis; 24 (10.7%), personality disorder; 5 (2%), mental retardation; and 9 (4%), nonpsychotic or having no disorder. Of the 155 persons categorized as psychotic, 128 were schizophrenic and 80 of these were of a paranoid type.

On June 30, 1976, the cut-off date for the research, 133 patients (59%) remained hospitalized; 67 (30%) had received complete discharges including 2 from conditional leave status, 3 from absent without leave status, and 3 from escape status. Two (0.9%) were in a family care program; 8 (3.5%) were on conditional release; and 1 (0.4%) was on leave over 60 days. Three

(1.3%) had escaped, and 2 (.9%) were absent without leave. Nine patients (4%) were deceased, a fairly high mortality rate for persons of this age group. Of these, 5 were known to have committed suicide.

Table 3 presents data on the length of hospitalization, by type of offense, for the 67 persons discharged by June 30, 1976. Hospitalization ranged from 1 to 1,235 days (both of these cases involved murder). Length of hospitalization did not seem specifically related to the severity of the offense, as would be expected since the criteria for discharge are recovery and lack of dangerousness rather than punishment for the criminal act.

Work is now under way to examine the arrest and rehospitalization rates of these persons acquitted by reason of insanity. Preliminary indications suggest that they are less often arrested than comparable groups of felons and felony defendants incompetent to stand trial. The next phase of the research will deal with crucial clinical and policy questions.

COMMENT

A number of the findings reported here serve to point out the need for empirical investigation into areas rampant with assumptions built on assumptions. Contrary to much popular opinion, defendants acquitted by reason of insanity are not all bizarre murderers. Only a slight majority (53%) of those acquitted in New York were murderers. More importantly, in all types of offenses the victims for the most part were acquaintances. The popular conception that acquittals due to insanity return persons to the street to kill more strangers is simply incorrect. Most of the prior violence was directed at family and friends, and it is probable that any subsequent violence would also be directed at these two groups.

Some of our findings were surprising. We had anticipated that the study group would include individuals with many pretrial psychiatric hospitalizations and only a few arrests. Instead, 56% of the men and 72% of the women had no prior psychiatric hospitalization; 95 men (44%) had at least 1 previous arrest, as did 5 women (17%). Further, these 100 individuals had a total of 492 arrests including 152 arrests for such property offenses as burglary (N=55), various forms of larceny (N=70), possession of stolen property (N=16), and 11 other such offenses. These prehospitalization arrests lead us to question the belief that all cases of insanity pleas in this particular population represent individuals who, as a result of or during a state of mental disorder, committed the crime of concern. Instead, a reasonable alternative thesis appears to be that this group represents at least two distinct subcategories. The first subgroup comprises those typically regarded as subjects for the insanity plea. These are persons whose act represents a criminal behavior associated directly with a mental disorder. The second subgroup might, instead, be representative of a larger criminal population that, like any other occupational group, contains a certain number of mentally ill individuals.

We also tend to believe that within the population using the insanity plea, there are other subgroups for which the classical classification of mental illness seems a misnomer. Among this group we include mothers who kill their children and an assorted category of persons whom we feel cannot be classified as mentally ill.

In our study population 14 of the 29 women killed their children. Although some in this group had obvious psychotic symptoms, from psychiatric reports there would seem to be many more whose basic condition was one of inadequacy as mothers and homemakers. Essentially, we should suspect that because of the mores of society concerning motherhood, there is a tendency for citizens, including psychiatrists and judges, to label hostility against children as a reflection of aberrant thought processes or behavior. Rather than accepting the fact that the child is that person with whom the mother probably has the most interpersonal contact and is therefore the most likely target for the mother's hostility, we instead view infanticide as abnormal and thus preserve our illusions of motherhood and the mother-child relationship.

Another subgroup of questionable insane status in our population appears to be "persons of respectability" for whom citizens can feel considerable empathy. This group included a professional who, hounded by racketeers for debts resulting from compulsive gambling losses, committed robbery and a bumbling, uncertain middle-socioeconomic-class youth who, rebuffed by female contemporaries, committed rape to determine whether he could, in fact, have an erection and ejaculation with a woman.

These latter classes of individuals, as well as the differential rate of successful insanity pleas among counties, cause us to question seriously whether the particular language of a given statute governing the insanity plea is the deciding factor in whether a person is so adjudicated. It seems more reasonable to us that more humane, less legalistic variables are involved and that, whatever the law might be, these factors will continue to operate.

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Psychiatry and the Rhetoric of Models

BY BEN BURSTEN, M.D.

The term "medical model" has been increasingly used in discussions about the future course of psychiatry. The author examines various types of models and suggests that "medical model" is a rhetorical device which tends to obscure many important issues. He suggests that psychiatry avoid overrestricting its area of expertise.

DURING THE PAST three decades the profession of psychiatry has traveled so far and so wide that we have been repeatedly impelled to reflect on who we are and what we are or should be doing. This identity crisis is not unique to psychiatry, of course; identity crises seem to be a hallmark of the rapidly changing times in which we live.

Recent discussions about our "proper" identity have to a great extent centered around the issue of the medical model. Several writers (1-4) have urged that we embrace or return to the medical model. For example, Hackett (4) stated, "I predict that psychiatrists, should they continue winging their way through the heady atmosphere of nonmedical conceptual and therapeutic models, will soon find themselves an endangered species."

On the other hand, writers such as Spiegel (5) have proposed quite different models. In his 1975 Presidential Address, Spiegel suggested a model based on helping individuals in difficult human situations—a sort of decision-making model that he termed "managing risky choices." Brown (6) predicted that well into the next century, psychiatrists will be concerned not only with schizophrenic and depressed people but also with those who have "problems in living." Engel (7), while advocating a medical model, called for a "new medical model"—one that might or might not be embraced by those who seek psychiatry's "return" to the medical model.

I do not propose to resolve psychiatry's "identity crisis" in this paper. Rather, my chief purpose is to point out how the use of the term "medical model" may be so confusing and ambiguous that it interferes with rational discussion of the very real questions before our profession.

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MODELS

The term "model" is used in two related but different ways. In the epistemological sense, "model" describes a way of thinking, a device employed to guide us in our approach to a problem. It gives us procedural rules or simplifications that make complex situations more comprehensible to us. I shall use the term "conceptual model" when referring to the epistemological sense. Conceptual models are chosen, wittingly or unwittingly, by the thinker. There is no absolute epistemology that enables us to get at the truth. As Conant (8) noted, "A scientific theory is not even the first approximation to a map of the structure of the universe, or a part of it; it is *policy*—an economical and fruitful guide to action by scientific investigations" (italics added). The informal rules of admissibility of data, procedure, viewpoint, acceptance of previous viewpoints, and so forth (many of which have been called "models") are part of this policy. Engel (7) is quite correct in stating that "a model is nothing more than a belief system" whether that model be the hypothetico-deductive method, mind-body dualism, systems theory, or free will and human choice. This is not to say that conceptual models cannot be useful. Often they are very useful, but sometimes they may lead us into unproductive channels. Although conceptual models have been important in the development of the physical sciences, Stent (9) pointed out that the sciences which aim to understand man have depended even more heavily on epistemological support.

There are various types of conceptual models. According to Langer's use of the term (10), "A model is an ad hoc construction designed to make easier the understanding of complex abstract theoretical propositions through the use of more easily encompassable *pictorial or verbal analogies*" (italics added). An example would be Freud's physics model of psychic energy or his military model of competing forces in mental conflict. Despite the fact that many of Freud's models were reified by some psychoanalysts, Gedo and Goldberg (11) make it clear that they are models—analogies—used in order to make the complexities of mental functioning more comprehensible. Another example of this kind of conceptual model is the computer model of the brain. Using this model, we do not imply that the brain is a computer but rather that by this simplified analogy, we may get some insight into the way the brain works. These models are selective in what they reproduce, as Langer has pointed out, and we

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may have to employ several different models to grasp the situation we are trying to comprehend.

In another vein, there are several models for conceptualizing the etiology of mental illness. Lazare (12) has outlined four such models—the medical (I would prefer the word "biological"), the psychological (really the developmental), the behavioral, and the social. Instead of being analogies, the model aspects of these concepts of etiology are rules of procedure. For example, the biological model says, in essence, "Whenever you see mental illness, look to biology for the significant etiological data." This can lead circularly to the position advocated by Ludwig and Othmer (3), wherein the conceptual model of disease (illness) is rooted in biology. Here the rule of procedure becomes, "Absent a biological etiology, do not call the condition an illness."

Minuchin and associates (13) illustrated still a different dimension of conceptual models for thinking about causation. Instead of considering biological, psychological, or social models with their various implicit assumptions about what "really" causes the condition in question, these authors considered a model of causation itself. They rejected the exclusive use of the linear model in which the psychosomatic illness "is seen as contained within the individual" and is produced by antecedent events in the person's life. By embracing an "open systems model," they have seen pathology in the family system whose communicative feedback mechanisms maintain the status quo of illness. It is important to recognize that the authors did not deny the psychosomatic illness per se or its linear antecedent causes, such as physiological vulnerability or the effect of stress on the designated patient, but that they maintained that using only that model of causation results in incomplete and less fruitful understanding than using the systems model.

Another type of conceptual model dictating rules of procedure is the hard versus soft controversy. Hard approaches are sometimes referred to as "scientific," "empirical," and "quantifiable." They count as data only those items which are observable by others and are recordable. They discount introspective data such as meanings, feelings, and hopes, which are directly observed only by the patient. However, some of these introspective soft data can be converted to hard data if the introspections are written down in response to printed questions, such as in the Minnesota Multiphasic Personality Inventory. In this case, however, no inference need be made about how the patient feels; the data are those which he or she reports.

Finally, we might briefly consider the conceptual model of illness itself. This is perhaps less a model and more a matter of definition. Some (1, 3) include in the category of disease only that which has a biological dysfunction. While social influences may be important, they must interact with a biological weakness or produce a biological aberration in order for the event to be called "disease." As Ludwig and Othmer (3) stated, "When no sustained biological dysfunction can be demonstrated or presumed, no disease can be said to exist." An alternative view, as expressed by Fabrega (14), is that illness is a social convention indexed by certain negatively toned changes in behavior that society validates as sick. Friedson (15) has presented an extensive discussion of this point of view. A good example of the definition of illness as a social convention was the American Psychiatric Association's decision to no longer classify homosexuality as a psychiatric diagnosis. If, in the near future, we were to find that circulating maternal hormones so influence the organization of the developing brain of the fetus that a certain area of this brain maintains dominance over another area and thus enhances same-sex attraction, we would have the biological underpinning for homosexuality. However, would this clarify the question of homosexuality as a disease any more than cerebral dominant factors may influence left-handedness without defining disease status?

The criteria for illness that Parsons (16) proposed included that the deviance does not arise by choice and is essentially beyond the patient's control and that what is wrong can be understood rationally. While Parsons' definitions of illness and the sick role are seminal in sociological thought, they have been revised, developed, and expanded by others such as Friedson (15). What these writers seem to agree on is that "illness" as defined by our society is a natural process (even if bad luck gave one cholera, the cholera itself is a natural process) and the patient cannot choose to be rid of it. This lack of choice rests on a causal conceptual model that includes elements of both psychological determinism and free will. Other deviations, such as sin or crime, contain larger measures of choice.

Beyond these various conceptual models (and there are many others) we find a second use of the term "model." This is the usage in the social sense; it refers to the process of development or socialization and speaks to the question, "Whom does one pattern oneself after?" When referring to the social sense, I shall use the term "role model." While conceptual models refer to the way we think, role models refer to the way we act. Clearly, the way we act is in large part predicated on the way we think. And just as clearly, the way we think, the way we conceptualize a problem, is predicated on who we are in terms of social role, how we were trained, what depends on our actions, and how those whom we pattern ourselves after would analyze the situation. However, there are also differences. We may understand a situation in terms of a conceptual model but take no action because we feel that it lies outside our role. Or we may take on certain attributes of our role model, such as wearing a white coat, that have little direct bearing on the conceptual models we employ.

As with the conceptual model, the role model is also embraced, chosen. However, in addition, it is more dependent on validation by others. If one embraces the supernatural model of causation (a conceptual

model), one may or may not find others in agreement, but one is relatively free to continue to think with that model.[1] However, if one embraces the role model of a minister, one needs the validation of people who are willing to be ministered to.

THE MEDICAL MODEL

I do not believe that there is a clear-cut medical model to which we can either return or aspire. Like the "good old days of yesteryear," the medical model of old never really existed, except in the nostalgia of those who would like a simple and more comfortable life. Nonetheless, the term has increasingly been used in the rhetoric of the last 20 years, as if we knew what we meant when we used it. The medical model is a conglomerate of several of the types of models outlined above, but the various people who employ the term refer to different types of models. Therefore the term is ambiguous, and unless one listens carefully, one is apt to miss the hidden meanings (hidden models). As such, it can become a code word, like "the American way," with pejorative and loyalty connotations masquerading as philosophy. As we shall see, for a variety of reasons most psychiatrists probably want to be regarded as physicians and may be induced to embrace a medical model without clearly knowing what they are embracing.

Freud started as a physician, and his early hope was to root his psychology in biology—not in the analog model sense but in actuality. As his thinking evolved, he had to resort more and more to analogies. He employed the conceptual models of physics and sometimes military encounters in order to grasp what he was primarily interested in—mental functioning. While the psychoanalytic model of etiology has always left the door open for biology, it has been largely developmental, assuming what I term "the average expectable brain."[2] For those who exclude from their definition of disease those situations without biological dysfunction, psychoanalysis does not deal primarily with illness. If one takes the position that the medical model is to deal with illness, psychoanalysis may be defined outside the medical model. If, however, one subscribes to the definition of illness that rests on responsibility and the ability to choose to be well, it is clear that psychoanalysis, with its deterministic thrust, deals with illness. Even when he advocated lay analysis, Freud had in mind those aspects of the medical role model which treated patients. However, he discarded those aspects of the model which mandated that one needs a medical degree and other trappings of the medical role.

To people such as Erikson (17), however, the psychoanalytic reliance on the illness concept (defined not by biology but by the ability to choose) leads to role confusions for both doctor and patient. The latter part of the 1950s saw the emergence of "therapeutic" communities in hospitals, which began to place more responsibility on the patients themselves. There was a shift in many hospital programs from a causal concept based on a model of patient helplessness to one based on relative free will—a shift from illness to a deviancy more like crime. Along with this shift in conceptual models, as Hoffman and Singer (18) have noted, came calls for a shift in role models—away from the model of traditional physician authority and prestige.

It is of interest that these calls for moving away from the medical model were not aimed exclusively at those who held biological etiological concepts or who defined illness as biological dysfunction; they were also aimed at psychoanalysts who embraced a developmental psychological conceptual model and who defined illness as an inability to choose.

In the early 1960s Szasz (19) declared that mental illness is a myth. The models that entered into Szasz's thinking in the ensuing years are many and complex; they have been well analyzed by Moore (20). At the risk of oversimplifying, it can be said that Szasz combined the biological definition of illness with the causal conceptual model of free choice. On both of these grounds, then, he was able to exclude most behavioral deviations from the realm of illness. Building on this, he argued that the authority aspects of the medical role model are inappropriate and that the legal powers given to physicians as social validation of their roles as protectors of the public's health are misapplied when dealing with problems of behavior.

In the turbulent 1960s the radical psychiatrists turned vigorously against the medical model. The medical model they opposed was not so clearly based on the conceptual model of choice versus linear deterministic causality. Those who conceptualized causality at all worked with the dimension of etiology, and they found etiology to lie in the "sick society" rather than in biology or development. However, as Talbott (21) has pointed out, much of their attack was aimed against the psychiatrist's professional prestige, economic well-being, and authority and control—especially over the mental health workers. This, then, was in large measure an attack against the role model aspects of medicine.

The attack on the psychiatrist's preeminent position in the field of mental health has come from other quarters as well. The schools of psychotherapy that Karasu (22) termed "experiential" arose largely from the work of both psychiatrists and psychologists. These therapies share the concept of real and nonauthoritarian relationships. Their etiological model is essentially social, with alienation being a prime factor in preventing personal growth and realization of one's potential. They generally do not embrace an illness model and often dissociate themselves from the physi-

[1] I do not imply here that one's thought models are independent of a social matrix; I am talking about relative degrees of social validation.

[2] Bursten B: Brain, objective-relations, and character (unpublished paper).

cian role. Nonetheless, they come under the social and financial rubric of mental health.

Although behavioral therapies are practiced by medically trained and nonmedical therapists alike, they seem to have found more favor with those who advocate a return to the medical model. It is interesting and instructive to inquire why this is so, inasmuch as they are essentially psychotherapies. The model of etiology is not primarily biological, with the exception, perhaps, of the physiological responses treated with biofeedback. The behavioral model rests heavily on the concept of a learning etiology (12, 22)—usually the domain of the educator rather than the physician. It is probably because the behaviors that these practitioners are concerned with are considered "illness" by our society's definition. Ludwig and Othmer's list of bona fide psychiatric diseases (3) includes anxiety, phobic reactions, alcoholism, and obesity—all of which may be treated with behavioral techniques. Another reason that these techniques may be considered legitimately medical is that they rest on hard data that may be plotted on a graph and quantified. Further, the role of the behaviorist vis-à-vis his or her patient is generally more authoritarian than that of other psychotherapists—the benevolent authority and the prescriptive aspects of the medical role model are used.

While much of the rhetoric of the 1950s and 1960s urged abandonment of the medical model, even though different authors meant different things when using the term, the rhetoric of the mid-1970s has advocated a return. Why is this so? For one thing, the biological frontiers of our profession, so long impenetrable, have opened up with many exciting discoveries and theories. For another, we are living in a time when technology and hard data are increasingly valued, and despite the fact that we decry the impersonality in our society, we continue to revere numbers, be they Social Security numbers or correlation coefficients. For those who use the term "medical model" to refer to biological etiology, biological definition of illness, and the hard data model, current social trends give important validation.

Other trends in our society give impetus to embracing the medical model, although the definition of the term is more on a role than conceptual model basis. In the first place, we have become considerably threatened by therapists who do not have the M.D. degree. Secondly, and strongly related to the first point, the economic picture has changed. In the 1960s there was money for innovative social approaches, and the rhetoric of the times was tailored to the monetary sources. Now much of that money has dried up, and there is talk of a national health plan, which means physicians and medical models in order to tap this economic resource. Similarly, the guidelines used by many insurance companies make the medical role model useful. However, while this role model depends on the concept of illness, it does not necessarily depend on biology or hard data models. A third trend is the new look in health care distribution. There is a strong emphasis (with funds for training) on primary rather than specialized care. Psychiatry has not been defined as a primary care enterprise, but psychiatrists can play an important role as consultants working with the psychiatric aspects of primary care situations. Here the medical model is largely role-based. Whatever one's conceptual models (so long as they do not preclude the concept of illness), we are enjoined by some to take them into the medical clinics and on the general hospital wards. This medical model does not need to imply a biological definition of illness or a hard-data conceptual model. Psychiatrists may work comfortably with behavioral, developmental, and social conceptual models, with linear or systems models of causality, and any analog models we find convenient, so long as we do it in an indisputably medical setting.

There is another, more subtle social trend that impels the rhetoric of the medical model. After the turbulence and permissiveness of the 1960s, our society is retrenching. There are calls for "return" in many areas. Often this return implies greater structure and an increase in the weight of traditional authority. In the role model of medicine, the psychiatrist is cast as offering both a great tradition and firm benevolent authority.

Is there *a* medical model? I do not believe there is. There are many social reasons impelling us to identify ourselves as physicians. There are, of course, historical reasons as well. The term "medical model" becomes rhetoric when we embrace it for these social and historical reasons without there being a consensus on the definitions and models that underline it. In the process, we unwittingly and often uncritically embrace concepts that we might question were they made explicit. Even the role model of the physician is blurred. While we physicians deal with illness, we may disagree on how we define it. We also deal with more than illness. The traditional physician in the "good old days" was counselor, advisor, teacher, and sometimes the voice of moral authority. The heavy medical reliance on hard data has not been characteristic of physicians throughout history; indeed, talking with patients to gather data was probably more characteristic of the old medical role than the one to which we are supposedly to return. Few would dispute that the plastic surgeon is a physician; yet when he or she does cosmetic surgery, the medical model is extended beyond disease. The best pediatricians are as interested in promoting growth as in curing illness.

Siegler and Osmond (1) have described three types of medical models: the clinical, the public health, and the scientific (investigator) models. The roles (and hence role models) of these three vary and at times may be antagonistic to each other. All too often, in medical schools future clinicians are taught by scientific investigator role models, while public health is relegated to a peripheral position in the value system.

If it is impossible to define a single medical model on the basis of the conceptual models we employ and the role models we use as patterns, our difficulties are

compounded when we consider the role of other professions in health delivery in our country. Optometrists, dentists, podiatrists, and osteopaths all treat what is considered illness—indeed, even biological illness. Are we to say, then, that even the M.D. degree is not defined as a requisite for the medical model?

PROPOSALS

I propose, therefore, that we abandon the term "medical model" because it is ambiguous, and like other rhetorical devices, it represents a social and political thrust rather than a clear-cut conceptual or role model. As Langer (10) said, models should simplify and clarify; the term "medical model" obscures. Gedo and Goldberg (11) reminded us that we often must use several different models in order to understand complex situations; the call for a return to the medical model is a call for allegiance.

If we abandon the medical model rhetoric, we are still left with the problems and disagreements that underlie the discussion. I propose that we talk about each of the various true models that I have described above so that others may know more precisely what conceptual vantage points we are employing. We must identify the conceptual models we use and separate them from the role models we advocate. We seem to be suffering an identity crisis, and I am quite content to throw my lot in with medicine; I am a psychiatrically trained physician (role). This need not define my conceptual models. Indeed, if we adopt narrow loyalties to conceptual models, we will restrict our response repertoire. This can lead, for example, to the overprescription of antianxiety medications because we cannot comfortably shift to a social model of illness causation. Our repertoire may be dictated by our version of the medical role model: we may overprescribe antianxiety medications because "doctors prescribe medicine" and "I'm going to act the way doctors do."

More importantly, embracing this role will not insulate psychiatry from the current trends in health care. Nonphysicians do biological treatment, as I indicated above. Occupational therapists, art therapists, psychologists, and social workers all treat, and as more of the dollar goes for health care, they will increasingly embrace the illness concept.

There is little difficulty in distinguishing us from optometrists, dentists, podiatrists, and general osteopaths. Some would bolster our identity and attempt to secure our role by separating us from the occupational and art therapists, the psychologists, and the social workers. The distinguishing feature would be to define disease as biological dysfunction. But there is no safety here. Nurses are increasingly seeking independent practice and are being given the legal right to dispense medication. Doctors of Pharmacy are dispensing medication and sometimes are willing to do so only if they are the "primary therapists." Psychologists, too, are asking for training and authority to prescribe and are conducting research on the biology of behavior. Blurred roles are increasingly becoming a feature of health delivery, and our identity as physicians will not necessarily ensure that the care assignments and the money that accompanies such assignments will go to us.

Hackett (4) has advocated our return to the general hospital: "Unless we are at home in the medical setting we are homeless." However, we are not the only inhabitants of that home. Nurse clinical specialists are training to do liaison work. In addition, psychologists are currently asking for hospital privileges and are increasingly employed by departments of pediatrics and family practice to do the kind of work we psychiatrists feel we should do. This is partly due to economic reasons; at present they often do the work more cheaply. History has shown that this type of economy is only temporary; the price rises as professional status becomes more secure. However, the liaison "home" is not one that we psychiatrists can claim is ours.

What I propose, then, beyond dropping the medical model rhetoric, is that we do not limit our capacities by trying to retreat into the shelter of a specialty distinct from all challengers. It simply will not work, and we may become so precious and specialized that there will be little demand for what we do. If we lose our expertise in the broad aspects of the human condition, we will be pushed to the periphery of the health delivery system where we will become specialized adjuncts to those other professionals who set policies for hospitals, clinics, legal matters, etc. Instead, we should make a specialty of being able to weave all of the factors together in a holistic fashion. We should be able to deal with thought and behavior from a biological, developmental, behavioral, and social standpoint. In order to do this, we must retain the ability to embrace a variety of conceptual models and to move from one to the other as the situation seems to demand. It is an enormous and difficult task—a task well suited to our aspiration as a profession.

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TOPICAL PAPERS: Biological Correlates of Some Psychiatric Disorders

Urinary MHPG Excretion in Minimal Brain Dysfunction and Its Modification by *d*-Amphetamine

BY WALID O. SHEKIM, M.D., HAROUTUNE DEKIRMENJIAN, PH.D., AND JAMES L. CHAPEL, M.D.

The authors studied the excretion of 3-methoxy-4-hydroxyphenlyglycol (MHPG) in 15 hyperactive boys and 13 controls. They further examined soft neurologic signs and clinical drug response to d*-amphetamine administration for two weeks in the hyperactive boys. MHPG excretion was significantly lower in the hyperactive boys than in the controls.* d*-Amphetamine decreased MHPG excretion significantly in the drug responders only. Pretreatment MHPG excretion did not predict clinical drug response. The responders had more soft neurologic signs than the nonresponders. Furthermore, soft neurologic signs were not related to pretreatment MHPG levels.*

IT IS GENERALLY recognized that the diagnostic category of minimal brain dysfunction (MBD), or hyperactive child syndrome, consists of heterogeneous groups (1, 2). Etiologies that are being considered include maturational lag (3), brain damage at birth or early infancy (4), a disorder of the arousal nervous system (5), and a disorder in the metabolism of one or all of the monoamines (1). It has been proposed that some of these disorders may result from familial-genetic predisposition (1, 2) and that the genetic component of the syndrome may be different in different groups (6).

The treatment of choice for MBD with hyperactivity has been *d*-amphetamine or methylphenidate (1, 2). In a majority of MBD children, these medications improve the symptoms of hyperactivity, impulsivity, and short attention span (1–4). The mechanism underlying the therapeutic action of *d*-amphetamine is unknown, but it is often called "paradoxical" in view of the calming effect this stimulant has on the behavior of hyperactive children.

Some 35% of hyperactive children fail to improve on *d*-amphetamine (7). Reasons for such "failures" may include misdiagnosis, inadequate plasma and tissue levels of the drug, individual differences in metabolism of the stimulants, and/or medication noncompliance. Another possibility is that the syndrome of MBD may consist of several biologically different entities which may or may not respond to stimulants. It has been suggested that the investigation of organic and genetic factors as they relate to treatment response in hyperactive children may clarify the identification of subgroups of MBD (2).

In an initial pilot study (8, 9), we presented data that suggested a specific relationship between central nervous system norepinephrine (NE) metabolism and MBD. We found that the basal level of 3-methoxy-4-hydroxyphenylglycol (MHPG), the main metabolite of CNS NE, was lower in 7 hyperactive boys than in 12 controls and that there was a further decrease when *d*-amphetamine, 0.5 mg/kg body weight, was given for 2 weeks. We also reported that those subjects who had a

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positive history of childhood hyperactivity in the natural father had higher pretreatment urinary MHPG levels than controls, while those hyperactive children without such a history excreted lower levels than controls and the other MBD children.

The present report extends the preliminary investigations to a slightly larger sample and examines whether pretreatment urinary MHPG and its change secondary to *d*-amphetamine administration, in both drug responders and nonresponders, can differentiate subgroups of children with MBD. We also examined whether such subgroups will differ in the presence or absence of soft neurologic signs, considered by many as indicators of delayed maturation of the CNS or as signs of "organicity" in children with MBD (10, 11).

TABLE 1
Comparison of Hyperactive and Control Samples

Item	Hyperactive Children (N=15) Mean	SD	Control Children (N=13) Mean	SD
Age (months)	111	19	125	14
Weight (kg)	30	6	34	7
Height (cm)	134	10	138	10
Body surface (m^2)[a]	1.04	0.15	1.12	0.16
Creatinine (mg/24 hours)	605	167	653	122
Urine volume (ml/24 hours)	965	515	823	234

[a]Obtained from a nomogram modified by C.D. West from data of E. Boyd (12, p. 1713).

METHOD

Fifteen hyperactive boys aged 7–12 with full scale IQs above 80 on the Wechsler Intelligence Scale for Children and with no gross neurologic abnormalities were studied. Informed consent was obtained from all parents and children. The diagnosis of hyperkinetic reaction of childhood (*DSM-II*) was made by the Children Diagnostic Classification and the Children Diagnostic Scale of the NIMH Early Clinical Drug Evaluation Unit Assessment Battery for Pediatric Psychopharmacology. The children were admitted to an inpatient research ward after being free from all medications for at least 2 weeks. All children were given a complete psychological, educational, and behavioral evaluation. They were all given items 1–36 from the neurologic part of the Physical and Neurological Examination for Soft Signs (PANESS). Items 37 to 43 (tapping and the string test) were not given because they were considered cumbersome to administer. A score on the PANESS was obtained for each child based on the sum of the rating scores on items 1–36. There was no diet control in this study. While they were on the unit, the children attended the in-house school, which was located on the same floor, and were allowed to engage in routine everyday activities. Twenty-four-hour urine collections were obtained and completeness of urine collections was monitored by urinary creatinine levels. We discarded samples of low volume (<500 ml/24 hours) and suspiciously low creatinine (<8 mg/kg body weight) (12). Duration of stay was extended as needed if there was a suspicion of urine loss. A male external catheter with adhesive liner and latex material that eliminates leakage was used on all enuretic children (N=5). Sodium-metabisulfite, 0.5 g, was added to the urine samples as a preservative for MHPG, and the samples were refrigerated immediately after patients voided. Aliquots were frozen and later analyzed by electron-capture gas-liquid chromatography according to the method of Dekirmenjian and Maas (13). At the end of the first admission, the children were discharged and went back to school on *d*-amphetamine, 0.5 mg/kg, for a 2-week period, after which they were readmitted and urine was collected again as previously outlined. A minimum of two 24-hour urine collections was obtained from each hyperactive child during each admission.

Conners' Teacher Rating Scales were completed by the children's classroom teachers initially during the week before the first admission when the child was free from medications and then again during the second week before readmission while the child was out of the hospital but was taking amphetamine.

Healthy controls were children of faculty members and staff of the Children and Youth Center, matched for age and sex. Controls were given MHPG determinations but no medication or placebo. A total of four 24-hour urine collections was obtained from each control.

RESULTS

No significant differences were found between hyperactive boys and controls as to age, weight, height, body surface, urinary creatinine, or urine volume (table 1).

MHPG values are expressed in three ways: micrograms per 24 hours, micrograms per one square meter of body surface per 24 hours, and micrograms of metabolite per milligram of creatinine per 24 hours. However, when comparisons are made between subgroups of hyperactive children, the data are expressed in micrograms of MHPG per milligram of creatinine to adjust for the variability in the excretion of metabolites secondary to differences in age, weight, and body surface and to minimize the problem of incomplete urine sample collections if it were present (14, 15).

The group of children with MBD, as a whole, excreted lower levels of MHPG than controls (table 2). The administration of *d*-amphetamine, 0.5 mg/kg divided over two doses daily for two weeks, decreased the levels of MHPG in the hyperactive group (table 3).

Drug Responders Versus Nonresponders

Nine children improved clinically on the drug and 6 children did not as determined by pre- and post-treat-

TABLE 2
MHPG Excretion in Hyperactive Children and Controls

	Hyperactive Children					Controls		
	Pretreatment		Posttreatment					
Measure	Mean	SD	Mean	SD	p[a]	Mean	SD	p[b]
MHPG								
Micrograms per 24 hours	761	340	614	272	n.s.	1078	256	<.05
Micrograms per 24 hours per meter squared	732	252	582	248	n.s.	934	171	<.05
Micrograms per milligram of creatinine	1.35	0.49	0.95	0.44	<.05	1.69	0.33	.05<p<.1
Creatinine (mg/24 hours)	605	167	671	283	n.s.	653	122	n.s.

[a]Two-tailed paired t test for pre- versus posttreatment.
[b]Two-tailed Student's t test for controls versus hyperactive children pretreatment.

TABLE 3
Comparison of Drug Responders and Nonresponders

	Responders (N=9)		Nonresponders (N=6)	
Item	Mean	SD	Mean	SD
Creatinine (mg/24 hours)	554	153	680	159
Age (months)	107	21	122	14
PANESS scores	46	9	31	3[a]
Conners' Factor 1				
Pretreatment	1.8	0.5	1.5	0.8
Posttreatment	0.5	0.2[b]	1.3	0.6[c]
Conners' Factor 4				
Pretreatment	2.6	0.3	2.3	0.4
Posttreatment	0.8	0.3[b]	1.9	0.1[a]

[a]Responders significantly different from nonresponders (p<.002, t test, two-tailed).
[b]Posttreatment significantly different (p<.002, paired t test).
[c]Responders significantly different from nonresponders (p<.05, t test, two-tailed).

ment scores on Factor 1 (conduct problems) and Factor 4 (hyperactivity) of Conners' Teacher Rating Scale. The responders scored significantly higher on the PANESS and tended to be younger than the nonresponders; however, the age difference was not significant (table 3). The administration of *d*-amphetamine decreased urinary MHPG levels in the drug responders but did not change them in nonresponders (table 4). Thus, when drug response was defined as in table 3, pretreatment MHPG level did not significantly differentiate the responders and nonresponders, although it tended to be higher in the responders (table 4). On the other hand, posttreatment MHPG levels did significantly differentiate responders and nonresponders (table 4).

Pretreatment Urinary MHPG Levels

Seven of the hyperactive group excreted MHPG levels that were higher than the control mean; these were considered normal to high levels. Three of the 7 gave a history of hyperactivity and impulsivity in the biological father; 2 other children had been adopted. The 3 children with a history of hyperactivity in the father and one of the adopted children were enuretic. The other adopted child in the study gave a history of enuresis that stopped before his admission to the study. *d*-Amphetamine administration in this group significantly reduced MHPG levels in 4 children who were drug responders, but there was no meaningful change in MHPG levels in the 3 children who did not respond to the drug (table 5).

The remaining 8 children in the hyperactive group excreted pretreatment MHPG levels that were at least one standard deviation below the control mean and were therefore considered the low MHPG group. Only 1 child from this group was enuretic. *d*-Amphetamine administration significantly decreased MHPG levels in the 5 children who were drug responders, while it increased MHPG levels in the 3 who were nonresponders (table 5).

Soft Neurologic Signs

The drug responders differed significantly from the nonresponders in their PANESS scores (table 3). Furthermore, the drug responders significantly differed from the nonresponders in both the normal to high pretreatment MHPG and the low pretreatment MHPG groups (table 5).

DISCUSSION

In man, as well as in other species, MHPG is the main metabolite of central nervous system NE (16). It has been estimated that over 50% of urinary MHPG comes from the metabolism of NE in the CNS in man (17). Its excretion in the urine has been shown to vary in response to changes in sympathetic activity or stress (18, 19). The variability in excretion in response to changes in motor activity is not as clear: it was shown to vary with changes in motor activity in one report (20), but in another no such relationship could be found (19). It is generally agreed that high urinary MHPG levels could reflect a primary increase in activity of the presynaptic neurons or in norepinephrine output, as well as a resultant compensatory increase (21). It is also agreed that reduced urinary MHPG levels may be considered secondary to decreased rates of synthesis, turnover, or neuronal discharge of NE (21).

TABLE 4
Urinary MHPG Before and After *d*-Amphetamine Treatment

MHPG Measure	Responders (N=9) Mean	SD	p[a]	Nonresponders (N=6) Mean	SD	p[a]	p[b]
Micrograms per 24 hours							
Pretreatment	732	252	<.05	853	301	n.s.	n.s.
Posttreatment	479	173		816	267		<.05
Micrograms per 24 hours per meter squared							
Pretreatment	715	251	<.05	774	222	n.s.	n.s.
Posttreatment	458	140		767	240		<.05
Micrograms per milligram of creatinine							
Pretreatment	1.40	0.52	<.01	1.18	0.40	n.s.	n.s.
Posttreatment	0.78	0.26		1.22	0.52		.05<p<.1

[a]Student's paired t test.
[b]Student's t test of independent means, two-tailed, comparison of responders and nonresponders.

TABLE 5
Comparison of Normal to High MHPG and Low MHPG Groups

Item	Normal to High MHPG: Responders (N=4) Mean	SD	p[a]	Nonresponders (N=3) Mean	SD	p[a]	p[b]	Low MHPG: Responders (N=5) Mean	SD	p[a]	Nonresponders (N=3) Mean	SD	p[a]	p[b]
Micrograms of MHPG per milligram of creatinine														
Pretreatment	1.92	0.23	<.002	1.70	0.14	n.s.	n.s.	0.99	0.24	<.01	0.87	0.22	n.s.	n.s.
Posttreatment	0.77	0.25		1.30	0.56		n.s.	0.78	0.27		1.14	0.46		n.s.
PANESS score	45	9		31	3		<.05	47	10		30	3		<.05
Age (months)	105	19		119	11		n.s.	108	22		123	16		n.s.
Milligrams of creatinine per 24 hours														
Pretreatment	442	113		643	204		n.s.	644	116		626	78		n.s.
Posttreatment	601	164		595	268		n.s.	717	78		916	454		n.s.

[a]Student's paired t test.
[b]Student's t test of independent means, two-tailed, comparison of responders and nonresponders.

Furthermore, destruction of NE terminals in animals has been shown to result in decreased urinary levels of MHPG (22).

Amphetamine has been shown to influence NE metabolism by increasing the release of NE from the presynaptic nerve terminal (23), by inhibiting the reuptake of NE by the presynaptic nerve terminal (24), and by inhibiting the intraneuronal enzyme monoamine oxidase (25). The first action tends to increase MHPG production, while the other two tend to decrease it (26).

In this study, reduction in urinary MHPG was correlated with clinical improvement in nine children and with no clinical improvement in three. However, the reduction in MHPG excretion was statistically significant in the drug responders only. In three other children who did not improve clinically, MHPG excretion increased.

The finding that hyperactive children with soft neurologic signs (high scores on the PANESS) respond better clinically to *d*-amphetamine is in keeping with findings of other clinicians (27, 28). Soft neurologic signs, which may indicate delayed maturation and/or minimal damage to the CNS (1, 10), have been used as a predictor of favorable clinical response to stimulant administration. In this study, all children with good clinical response to *d*-amphetamine had soft neurologic signs (i.e., high scores on the PANESS), while none of the children who lacked soft neurologic signs (i.e., low scores on the PANESS) responded favorably to the drug. There seems to be a developmental trend in PANESS scores (29), and the higher PANESS scores in the drug responders may have been a result of such a trend, since the responders were younger. Thus, it might follow that whatever abnormality is causing the soft signs also renders the CNS NE system more responsive to stimulant medication.

COMMENT

Our data so far suggest that there is a relationship between minimal brain dysfunction and CNS NE metabolism and that there may be a relationship between

clinical drug response to *d*-amphetamine and change in NE metabolism.

We also found that pretreatment urinary MHPG levels do not differentiate responders and nonresponders to *d*-amphetamine in the fixed dose of 0.5 mg/kg divided over two doses daily for two weeks when drug response is determined by a decrease (at least 2 SDs) from the population mean on the hyperactivity factor of the Conners' scale. There is evidence that drug responders may come from the group with normal to high MHPG as well as from the group with low MHPG. Moreover, the presence of soft neurologic signs in hyperactive children improves their chances to respond favorably to *d*-amphetamine.

Finally, there does not seem to be a relationship between soft signs and pretreatment urinary MHPG levels. Children with soft signs fall into the normal to high MHPG group and the low MHPG group.

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Amino Acids in Bipolar Affective Disorders: Increased Glycine Levels in Erythrocytes

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The authors measured the concentrations of 20 amino acids in the erythrocytes and plasma of 13 female bipolar patients and 10 female normal controls. The concentration of glycine in the erythrocyte was significantly elevated in the patient group. No differences were present in the plasma. Preliminary findings indicate that the high glycine levels were present in patients who were depressed, manic, or in remission and were unaffected by electroshock therapy.

A NUMBER OF abnormalities have been reported in the erythrocytes of patients with affective illnesses. The activity of erythrocyte catechol *O*-methyltransferase (COMT), an important enzyme in biogenic amine metabolism, has been reported by some, but not all, investigators to be different in patients with primary affective disorders than in normal control groups (1). The biochemical properties of the erythrocyte enzyme indicate that it is probably identical to brain COMT (2). The erythrocyte also contains an active cation transport system similar to that of the neuron (3). In patients with affective illness there is a decrease in the activities of the erythrocyte Na^+ pump and ouabain-sensitive Na^+,K^+-ATPase (4). Both are stimulated with the administration of lithium, and the increase in activity may be related to the improvement of depressive states and the prophylactic efficacy of lithium maintenance therapy (5).

The erythrocyte, therefore, is a useful cellular model for investigating some of the physiological characteristics of the affective disorders. Because amino acid transport also is under the control of an active, energy-dependent carrier system (6), the concentrations of amino acids in the erythrocytes and plasma of manic-depressive patients were examined.

METHOD

The patients consisted of 13 women, 33–72 years old, who had affective illnesses of 10 to 20 years in duration. Ten were bipolar manic-depressives, and 3 were considered to have schizo-affective illness because of their psychopathology during acute episodes. However, because there were no residual symptoms, decrement in functioning, or personality deterioration after remission from multiple acute phases of illness, a diagnosis of manic-depression could be equally valid. The control group consisted of 10 female hospital personnel, aged 24–58 years, without personal or family history of an affective illness. They were in good health and not on any medication. Neither group was taking oral contraceptives. Six patients and 8 controls were premenopausal, and 7 patients and 2 controls were in menopause.

Of the 10 bipolar patients 3 were in remission and receiving 600 mg/day of lithium carbonate. These patients had not had an affective illness for at least 1 year, were in good health, and were not hospitalized for this study. Lithium carbonate was stopped for 7 days in these patients before blood was taken for amino acid analysis. Of the 10 hospitalized patients, 8 were depressed. Prior to admission they were under treatment with inadequate dosages of tricyclic antidepressants and had not been on maintenance lithium carbonate therapy. Two of the hospitalized patients were in the manic phase of their illness. At the time of hospitalization, all medication was discontinued for the depressed patients but was not interrupted for the 2 manic patients, 1 of whom continued to receive lithium carbonate (1200 mg/day) and the other haloperidol (10 mg/day). All 13 patients underwent routine physical examinations and complete blood and laboratory studies. Except for 1 patient, a known diabetic, all of the findings were within normal limits. The diabetic patient had an elevated fasting blood glucose of 180 mg/100 cc but was not excreting ketone bodies. This

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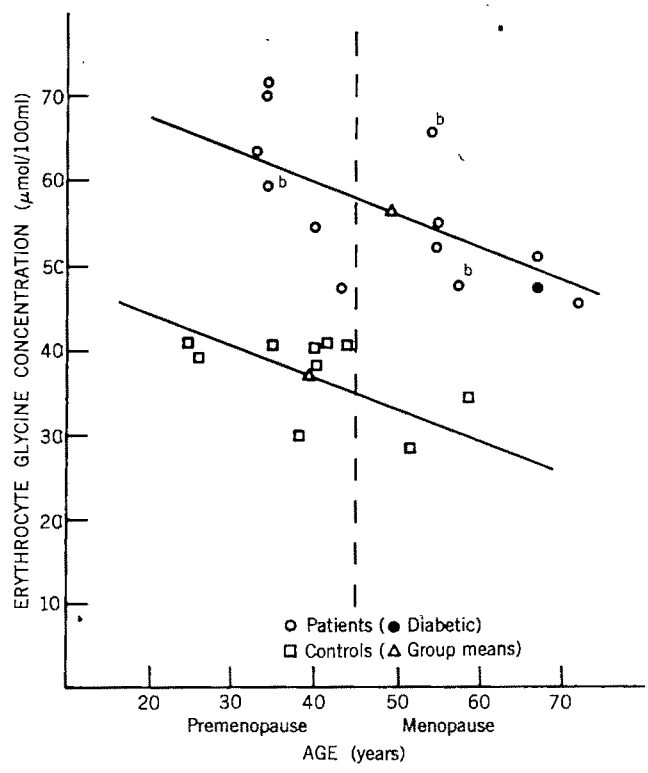
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patient was placed on a 1,200-calorie diet and given additional insulin, which brought the diabetes under control. Dietary control was maintained in the other patients by the use of liquid nutritional supplement (Sustecal). The patients drank 12 ounces of the supplement whenever they ate less than 50% of any meal. The daily caloric intake of these patients was never less than 2,000 calories. The patients were on the dietary regimen in addition to being drug-free for at least 5 days before blood was drawn for amino acid analysis after an overnight fast. The control subjects were instructed to adhere to their usual diets. It was ascertained that they were on conventional balanced diets; none was on a weight reduction diet.

Blood (15 cc) was drawn into heparinized vacutainer tubes and immediately centrifuged at 4,000 *g* for 25 minutes at room temperature. After the plasma was aspirated, the buffy coat of white blood cells and platelets was carefully removed with a Pasteur pipette. An additional 2 ml of the adjacent packed erythrocytes was also removed and discarded. The remaining erythrocytes were diluted with 2 volumes of distilled water and vigorously stirred. The lysed cells and plasma were stored at −70°C until analyzed. Amino acid concentrations in the plasma and erythrocytes were determined with an automated Technicon amino acid autoanalyzer (7).

FIGURE 1
Concentrations of Erythrocyte Glycine in Patients and Controls[a]



[a]Prediction equations: patient, glycine=75.79−0.3932 × Age; control, glycine=53.09−0.3932 × Age.
[b]Nonhospitalized patients.

RESULTS

The concentration of erythrocyte glycine was significantly higher in the patient group than in the control groups (56.3 μmol/100 ml±2.5 versus 37.4 μmol/100 ml±1.8) ($p<.001$). There were no differences in plasma glycine. Except for a modest increase in serine ($p<.02$) in the patients' erythrocytes (but not in their plasma) no further differences were observed. The other amino acids measured were taurine, aspartate, threonine, asparagine, glutamate, glutamine, proline, alanine, valine, leucine, isoleucine, tyrosine, phenylalanine, ornithine, lysine, histidine, methionine, and glutathione.

Erythrocyte glycine concentrations, ages, and menstrual status are shown in figure 1. No overlap was present between the patient and control values. Glycine levels of the patients were significantly higher than those of the controls as shown by parallel prediction equations. Glycine levels decreased with age. The slope of glycine versus age was −0.2456 for the controls and −0.4530 for the patients, and a t test for difference between the two slopes was not significant ($t=.92$, $p<.30$). The pooled estimate of the slope was −0.3932. The mean age was 39.8 years for controls and 49.6 years for patients.

The differences between the erythrocyte glycine levels of the premenopausal patients and controls could conceivably have been due to the time of sampling, with respect to the stage of the menstrual cycle. These data were not obtained at the time of the study. Although there are no reports as to changes in erythrocyte amino acids with the menstrual cycle, variations in plasma amino acids do occur. In normal women there is a decrease in serine ($p<.02$), glutamate ($p<.02$), and ornithine ($p<.01$) in the second half of the cycle (8). We observed no significant differences in these plasma amino acids between the patient and control groups, which would suggest that the time of sampling of these two groups was not skewed toward different stages of the menstrual cycle.

Three patients (2 manic and 1 depressed) underwent a course of electroshock therapy during which time all psychotropic drugs were stopped. Additional amino acid studies were done throughout this period and again about 3 weeks following the last treatment. These 3 patients were in clinical remission at this point, had been home for 2 weeks, and were not taking psychotropic drugs. No significant changes occurred in the levels of glycine or other amino acids in the erythrocytes or plasma. Although these conditions would appear to rule out the role of drugs in contributing to the elevated glycine levels, residual drug effects cannot be completely eliminated at this time. Long-term malnutritional effects must also be considered among those variables that might have contributed to the high glycine values. In addition, a number of control subjects will be placed on the same dietary regimen as the patient group in future studies.

DISCUSSION

The increase in erythrocyte glycine without significant changes in the other amino acids makes it unlikely that the differences found are due to an alteration in cellular transport. Glycine shares a carrier-mediated transport system with proline and alanine (9), and the concentrations of these amino acids were not affected. The elevation of glycine could not be attributed to a diminished synthesis or increased degradation of gluthathione in the erythrocytes since the tripeptide concentration was not decreased in the patient group.

Although glycine undergoes numerous metabolic transformations, its primary metabolic pathway is via the glycine cleavage system (10). It is therefore reasonable to speculate that the observed increase in glycine levels may be due to an impairment of this system, which degrades glycine to CO_2+NH_3 with concomitant formation of *N*-5,10-methylenetetrahydrofolate from tetrahydrofolate. Methylenetetrahydrofolate reductase and serine hydroxymethyltransferase but not the glycine cleavage system have been measured in the erythrocyte (11). Although the glycine cleavage system is present in mitochondria that are absent in the erythrocyte, the exclusion of this system from the erythrocyte has not been demonstrated. Glycine, however, may be increased in other tissues and transported to and retained in the erythrocyte. It has been shown that following parenteral infusions of amino acids, tissue levels can be elevated without concomitant changes in plasma levels (12).

It is not known whether the increase in glycine in the erythrocytes is also present in the brain where the glycine cleavage system is the main degradative pathway (13). It is conceivable that a moderate elevation in the central nervous system may affect amino acid and protein metabolism. Glycine is also an inhibitory neurotransmitter in the lower brain and spinal cord and an increase in cellular availability might be expected to affect this action (13).

The consequences of a defect in the glycine cleavage system is seen in the genetic disease, nonketotic hyperglycinemia, which is due to a severe inborn deficiency of this system. The disease results in marked neurological damage, convulsions, and death (14); however, there is a recent report of several affected infants in whom minimal or no neurological symptoms were found (15). Apparently a mild form of this disease can exist that is symptomless and compatible with life.

Although no apparent differences were noted between patients in mania, depression, and remission, the number in each group was too small for statistical analysis. A larger series of patients should be studied longitudinally with multiple sampling through various stages of the illness and remission in order to determine whether the glycine levels remain unchanged. If this situation is replicated, one could conclude that an elevated erythrocyte glycine level represents a metabolic defect (underlying amino acid disorder) that predisposes to the development of bipolar illness (in women) or is a genetic marker for those at risk. To our knowledge, this is the first observation of an abnormality in the affective illnesses that involves an amino acid not directly related to biogenic amine metabolism. This preliminary finding requires replication with a larger number of patients before it can be considered a specific and distinguishing characteristic of this patient subgroup.

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Beta-Adrenergic Receptor Function in Affective Illness

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In a study of β-adrenergic receptor sensitivity, the authors determined the response of cyclic AMP synthesis to in vitro addition of norepinephrine (NE) and isoproterenol (IP) in leukocytes of patients with affective illness and schizophrenia and of normal controls. IP-stimulated increase in ^{3}H-cyclic AMP synthesis in depressed patients was significantly lower than in normal subjects and schizophrenic patients. These results suggest that β-adrenergic receptor sensitivity is reduced in depressive illness.

IT HAS BEEN proposed that affective illness and schizophrenia may be associated with a dysfunction of biogenic amines. A functional deficiency of norepinephrine (NE) or serotonin (5-HT) may be involved in certain types of depressive illness, while an excess of these amines may be present in mania. Schizophrenia, on the other hand, has been related to hyperactivity of the dopaminergic (DA) system. Abnormality of amine function may be caused by alterations in amine levels as a result of changes in their biosynthesis, release, reuptake, or metabolism, or it may be due to an imbalance in the transmitter systems. It is also possible that amine dysfunction results from altered sensitivity of the amine receptors. This has recently been reemphasized by Aschroft and associates (1), who suggested that tests of neuronal sensitivity in man should be developed to further evaluate this possibility.

For the past several years we have investigated the possibility that an abnormality of catecholamine receptors is associated with schizophrenia and depression. Using a neuroendocrine strategy, we recently demonstrated that hypersensitive DA receptors may be associated with a subgroup of patients diagnosed as having acute schizophrenia (2). Another indirect method for the study of biogenic amine receptor function in psychiatric illness involves the use of the cyclic-nucleotide system.

Cyclic AMP, which is formed by the catalytic action of the enzyme adenylate cyclase on adenosinetriphosphate (ATP), mediates the cellular effects of several hormones and neurotransmitters in both peripheral and brain tissues (3, 4), and neurotransmitters such as NE have been shown to increase the concentration of cyclic AMP in the brain (5, 6). Cyclic AMP, which has been implicated in synaptic transmission (7), has been found to be related and closely coupled to adrenergic receptors (7, 8). It is thus possible to study adrenergic function by studying adenylate cyclase responses to NE produced by human peripheral tissues.

In order to investigate adrenergic receptor sensitivity in affective illness further, we have studied leukocyte adenylate cyclase and its responsiveness to NE and isoproterenol (IP). It has been reported that alterations in cyclic AMP synthesis produced by NE and IP in human leukocytes are primarily β-adrenergic receptor responses (9). The presence of adrenergic receptors in human leukocytes was demonstrated by Williams and associates (10) in direct binding studies with ^{3}H-dihydroalprenolol (DHA).

Because human leukocytes are easily available, they provide a useful peripheral model for the study of β-adrenergic receptor sensitivity in a variety of pathologic conditions. Leukocyte adenylate cyclase and its responsiveness to NE and IP have been studied in subjects with bronchial asthma (11-13); however, to our knowledge, studies of leukocyte adrenergic receptor sensitivity have not been reported in patients with psychiatric disorders.

METHOD

Subjects were patients hospitalized on the research ward of the Illinois State Psychiatric Institute. Diagnoses, according to the Research Diagnostic Criteria of Spitzer and associates (14), were as follows: major unipolar depression, N=21; bipolar mania, N=9; and schizophrenia, N=29. Bipolar patients were all manic at the time of admission. Normal controls (N=26) were nonhospitalized volunteers who were free of any known major medical or psychiatric disorders. The subjects gave written informed consent to participate in the study.

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TABLE 1
Percent Conversion of Total ^{3}H-Nucleotides to ^{3}H-Cyclic AMP In Intact Human Leukocytes[a]

Diagnostic Group	Basal[b]			PGE_1 (10^{-5} M)			NE (10^{-4} M)[c]			IP (10^{-4} M)[d]		
	N	Mean	SD	N	Mean	SD	N	Mean	SD	N	Mean	SD
Normal subjects	26	0.25	0.02	26	1.77	0.18	26	0.42	0.04	21	0.68	0.06
Unipolar depressed patients	21	0.29	0.03	21	1.34	0.12	19	0.20	0.03	17	0.31	0.05
Bipolar patients with mania	9	0.24	0.02	9	1.54	0.26	9	0.28	0.04	8	0.40	0.05
Schizophrenic patients	30	0.27	0.02	29	1.48	0.21	30	0.26	0.05	26	0.51	0.08

[a]The percent conversions in the presence of NE, IP, or PGE_1 are those observed with the addition of these drugs over the basal activity and have been referred to as stimulated activity.
[b]Percent conversion when no test drugs were added to the incubation mixture.
[c]NE stimulation differed significantly between the groups, $F(3, 80)=4.14$, $p<.009$, and between normal subjects and patients, $F(1, 80)=10.08$, $p<.002$.
[d]IP-stimulated activity differed significantly between groups, $F(3, 68)=4.59$, $p<.006$; for normal subjects versus patients, $F(1, 68)=10.08$, $p<.002$; for normal subjects versus depressed patients, $F(1, 68)=12.61$, $p<.001$; and for schizophrenics versus depressed patients, $F(1, 68)=4.04$, $p<.05$).

Leukocyte adenylate cyclase studies of patients were performed at the end of a three-week drug-free period. Intact leukocytes were isolated according to the method described by Bourne and associates (15, 16). For the determination of adenylate cyclase, the pulse-labeling technique previously described by Kuo and DeRenzo (17) and by Shimizu and associates (18) was used; the procedure is similar to that described by Wang and associates (19). Leukocyte pellets were suspended in Tris-balanced buffer (pH 7.4) and incubated with ^{3}H-adenine (50 μCi) for 1 hour at 37°C. The suspension was then centrifuged at 2,500 g for 15 minutes, and the pellet was washed twice with Tris buffer and suspended in the buffer (1.5 to 2.5 ml). Final incubation of aliquots of the suspension was done at 37°C for 10 minutes in Tris-balanced buffer containing 10 mM $MgCl_2$, 1mM theophylline, and in the presence or absence of NE, isoproterenol (IP), or prostaglandin E_1 (PGE_1) in a total volume of 0.5 ml. The reaction was terminated by addition of a solution of cyclic AMP. The ^{3}H-cyclic AMP formed was isolated and quantitated according to the method of Krishna and associates (20) as described by Wang and associates (19). The results are expressed as percent conversion of total incorporated ^{3}H-nucleotides to ^{3}H-cyclic AMP.

RESULTS

Addition of dextro-levo IP, the β-adrenergic agonist, in vitro causes a dose-dependent increase in the production of ^{3}H-cyclic AMP. Similarly, NE and PGE_1 cause a dose-dependent increase in ^{3}H-cyclic AMP synthesis in leukocytes. The increase produced by IP is blocked by the addition of propranolol, a β-adrenergic antagonist, but not by phentolamine, an α-adrenergic antagonist. Because similar results are observed with NE, this suggests that the stimulation of ^{3}H-cyclic AMP synthesis in human leukocytes produced by NE and IP is a β-adrenergic receptor response. Stimulation of ^{3}H-cyclic AMP produced by the addition in vitro of PGE_1, however, is not blocked by propranolol.

Adenylate cyclase activity and the stimulation produced by addition in vitro of PGE_1 (10^{-5} M), IP (10^{-4} M), or NE (10^{-4} M) were determined in the 26 controls and 59 patients. The mean basal activity observed in the patient population was not significantly different from that in the control group (table 1). In vitro addition of PGE_1 (10^{-5} M) produced an approximately 6-fold increase in net ^{3}H-cyclic AMP synthesis in the intact leukocytes over the basal activity. One-way analysis of variance showed that the mean PGE_1-stimulated activity in the patient groups did not differ significantly from that in the normal controls.

In vitro addition of NE (10^{-4} M) produced an approximately 1.5-fold increase in net ^{3}H-cyclic AMP synthesis in normal controls. The mean activity observed in the patient groups, however, was significantly lower than that in the controls, as determined by one-way analysis of variance (table 1). This suggested that the ^{3}H-cyclic AMP synthesis produced by NE, although lower in the patient population, could not distinguish between patients with affective illness and schizophrenic patients (p=n.s., Scheffe's test). Synthesis of ^{3}H-cyclic AMP by human leukocytes was also measured after addition in vitro of IP (10^{-4} M). This addition produced an approximately 2-fold increase in net ^{3}H-cyclic AMP synthesis above the baseline.

One-way analysis of variance indicated significant differences between the various subject groups (table 1). Further analysis by Scheffe's test indicated that patients diagnosed as having major unipolar depressive disorders showed significantly lower IP-stimulated activity than normal controls ($p<.001$) and schizophrenic patients ($p<.05$). Patients with bipolar mania were significantly different from normal controls but did not differ from the unipolar depressed patients. Schizophrenics did not differ significantly from normal controls.

DISCUSSION

Studies of adrenergic receptor function in affective illness with the use of platelet adenylate cyclase and its responsiveness to PGE_1 and NE have been reported

by Wang and associates (19) and Murphy and associates (21). The platelet responses observed in depressed patients were found to be indistinguishable from those of normal controls. The responses produced by NE in human platelets are primarily α-adrenergic, whereas elevation of cyclic AMP synthesis produced by NE in the brain has been reported to result primarily from β-adrenergic receptors, although α-receptors may also be involved (19–21). Studying α-adrenergic receptor response in human platelets to evaluate a generalized abnormality in the adrenergic response may be of value. However, in the light of the negative results obtained for platelet adenylate cyclase, it may be useful to perform such studies in a peripheral tissue in which alterations in cyclic AMP synthesis are produced by responses of the β-adrenergic receptors.

Our leukocyte studies indicated that there was no difference in either the basal activity or the PGE_1-stimulated activity among the groups studied. NE-sensitive leukocyte adenylate cyclase activity was significantly lower in patients than in normal controls, and IP-stimulated activity was significantly lower in the depressed group than in controls and schizophrenic patients. Although these results generally suggest a reduced sensitivity of β-adrenergic receptors in the leukocytes of patients with affective illness, we did not expect to find significantly lower NE-sensitive leukocyte adenylate cyclase activity in schizophrenics than in controls.

Several explanations could be offered for this observation. Norepinephrine is both an α- and a β-adrenergic agonist, and the net increase in cyclic AMP synthesis produced by NE in human leukocytes is a cumulative response of both α- and β-adrenergic receptors (22, 23) because some α-receptors may be present in human leukocytes (15). Since the function of α-adrenergic receptors is usually opposite to that of β-adrenergic receptors, a reduction in cyclic AMP synthesis in the leukocytes produced by NE in the patient groups could be caused by either subsensitivity of β-adrenergic receptors, which stimulate cyclic AMP synthesis, or hypersensitivity of α-adrenergic receptors, which inhibit the synthesis of cyclic AMP. It is possible that the decreased response produced by NE in leukocytes reflects hypersensitivity of α- and normal sensitivity of β-adrenergic receptors in schizophrenic patients and subsensitive β- and normal α-adrenergic receptors in affectively ill patients. The net response produced by NE in either situation is a reduction in the cyclic AMP synthesis.

Further observation that ^{3}H-cyclic AMP synthesis produced by IP, which is a much more specific β-adrenergic agonist, is not significantly different in schizophrenic patients and controls tends to support the above assumption. Thus, whereas increased α-adrenergic receptor sensitivity may be associated with schizophrenic illness, a reduction in β-adrenergic receptor sensitivity may be involved in affective illness. It is also possible that a common biologic abnormality of β-adrenergic receptor deficiency, not specific to either disease, is associated with altered behavior.

Our results suggesting a reduction in the β-adrenergic receptor sensitivity in depressed patients are similar to those reported by Prange and associates (24), who observed diminished systolic pressor response to NE in depressed patients. However, these findings are contradictory to those reported by Ghose and associates (25) and Friedman (26), who observed an increased pressure response to intravenously administered amines. It may be difficult to compare our studies, which determine an in vitro biochemical response in an isolated cell preparation, to their studies, which measured an in vivo physiologic response. Furthermore, the sensitivity of receptors developed in different areas may be different and unrelated, as reported by Friend and associates (27).

Some reports suggest that there is a significant decrease in IP-stimulated cyclic AMP synthesis in patients suffering from bronchial asthma (11–13). Recently, it has been reported that some depressed patients also suffer from bronchial asthma and that they benefit from treatment with lithium (28). Although this report was based on two cases, one of the authors has found similar results in additional patients (S. J. Nasr, personal communication). The relationship of bronchial asthma, depression, and lithium response to adenylate cyclase is not clear, but these observations may provide useful clues for future investigations on the etiology of depression.

The reported results of reduced β-adrenergic receptor sensitivity in depression need to be investigated further by other methods, such as direct quantitation of β-adrenergic receptor sites in human leukocytes by binding techniques with radiolabeled antagonists such as ^{3}H-DHA. This preliminary study needs to be further investigated and replicated by other researchers.

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The Role of Plasma Amine Oxidase, Platelet Monoamine Oxidase, and Red Cell Catechol-*O*-Methyl Transferase in Severe Behavioral Reactions to Disulfiram

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The authors assayed platelet monoamine oxidase (MAO), plasma amine oxidase (AO), and red cell catechol-O-methyl transferase (COMT) in 32 male alcoholics before they began disulfiram treatment. Seven subjects developed psychotic reactions to disulfiram; these 7 had significantly lower pretreatment MAO and AO levels and significantly higher COMT than the patients who had no adverse reactions to disulfiram, which suggests that severe behavioral reactions to disulfiram are associated with differences in enzyme activities.

FACTORS MOST COMMONLY used to explain individual differences in drug responses include variations in the absorption, distribution, metabolism, or excretion of a drug (1). There are also psychological determinants of pharmacological response, such as the initial state of individual arousal determining the response to a drug acting on arousal mechanisms or preexisting mood determining the affective response to a mood-altering drug (2–4).

More recently a growing number of unusual drug responses have been shown to be genetically determined, for example, prolonged apnea secondary to succinylcholine in patients with genetically atypical pseudocholinesterases (5), polyneuritis as a toxic complication of isoniazid in patients with genetically determined slow acetylation (6, 7), and hemolysis secondary to primaquine in individuals with glucose-6-phosphate dehydrogenase deficiencies (8). Prior to the discovery of the genetically determined enzyme abnormality involved, the above-mentioned unusual drug reactions were called idiosyncratic reactions. In this paper we look at another idiosyncratic reaction, disulfiram psychosis, in an attempt to find some common underlying mechanism that may be responsible for these atypical drug reactions.

Disulfiram was first introduced in the late 1940s after it was noted to cause an adverse reaction when alcohol was ingested (9). This adverse reaction was shown to be the result of disulfiram's inhibition of aldehyde dehydrogenase, an enzyme necessary for the metabolism of alcohol (10, 11). Disulfiram has also been shown to inhibit other enzymes (12), particularly dopamine beta hydroxylase, the enzyme responsible for the conversion of dopamine to norepinephrine (13). Initial widespread use of the drug resulted in the reporting of a high incidence (30%) of psychotic reactions (14, 15). Further studies revealed that the psychotic reactions were dose related, more likely to develop in patients who developed other side effects such as lethargy and fatigue (15), and more common in individuals with depressive, borderline, or schizophrenic diagnoses (16).

We have previously shown differences in the enzymes of monoamine metabolism in individuals with affective disorders (17, 18) and schizophrenia (19). The hypothesis that differences in one or more of the enzymes of monoamine metabolism might be involved in psychotic reactions to disulfiram led to the present retrospective review.

METHOD

Our study included 32 patients from an alcohol rehabilitation unit that used disulfiram in its treatment program. They were part of a larger study investigating platelet and plasma amine oxidase (AO) in 99 alcoholic individuals (20). Because of limited resources, erythrocytes for the assay of catechol-*O*-methyl transferase (COMT) were collected but not routinely assayed on all 99 subjects. Instead 32 individuals were selected as a pilot group and all erythrocyte samples on these individuals were assayed for COMT. All patients with adverse reactions to disulfiram (N=12) were included in order to test our hypothesis. The remaining 20 individuals were randomly selected from the larger group in order to arrive at 16 individuals who had received disulfiram in doses of 500 mg/day and 16 individuals who had received 250 mg/day to test for

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possible dose-dependent effects of disulfiram on COMT. Samples had been collected both before and after disulfiram treatment.

The patients' age range was 18 to 50 with a mean (± SEM) of 33±1.9 years. All patients were free of cardiovascular, renal, or liver disease and met *DSM-II* criteria for habitual or episodic excessive drinking and Spitzer and associates' Research Diagnostic Criteria (RDC) (21) for alcoholism. All 32 patients had been abusing alcohol for more than 2 years (range=2-20+) and had a score of greater than 10 on the Michigan Alcoholism Screening Test (22), a series of 24 questions designed to gauge the degree of a patient's alcohol abuse. Their mean score was 15.6, out of a possible 24; 6 is considered the cut-off score indicating a significant alcohol problem. None of the patients had a diagnosis of psychosis or organicity according to *DSM-II* or the RDC.

All 32 subjects were drug free, except for alcohol, for at least 14 days before admission to the hospital, and while in the hospital they were drug free except for disulfiram. All other drugs were carefully avoided during the study.

Within 5 days of admission and before disulfiram was started, venous blood samples were collected. Platelet-rich plasma (PRP) was prepared by sequential 10-minute centrifugations at 175 and 300 *g*; the PRP was removed and pooled after each centrifugation and then frozen at −70°C after an aliquot was removed for platelet counts. The sample remainder was then centrifuged at 8000 *g* for 10 minutes; plasma was removed and stored at −70°C and 2 ml of packed red blood cells removed and frozen at −70°C. Platelet and plasma amine oxidase activity were determined by Murphy and associates' method (23) and red cell COMT by the method of Jonas and Gershon (24).

After the initial venipuncture, 16 individuals received disulfiram, 500 mg/day, and the remaining 16, 250 mg/day. All medication was crushed (in order for patients to be blind as to amount received) and given in milk under observation to insure compliance. Every patient was evaluated daily by the staff psychiatrist for the initial 6-week period of disulfiram treatment as part of the rehabilitation program. The patients who developed psychiatric reactions to disulfiram were evaluated and diagnosed by a psychiatrist who was blind to the medication they had received and to the pretreatment biochemical data. Repeat blood samples were collected at 3 weeks and 6 weeks.

RESULTS

Individual pretreatment enzyme values for all 32 subjects are listed in table 1. Both plasma amine oxidase activity and platelet monoamine oxidase activity were significantly lower in those individuals who later developed psychotic reactions to disulfiram ($p<.01$, Student's two-tailed t test) (table 2). Similar results were found when the larger group (N=99) was examined ($p<.001$, Student's two-tailed t test). Pretreatment red cell COMT was significantly higher in those individuals who developed psychotic reactions ($p<.001$, Student's two-tailed t test) (table 2). The five individuals who developed minor reactions to disulfiram tended to have lower platelet and plasma amine oxidase activity and higher COMT activity, but these differences did not reach statistical significance (table 2). Age, severity of drinking, chronicity of drinking, and time of last drink were not significantly different in the groups with and without reactions to disulfiram and were not significantly correlated with MAO, AO, or COMT activities.

Using discriminate function analysis, red cell COMT was the best single pretreatment indicator of future behavioral side effects during disulfiram treatment, correctly placing 22 of the 32 individuals (68.8%)

FIGURE 1
Relationship Between Plasma AO, Platelet MAO, and Red Cell COMT Activities and Adverse Reactions to Disulfiram

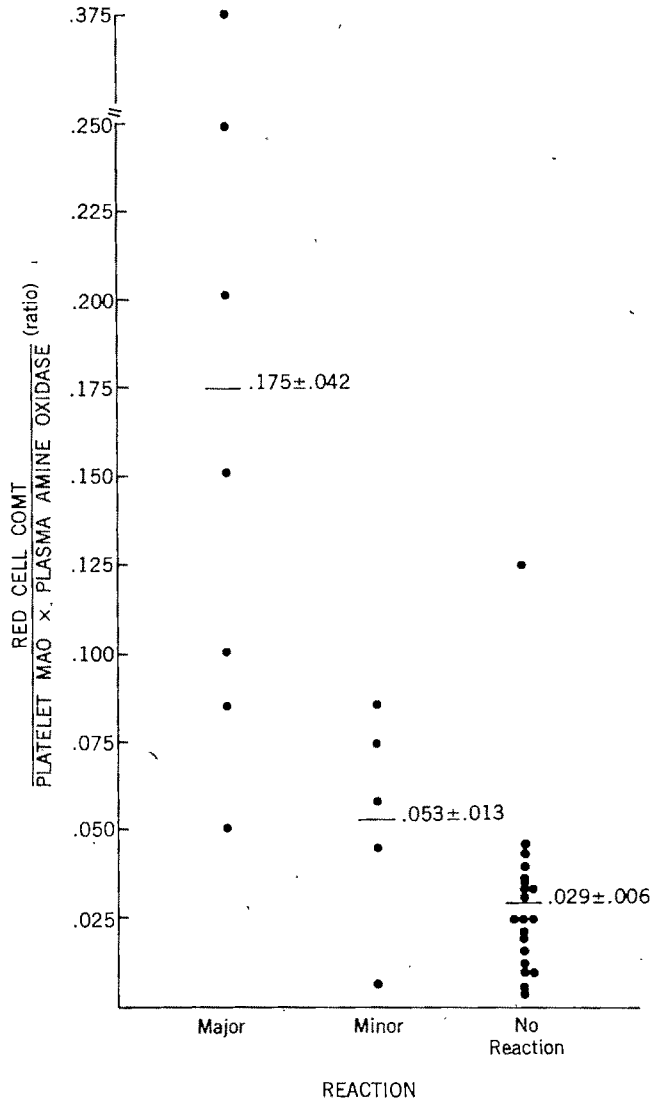


TABLE 1
Relationship of Pretreatment Plasma AO, Platelet MAO, and Red Cell COMT Activities to Adverse Reactions to Disulfiram

Reaction to Disulfiram, by Patient	Age (years)	Plasma AO[a]	Platelet MAO[a]	Red Cell COMT	$\frac{\text{COMT}}{\text{Platelet MAO} \times \text{Plasma MAO}}$ (ratio)	Subsequent Dose of Disulfiram (mg)
Mixed toxic-schizophrenic[b]						
1	21	13.9	4.5	13.16	.210	500
2	22	7.9	3.9	11.57	.376	500
3	21	13.8	4.9	6.98	.103	500
4	29	13.7	6.7	7.80	.085	500
5	28	13.6	6.1	4.15	.050	500
Mixed toxic-manic[b]						
6	31	14.1	2.7	5.89	.155	500
Mixed toxic-depressed[b]						
7	30	10.2	3.5	8.83	.247	500
Fatigue, lethargy[c]						
8	36	14.8	4.5	5.73	.086	250
9	23	18.6	4.7	6.23	.072	500
10	42	15.3	5.3	3.57	.044	250
11	27	10.1	4.4	2.59	.058	250
Mild confusion[c]						
12	21	17.9	14.3	1.47	.006	500
No reaction						
13	33	19.4	8.9	8.05	.046	500
14	19	20.8	8.8	6.87	.038	250
15	18	19.7	4.8	3.04	.032	500
16	23	16.4	11.2	1.00	.005	250
17	27	17.9	9.1	3.09	.019	250
18	40	14.7	3.8	2.00	.036	250
19	21	7.5	5.9	1.41	.032	500
20	18	20.2	8.5	5.11	.030	250
21	46	14.3	12.5	1.80	.010	500
22	41	17.2	10.3	4.31	.024	250
23	22	19.1	10.8	0.89	.004	250
24	50	27.6	6.5	1.73	.010	250
25	34	17.8	7.6	3.02	.021	250
26	22	20.2	4.7	1.13	.011	250
27	50	19.5	12.9	6.25	.025	500
28	24	15.9	7.4	2.83	.024	500
29	36	19.4	4.0	3.40	.044	250
30	37	17.4	9.7	5.75	.034	250
31	47	11.5	5.8	8.27	.124	500
32	26	15.4	6.3	1.53	.016	500

[a]See table 2 for units for enzyme activities.
[b]Psychotic reaction, medication discontinued.
[c]Nonpsychotic reaction, medication reduced.

into one of the three groups: major reaction, minor reaction, or no reaction. When platelet and plasma amine oxidase were added into the discriminate function analysis, 25 of the 32 individuals (78.1%) were correctly categorized. Adding in age as a fourth variable had no effect. A ratio was formed by dividing an individual's COMT value by the product of his two amine oxidase values (platelet and plasma), in order to obtain a single value that would represent an individual's capacity for methylation versus oxidation. The resulting ratio was able to correctly place 28 of the 32 individuals (87.5%) into one of the three groups (table 2). Figure 1 is a point graph of individual ratios in the three groups.

Seven of the 99 individuals who were admitted to the unit developed psychotic reactions to disulfiram. Five of these 7 were classified according to *DSM-II* as toxic-schizophrenic psychoses, one as a toxic-manic psychosis, and one as a toxic-depressive psychosis by a psychiatrist blind to the study. All 7 individuals had been on 500 mg/day of disulfiram, an incidence significantly different from that of the patients who received 250 mg/day of the drug ($p<.01$, Fisher exact test). Five additional individuals had developed nonpsychotic behavioral changes that responded to a reduction in disulfiram dosage. Four of these reactions consisted of fatigue and lethargy and the fifth of mild confusion and difficulty concentrating. Two of these individuals had been taking 500 mg/day and the remaining 3 were taking 250 mg/day. As a check on randomization in the different dosage groups, a medium split was done for each set of pretreatment enzyme values and a chi square done to test for equal distribution in the two dosage groups. No significant differences were found in the two dosage groups for the three enzymes measured. After 3 and 6 weeks on di-

TABLE 2
Plasma AO, Platelet MAO, and Red Cell COMT Activities in Individuals With and Without Reactions to Disulfiram

Type of Reaction	Plasma AO (nmol/ml/hr)		Platelet MAO (nmol/10^8 platelets/hr)		Red Cell COMT (nmol/ml packed RBC/hr)		COMT / Platelet MAO × Plasma MAO (ratio)	
	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
Psychotic (N=7)	12.46[a]	.92	4.61[a]	.54	8.34[b]	1.19	.175[c]	.042
Nonpsychotic (N=5)	15.34	1.49	6.64	1.91	3.92	.90	.053	.013
No reaction (N=20)	17.60	.90	7.98	.61	3.58	.53	.029	.006

[a]$p<.01$ compared to no-reaction group.
[b]$p<.001$ compared to no-reaction group.
[c]$p<.0001$ compared to no-reaction group.

sulfiram, MAO, AO, and COMT were not significantly different from baseline, indicating that disulfiram, 250 or 500 mg/day, apparently had no direct effect on these enzymes.

DISCUSSION

Significant monozygotic versus dizygotic twin differences and sibling-sibling correlations have been found in humans for platelet and plasma amine oxidase and COMT activity in plasma and blood cells, indicating genetic influence (25–27). There are also similar data that indicate genetic contributions to schizophrenia and bipolar affective disorder (28). Individual differences in neurotransmitter-related enzymes would provide one possible mechanism for mediating genetic vulnerability for these disorders. COMT has been found by some investigators to be elevated in schizophrenia (29–31) and both unipolar and bipolar affective disorder (32), but other studies have failed to confirm these findings (33–37). In the case of platelet MAO, many investigators have found decreased activity in schizophrenics (19, 38–59) and bipolar affective disorder (17, 41, 44, 60). Other investigators, however, have not confirmed these results (51, 61–71). The reason for these discrepant results is as yet unclear. Plasma amine oxidase has not been so intensely studied, and in the two available studies (72, 73) no differences in plasma amine oxidase have been reported between depressed or manic patients and controls.

Enzyme measurements in other areas of medicine have proved to be of value in predicting side effects from drugs (e.g., isoniazid toxicity in patients with low acetylase activity and succinylcholine supersensitivity associated with low plasma pseudocholinesterase activity). The present study shows that platelet and plasma amine oxidase activity and red cell COMT activity may be of value in predicting behavioral side effects from disulfiram administration. Since these enzymes are not necessary for the metabolism of disulfiram, it is unlikely that differences in enzyme activity resulted in differences in circulating levels of disulfiram to account for the adverse reactions. Disulfiram, however, is a potent inhibitor of dopamine beta hydroxylase and would be expected to result in a decrease of norepinephrine and an increase of dopamine in noradrenergic neurons, as has been shown in animals (74). We have previously shown that patients with psychotic reactions to disulfiram had significantly lower pretreatment cerebrospinal fluid levels of dopamine beta hydroxylase than patients with no adverse reactions (75), which suggests that the noradrenergic system is involved in the emergence of psychotic symptomatology. It is unlikely that the decreased formation of norepinephrine is responsible since MAO inhibitors, which would be expected to increase central norepinephrine, are associated with an increase in disulfiram-induced psychosis (76, 77).

The other effect of inhibiting dopamine beta hydroxylase is to cause a build-up of dopamine in noradrenergic neurons (74). Stein and Wise (78) have postulated a similar mechanism in schizophrenia—a genetic defect in dopamine beta hydroxylase resulting in a build-up of dopamine in noradrenergic neurons that could then be auto-oxidized to 6-hydroxy dopamine, a neurotoxin. Some investigators (19, 38–59) have also shown patients with schizophrenia to have low platelet MAO. If low platelet MAO is a reflection of low MAO activity in other tissues, such as the brain, we could speculate a relationship between MAO activity and the ability to metabolize dopamine in noradrenergic neurons, with low MAO activity resulting in a greater build-up of dopamine in noradrenergic neurons. The results of the present study are consistent with this general formulation. Individuals who developed disulfiram psychosis had significantly lower platelet MAO and plasma AO activity, possibly reflecting a generalized decrease in amine oxidizing capacity. They also had significantly elevated red cell COMT activity. Again, there is no evidence that red cell COMT activity is correlated with brain COMT activity, although such evidence does exist for other enzymes (79, 80). The most intriguing hypothesis stemming from the present study is that the ratio of various metabolizing enzymes, i.e., those involved in methylating, oxidizing, and β-hydroxylating, is important in the manifestation of psychotic or toxic symptomatology. Both MAO and dopamine beta hydroxylase have been shown to be important degradative enzymes necessary to metabolize trace amines and abnormal metabolites (81, 82). Also, COMT in conjunction with other

methylating enzymes may produce abnormal metabolites that might result in psychotic symptomatology if they are not rapidly metabolized (83). An individual's ability to tolerate an amine load, tolerate a drug that alters the biogenic amines, or even tolerate stress, may in part depend on the ratio of methylating ability to oxidizing or β-hydroxylating ability. The validity of such a hypothesis awaits further investigation.

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Reduced Cyclic AMP Production in the Blood Platelets from Schizophrenic Patients

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The authors assessed α-adrenergic receptor function in blood platelets from chronic schizophrenic patients and normal control subjects. The number of receptors was measured by the specific binding of the α-adrenergic antagonist [^{3}H]dihydroergocryptine to the platelets. A physiological response of the platelets to agonist occupancy of the α-adrenergic receptors was measured by the norepinephrine inhibition of prostaglandin E_1 (PGE_1)-stimulated cyclic AMP (cAMP) production. cAMP production in male schizophrenic patients was lower than in normal male subjects. α-Adrenergic receptor function was similar in patients and normal control subjects of both sexes. Normal male subjects had about 1.5 times the number of α-adrenergic receptors as normal females and generated about 1.8 times the quantity of PGE_1-stimulated cAMP.

THE IMPLICATION of catecholamines in the pathogenesis of schizophrenia (1, 2) suggests that an assessment of catecholamine neurotransmitter function in schizophrenic patients might be of interest. The α-adrenergic receptors on human platelets (3) share pharmacological properties with α-adrenergic receptors from brain and other tissues (4–6), and norepinephrine inhibition of prostaglandin E_1 (PGE_1)-stimulated cyclic AMP (cAMP) production in platelets measures the physiological response to occupancy of the α-adrenergic receptors (3).

Platelets isolated from a fresh blood sample were used to measure the specific binding of [^{3}H]dihydroergocryptine (^{3}H-DHE) to platelet membranes and the production of cAMP under basal and stimulated conditions in whole platelets (3). The reliability of the measurements over time was assessed in controls on two separate blood samples. The correlation coefficient between the two values obtained for PGE_1-stimulated cAMP production in 16 subjects was 0.561 ($p<.05$) and for ^{3}H-DHE binding in 7 subjects was 0.781 ($p<.05$). At the concentration of ^{3}H-DHE used to measure specific binding (35 nM, the K^d for ^{3}H-DHE binding), a change in binding would indicate an alteration either in the number of receptors or in the affinity of the receptors for ^{3}H-DHE (3). Because the affinity of the α-adrenergic receptor for ^{3}H-DHE is similar in normal and schizophrenic individuals (unpublished data), ^{3}H-DHE binding measures the number of α-adrenergic receptors.

Eleven male and 9 female inpatients on a schizophrenia research unit took part in the study. All patients were diagnosed schizophrenic with four or more symptoms of schizophrenia according to the International Pilot Study of Schizophrenia (7). According to Research Diagnostic Criteria (8) 8 of 11 male patients were schizophrenic, and 3 were schizo-affective; 3 of 9 female patients were schizophrenic, and 6 were schizo-affective. The mean (ISE) age, duration of illness, and length of hospitalization were not significantly different in male and female patients (24.9±2 years, 4.4±1.4 years, and 30±18 months, respectively, and 23.4±2 years, 3.2±0.8 years, and 11.3±5, respectively).

The control subjects were blood bank donors. As there was no correlation between the age of control subjects and any of the biological variables measured, the control subjects used ranged in age from 18 to 65 years (mean=45 years).

RESULTS

Male patients had the same number of α-adrenergic receptors as male control subjects (table 1) but showed a decrease in cAMP production of 33% under basal conditions and 37%–44% with PGE_1 stimulation. The percentage of norepinephrine inhibition was similar in both groups (table 1, figure 1).

Female patients and their sex-matched controls had the same number of α-adrenergic receptors and the same level of cAMP production. The percentage of norepinephrine inhibition of PGE_1-stimulated cAMP production was the same in both groups (table 1).

Male control subjects had about 1.5 times as many α-adrenergic receptors on their platelet membranes as female control subjects. cAMP production in the plate-

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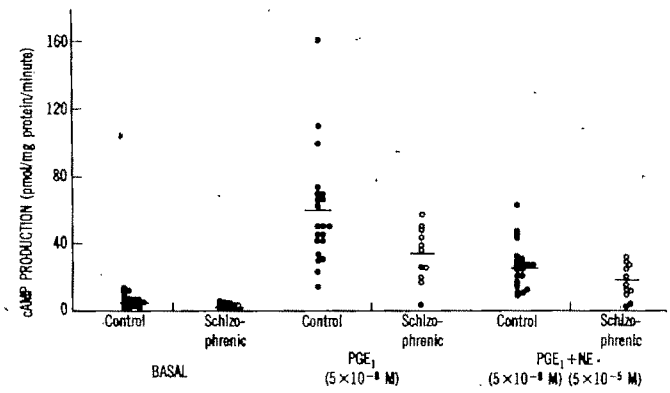
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TABLE 1
cAMP Production and ^{3}H-DHE Binding in Platelets of Schizophrenic Patients and Their Sex-Matched Controls

Sex and Diagnosis	cAMP Production (pmol/mg protein per minute)[a]								^{3}H-DHE Binding[b] (fmol/mg protein)		
	Number of Subjects	No Drug Mean	No Drug SE	PGE_1 Mean	PGE_1 SE	PGE_1+Norepinephrine Mean	PGE_1+Norepinephrine SE	%[c]	Number of Subjects	Mean	SE
Male											
Normal control	21	5.8	0.6	58.2	7	26.5	3	54	17	179.9	21
Schizophrenic	11	3.6[d]	0.5	32.6[e]	5	16.5[d]	3	49	11	187.4	24
Female											
Normal control	17	4.6	1	31.9	5	13.7	2	57	12	122.2	11
Schizophrenic	9	4.6	0.8	28.4	5	13.1	3	55	9	191.2	35

[a]The concentration of PGE_1 was 5×10^{-8} M and of norepinephrine was 5×10^{-5} M; for stimulated cAMP production male controls differed significantly from female controls with and without norepinephrine ($p<.01$ and $p<.005$, respectively).
[b]Binding data were not available for all controls for whom cAMP data were measured; male controls differed significantly from female controls ($p<.05$).
[c]Norepinephrine inhibition of PGE_1-stimulated cAMP production.
[d]Male schizophrenics differed significantly from their controls ($p<.05$).
[e]Male schizophrenics differed significantly from their controls ($p<.025$).

FIGURE 1
cAMP Production in Male Schizophrenic Patients and Control Subjects[a]



[a]cAMP values are means of triplicate measurements under three conditions: with no drug, with PGE_1 (5×10^{-8} M) and with PGE_1 (5×10^{-8} M) and norepinephrine (5×10^{-5} M). The differences are significant at $p<.05$ for PGE_1-stimulated cAMP production and at $p<.025$ for basal and norepinephrine-inhibited PGE_1-stimulated cAMP production.

lets of male control subjects was the same as that of female control subjects under basal conditions, but PGE_1-stimulated cAMP production was almost twice as great. The percentage of norepinephrine inhibition of PGE_1-stimulated cAMP production was similar in male and female control subjects (table 1).

DISCUSSION

Because normal male control subjects have more α-adrenergic receptors and greater PGE_1-stimulated cAMP production than normal female control subjects, the schizophrenic patients were compared with their sex-matched controls. Whether sex hormones contribute to the observed differences between males and females requires further study. α-Adrenergic function does not appear to be abnormal in the platelets of schizophrenic patients as the number of α-adrenergic receptors and the percentage of inhibition by norepinephrine of PGE_1-stimulated cAMP production is the same in the patients and their sex-matched controls.

Male, but not female, schizophrenic patients produce less cAMP in their platelets under basal as well as stimulated conditions. The low level of cAMP in male, but not in female, schizophrenic patients may indicate that the level of cAMP production is unrelated to schizophrenia. On the other hand, if the decrease in cAMP production is related to schizophrenia, the finding would lend support to the hypothesis that schizophrenia is not a single disease but a group of diseases with common clinical features and different etiologies (9). Within the disease spectrum decreased cAMP production might characterize a form of schizophrenia manifested only in males.

The specificity to schizophrenia of decreased platelet cAMP production can be assessed by comparing platelet cAMP production in the platelets from schizophrenic patients and patients with other psychiatric disorders.

Some factors that could contribute to the decreased cAMP production observed in male schizophrenic patients were considered. Neither the age, the duration of illness, nor the length of hospitalization in male or female schizophrenic patients showed any correlation with the cAMP level. It seems unlikely that neuroleptic treatment is responsible for the decrease in cAMP production for several reasons. Chlorpromazine (10^- M) incubated for 5 minutes in vitro with platelets from four normal individuals did not alter cAMP production. In patients on neuroleptic therapy most of the platelets would not have been exposed to the drugs since the patients had been drug-free for at least 2 weeks prior to the study and platelets have a turnover time of about 10 days (10). Finally, the female patients whose demographic characteristics corresponded roughly with those of the males and who had received as well as could be determined, comparable amounts of neuroleptics, showed no such decrease.

The possibility that low cAMP production might be due to an inherited trait could be investigated by mea

suring whether cAMP production in the relatives of male schizophrenic patients is significantly less than that in normal control subjects.

Murphy and associates (11) have shown that norepinephrine inhibits PGE_1-stimulated cAMP production in platelets from normal subjects and patients with affective illness treated with lithium carbonate. PGE_1-stimulated cAMP production was reduced in the lithium-treated patients but was not measured in patients who were not receiving the drug. Pandey and associates (12) found elevated PGE_1-stimulated adenylate cyclase activity in 5 acute schizophrenic patients and no difference between PGE_1-stimulated adenylate cyclase activity in control subjects and 4 chronic schizophrenic patients. Platelets from male and female subjects were not studied separately. Pandey and associates (13) found no change in basal, PGE_1-stimulated, or α-adrenergic-mediated inhibition of adenylate cyclase activity in platelets from patients with affective illness; however, they found a diminution in β-adrenergic-mediated stimulation of adenylate cyclase activity in leukocytes from patients with affective illness. Abdulla and Hamadah (14) observed that PGE_1 synthesis was very low in platelets from schizophrenics compared with platelets from normal controls or patients with affective illness. Low concentrations of prostaglandins (or prostacyclins), in addition to diminished responsiveness of cAMP production to prostaglandin stimulation, might result in an even more profound cAMP deficit in the platelets of male schizophrenic patients. Peroutka and associates (15) showed that in the rat most neuroleptics, including chlorpromazine, had α-adrenergic antagonist activity. In the current study chlorpromazine has no α-adrenergic antagonist activity against norepinephrine-inhibited PGE_1-stimulated cAMP activity in human platelets.

In many cells, including platelets and neurons, cAMP increases, through phosphorylation, the activity of cell proteins (16, 17). It is possible that the decrease in cAMP production is limited only to platelets. Perhaps decreased cAMP production, such as that seen in the male schizophrenic patients, is a more general phenomenon resulting in deficits in neuronal systems etiologically important to schizophrenia.

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EDITORIAL

Care of the Chronically Mentally Ill—Still a National Disgrace

WRITING IN THIS SPACE in 1973, Robert Reich, M.D., called the care and treatment of the severely and chronically mentally ill a national disgrace (1). Since then things have grown worse.

They have grown worse because, while deinstitutionalization has proceeded, states still have not found a way to enable monies to follow patients from state hospitals to community settings and because with the recent fiscal crunch, all levels of government have again placed a lower priority on mental health than on education, highways, police, fire, and sanitation. They have grown worse because with the budgetary constraints and the threat of loss of accreditation to state hospitals, states have predictably shunted money into state rather than community settings and, because with the current emphasis on cost containment in health care, chronic patients in need of long-term supportive care and treatment have been the first to be discriminated against by reimbursement mechanisms such as Medicaid. Finally, they have grown worse because despite the lesson learned over the past 20 years that chronic patients need a vast array of supportive services including housing, income, education, and vocational and social rehabilitation, in addition to a full range of medical and psychiatric services, there are only isolated examples in the United States of programs providing such comprehensive care.

Historically, many attempts have been made to care for the chronically mentally ill. A succession of institutions have been proposed as *the* solution to the problem. Community mental health centers were designed to replace state hospitals, which replaced almshouses and workhouses, which replaced prison-like outhouses. Each "solution" has failed to solve the problem. And the latest fashion, community care, will join their company if it is allowed to proceed in its present form. It may be a debatable question (2, 3) whether or not state hospitals have improved over the last 20 years, despite the fact that they now house fewer than one-third their previous population and have vastly improved staffing, now approaching a 1:1 ratio. But there is no question that the current care provided to chronic patients in the community, whether deinstitutionalized or never admitted, is inadequate.

In 1975 APA charged an Ad Hoc Committee on the Chronic Mental Patient to study the issues and to propose a national public policy. The committee commissioned eight new studies on key areas: where the chronic patients are, their needs, the programs that help them, the obstacles that impede effective treatment, the economic issues, the legal issues, what levels of government should have what responsibility, and the pros and cons of case management. These studies served as a basis for a national invitational Conference on the Chronic Mental Patient (4), held in January 1978 in conjunction with the President's Commission on Mental Health. A report emanating from the conference, in the form of a call to action for a public policy, has been reviewed and approved by all components of the APA (district branches, committees, Councils, the Assembly, and the Board of Trustees) and is published in this issue of the *Journal* as a position statement.

The committee sought to achieve consensus but in certain instances took controversial positions, which may seem provocative to some readers. For instance, the statement calls for an end to the government's conflict of interest in both

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operating and contracting for mental health services (at federal, state, and local levels); it calls for *local* government to designate who shall provide services for chronic patients. It also calls for private practitioners to be encouraged in caring for this population; for the adoption of a case management approach rather than case managers; and for an end to discriminatory reimbursement for long-term treatment and chronic care, coincident with the introduction of innovative methods (e.g., a voucher system) to provide appropriate care settings such as halfway houses and apartments for patients, rather than nursing or board-and-care homes.

The bulk of the statement, while sweeping, detailed, and comprehensive, will probably not incur dissent, for it addresses in detail the measures needed to reverse the current situation. It suggests that financing may be the key to most of the problems encountered by chronic patients. More money is not necessarily needed, but existing monies certainly must be redeployed from restrictive, inappropriate settings to a range of graded opportunities. The statement also suggests that discrimination against the chronically ill in health insurance, housing, vocational rehabilitation, and employment needs to be reversed. It redefines roles for all levels of government: the federal government to become responsible for national priorities and guidelines; state governments for regional priorities and plans; local governments for designation of providers; and the actual services provided by contract by the smallest entities possible (e.g., catchment areas). It challenges psychiatry to engage in two previously neglected areas: training and research related to chronic illness. The statement recommends that psychiatric residencies incorporate experience with the chronically mentally ill in community settings and that research efforts be directed toward patient outcome studies, comparing different treatment modes and analyzing model service systems. It also encourages psychiatrists and psychiatry to become involved in planning and delivering services to chronic patients, even though these patients' primary problem may involve disability, not disease. And finally—but most important in the long run—it calls for a vast program of professional and public education regarding chronic mental illness and disability, without which none of the above will be realized.

The statement was intended as a comprehensive blueprint for public policy. It will be successful only if it is translated into effective action through legislation, governmental procedures, and professional initiative. While deriving *from* APA, it is not solely *of* APA, and it will become a reality only if all psychiatrists and all mental health workers and professional organizations fight for the adoption of each of its broad policies and detailed recommendations.

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BRIEF COMMUNICATIONS

State and Trait in Psychiatric Practice

BY IRL EXTEIN, M.D., AND MALCOLM B. BOWERS, JR., M.D.

The authors present a schema for conceptualizing psychiatric illness in terms of state and trait disorders. These disorders are relatively independent components, one or both of which can be present in one patient. They usually require treatment by different methods. State disorders, such as schizophrenic psychosis, mania, and depression, are time-limited and autonomous and respond better to pharmacological than psychosocial therapy. Trait disorders, such as neurotic and characterological disturbances, are long-lasting and respond better to psychosocial therapies. The authors discuss the implications of this schema for the integration of pharmacological and psychosocial treatments.

WE PROPOSE a schema for conceptualizing psychiatric illness that has implications for teaching and therapy. The basic approach to clinical syndromes embodied in our schema is implicit in much of contemporary psychiatric practice. It is our aim to refine and make explicit this approach by organizing it into a schema around the focal concepts of state disorder and trait disorder.

So far as we can determine, the state-trait dichotomy was first used by Spielberger (1), who distinguished anxiety in the present (state anxiety) from the frequency of anxiety in the past (trait anxiety). Our own interest in this distinction (2) stems from clinical work and neurobiological concepts of plasticity (3). These have led us to use the terms "state" and "trait" to refer to two qualitatively different processes. The concept of "state" is, in our schema, related to a plasticity in biobehavioral systems that allows reversible and relatively stereotyped behavior and which accommodates biobehavioral rhythms, central nervous system activation, depression, and the like. It has been called "activational" plasticity. The concept of "trait" is related to a different kind of plasticity that has been called "organizational" and refers to that continuing process of interaction between genetic endowment and environment (with critical periods implied) that results in organismic development and maturation. These concepts were originally developed in the context of animal research dealing with the effects of hormones on sexual gender differentiation (organizational plasticity) and mating behavior (activational plasticity) (3, 4). Our attempt here is to propose their relevance to clinical psychiatry.

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THE STATE-TRAIT SCHEMA

We consider disorders of state and trait to be relatively independent components of psychiatric illness (2) that may be present alone or together in one individual. They must to a large extent be diagnosed separately and treated by different techniques. "State disorder" refers to those qualitatively different mental conditions which tend to be time-limited and autonomous in that, once present, they are relatively insensitive to environmental input. They respond poorly to verbal or other psychosocial treatment but usually respond to medication or other somatic treatment (5–7). Specifically, state disorders include schizophrenic and other functional psychoses, mania, major depression, severe anxiety, acute organic brain syndrome, and seizures. Most of these state disorders probably involve a variety of environmental stressors acting on genetic or other biological vulnerability (8–12). "Trait disorder" refers to those dysfunctional qualities which individuals tend to develop and carry throughout life and which become manifest as predictable patterns of

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interaction and response to stress. Such patterns tend not to be responsive to medication but respond better to psychosocial treatment (5–7). Trait disorders include neurotic and characterological disturbances, including subnormal intelligence, and seem to be learned as a result of the interplay between innate characteristics and parental and social experience (6).

It is important to consider how this schema can be used over time, because psychiatric illnesses are not static entities and treatment approaches must shift. The first question to ask in evaluating a patient might be, "Is a state disorder present?" If the answer is "yes," then medication is indicated. As a rule, we do not think that effective psychosocial intervention can be made in a patient with a state disorder. Patients can not be "talked" out of state disorders, although doctor-patient interaction is critical to patient care in the early stages of state disorders, such as psychosis or depression, for example. Our point is that the definitive treatment of state disorders is not primarily psychosocial. Because the symptoms of a state disorder can obscure or highlight excessively the usual personality traits of an individual, the possibility that a trait disorder exists cannot be accurately assessed while a state disorder is present. Even if a clinically significant trait disorder does exist in an individual with a state disorder, one must usually wait to treat the trait disorder until the state disorder has cleared. For example, it is difficult to assess or explore insecurities about interpersonal relationships in a patient who is manic or has a paranoid psychosis. Once the state disorder has cleared, or if a state disorder was never felt to be present, the question can be asked, "Is a trait disorder present?" This aspect of diagnosis or evaluation is usually made by examining the patient's patterns of interaction with significant persons in his or her life. If the answer is "yes" then the appropriate psychosocial intervention can begin.

The role of psychosocial treatments in psychiatry is controversial, and the place given them in our schema requires clarification. In discussing state disorders, we are not saying that a patient with a schizophreniform psychosis, for example, does not respond to some degree to the environment and the people in it or that the content of the psychosis cannot at times have authentic personal meaning. Rather, we are saying that the psychotic process itself cannot be significantly altered by psychosocial means (5–7, 13). In addition, while stating that trait disorders should be treated by psychosocial methods such as psychotherapy, we acknowledge that the efficacy of psychotherapy has been difficult to document (7, 14).

We do not mean to neglect the importance of life stress and developmental issues. These components can be considered as separate dimensions of psychiatric illness and are compatible with our state-trait distinction. Stress can certainly precipitate a state disorder in an individual genetically or otherwise biologically vulnerable (15, 16), as when a loss precipitates a depression or the first year away from home is associated with the onset of schizophrenic psychosis. However, the importance of life stress cannot be adequately assessed or taken up therapeutically until the state disorder has been treated. Stress can also exaggerate personality trait disorders. In some disturbed patients a pathological state or trait disorder is not present, and the disturbance can be best described as a situational adjustment or developmental crisis. In such cases psychosocial therapies, including crisis intervention, are usually indicated (17).

Disorders of state and trait can mimic each other and make the choice of proper treatment modality difficult. For example, the behavior, mood, and thinking of patients suffering from the borderline syndrome often fluctuate markedly (18). Sometimes psychotropic medications are tried when, in fact, a drug-responsive state is not present and the fluctuations are best treated as part of the character disorder by psychosocial means. At other times drug-responsive mood disorders, psychoses, or seizures in a patient with the borderline syndrome are missed because of the continuing and obscuring presence of a severe character disorder. These examples underline the importance of accurate knowledge of a patient's baseline personality traits in diagnosing a superimposed state disorder and emphasize the importance of therapeutic medication trials in ambiguous cases.

The use of this schema might help foster complementarity of assessment and treatment ideologies in the clinical setting. The issue of drugs versus psychotherapy may be in large measure a false dichotomy. More relevant to clinical care are issues involving the use of the proper treatment modality for the appropriate component problem at the proper time in the course of a psychiatric illness. For example, consider a patient with a severe depression, of the type we define as a state disorder (19), superimposed on chronic marital difficulties. This patient would probably benefit little from psychotherapy initially but might benefit a great deal once his or her depressive state had been treated with antidepressant medication. Recent work with women suffering from unipolar depressions supports the notion of state and trait disorders as independent target problems requiring different treatment. Antidepressants have been shown to be more useful than psychotherapy in alleviating depressive symptoms and maintaining symptomatic remission (5, 20). However, psychotherapy has been found to be more useful than antidepressants in improving social functioning after treatment of the acute depression with medication (21).

The use of this two-dimensional schema cuts across conventional one-dimensional diagnostic categories. The neuroses and character disorders are conceptualized as trait disorders, although again with the caveat that the existence of neurotic traits does not exclude the possibility that a state disorder may develop and require treatment. Mood disorders, both bipolar and unipolar, are conceptualized as state disorders. However, patients with mood disorders may suffer from

character pathology or social maladjustment that may require psychosocial treatment. The important point is that these traits are to a large degree independent of the state disorder at the clinical level. Schizophrenia might be conceptualized as having two components: a trait disorder with chronic disturbance of thought, mood, and social functioning, on which are superimposed recurrent state disorders marked by schizophreniform psychotic episodes. Antipsychotic medications are more useful in the acute and prophylactic treatment of the psychotic episodes than in the treatment of the schizophrenic traits (5, 6, 13, 22). Recognition of the many patients who have one or more schizophreniform psychotic episodes with otherwise normal functioning emphasizes the relative independence of the schizophreniform psychotic state from the schizophrenic character traits (2, 23, 24). Others have tried to make this same distinction by dividing schizophrenia into reactive and process types (23). The definitions of schizophrenia and mood disorder in our schema are rooted in those of Kraepelin, who contrasted the chronicity of schizophrenia (25) with the episodic nature of manic-depressive illness (26).

The use of this conceptual schema might clarify certain issues concerning the proper use of psychotropic medications. One way to conceptualize the role of psychotropic medications is to say that their primary value is in the treatment and prophylaxis of state disorders. In cases in which it is difficult on clinical grounds alone to determine whether a state disorder is present, a therapeutic drug trial can be helpful. It is often useful for future treatment to define a state disorder retrospectively as a disorder characterized by lack of response to psychosocial methods but responsive to a certain drug regimen. The best that can be expected from the pharmacological treatment of a state disorder is a return to baseline (premorbid) functioning. Thus it is crucial to know a patient well and to know his or her baseline in order to assess accurately both the need for medication and the therapeutic end point. The use of medication as prophylaxis against the recurrence of future episodes of a state disorder should be distinguished from the treatment of a particular episode (20, 27). It is during this medication maintenance phase of the treatment of patients with state disorders that psychotherapeutic work, if needed, can be pursued. The importance of a therapist's psychosocial skills in the proper use of medication, including securing patient compliance, should not be ignored.

DISCUSSION

Although for conceptual purposes we have emphasized the independence of state and trait disorders, these processes may in certain ways be related. For example, it is conceivable that the same biological process responsible for manic psychosis also functions in an "organizational" fashion in the development of the personality of the individual with manic-depressive illness. Questions related to the underlying processes in the determination of state and trait components of clinical syndromes await further research. In addition, character traits may color the expression of a state disorder and can affect a patient's ability to cope with and cooperate in the treatment of a recurrent state disorder, such as manic-depressive illness or temporal lobe epilepsy. On the other hand, repeated episodes and hospitalization for these and other recurrent state disorders can affect character traits such as self-confidence and social skills.

There are certain psychiatric disturbances that do not fit well into the schema presented here. There are state disorders that do not respond to current somatic treatment, such as drug-resistant psychosis in schizophrenia (28). Certain trait disorders, such as antisocial character disorder (29), respond relatively poorly to current psychosocial treatment. Minimal brain dysfunction is an atypical disorder in that it has certain trait characteristics but often responds to stimulant medication (30). There are other disorders that we have not attempted to integrate into our present schema, including sexual dysfunction, psychophysiological disorders, alcohol and drug abuse, and chronic organic brain syndrome. However, we feel that the state-trait distinction as presented here may be useful in the analysis of these problems.

In conclusion, we have conceptualized major psychiatric illness as having at least two major independent components that usually require treatment by different methods. The ideas in our schema are consistent with the practices of many contemporary psychiatrists who use pharmacotherapy and psychosocial therapies for generically distinct clinical problems (6). The precision gained by using this schema is helpful in establishing the proper indications for and timing of pharmacological and psychosocial therapies.

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Suicide Among U.S. Women Physicians, 1967–1972

BY FERRIS N. PITTS, JR., M.D., ARTHUR B. SCHULLER, M.D., CHARLES L. RICH, M.D., AND ANDREW F. PITTS

The authors examined AMA records of the deaths of 751 women physicians during 1967–1972. Forty-nine (6.56%) committed suicide. The suicide rate for women physicians (40.7 per 100,000 per year) was higher than for men physicians and about four times that for white American women of the same age. From the known morbid risk for suicide among women with primary affective disorder, the authors calculated that about 65% of American women physicians have primary affective disorder.

CRAIG AND PITTS (1) analyzed suicide among physicians by examining the records of 8,372 consecutive physician deaths ascertained by the AMA during 1965-1967. To our knowledge, that was the first time the greatly higher suicide rate of women physicians was recognized and reported.

In the Craig and Pitts study the numbers of women physicians who committed suicide were so small (base population 21,000, 297 deaths, 17 definite suicides) that we conducted a second study to see if the high rate of suicide is constant or variable. We examined all AMA records of deaths of women physicians for the 5-year period 1967–1972. As this report illustrates, the rate of suicide for women physicians continued to be high and nearly constant from year to year throughout this five-year period.

METHOD

The comments on and description of AMA physicians' records and *JAMA* death notices by Craig and Pitts (1) still apply with one exception: physicians are better known to the AMA now as time runs out on the pre-Flexner Report M.D.s, and many fewer need to be declared dead because of their advanced age and unknown address. In the earlier study 5% of physician deaths were by declaration, but such death declarations have declined to approximately 1%. Thus, essentially all physician deaths (99%) are now known to the AMA. Approximately 5% of physician deaths are still reported to the AMA too late for obituary in *JAMA*; about half of these late reports include cause of death; cause of death is not sought for the other 2.5%. Our current sample includes knowledge of cause of death for more than 95% of physician deaths, rather than 92.5% in the previous sample studied.

Because less than 3% of physicians' deaths are suicides, a greatly increased suicide rate in the 5% of physician deaths not reported in *JAMA* could significantly increase the rate of suicide among physicians. A total absence of suicides in this 5%, on the other hand, would not reduce the suicide rate appreciably.

Analysis of Data on Physician Deaths

We examined the materials collected by the *JAMA* deaths editor to obtain as clear a picture as possible of the cause of death. Differentiating suicide, probable suicide, and possible suicide was particularly important because published *JAMA* obituaries are not always accurate in these matters. *JAMA* publishes the cause of death given on the death certificate if available regardless of other circumstances, and in a number of instances of obvious suicide the attending physician chose a euphemistic cause of death. Thus, "asphyxia" was recorded in place of suicidal hanging, "accidental gunshot" instead of self-inflicted gunshot wound in the head, and "accidental drowning" in place of suicide by drowning. These reports have occasionally been made even when there has been collateral evidence of suicidal intent—a suicide note or circumstantial evidence that the deceased physician took great pains to be isolated for sufficient time to achieve "accidental death."

We reviewed the materials collected by the deaths editor for those physician deaths reported by obituary in *JAMA* between May 22, 1967, and May 30, 1972. We also reviewed all available records on deaths of physicians reported to the AMA too late for *JAMA* obituary during these 5 years. This examination of the original data enabled us to clarify many of the ambiguous or euphemistic causes of death listed in *JAMA* obituaries and to increase the category of definite suicides by 18.5% for all physicians and 20.0% for women physicians.

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Our criteria for "definite suicide" were hierarchical. First, a certification of suicide on the death certificate by the attending physician was automatically counted as suicide. A certification by letter from the deceased physician's personal physician, colleague, or physician relative that the death was a suicide (when there was no death certificate available or the death certificate gave a euphemistic cause) was also automatically counted as a suicide.

In the absence of other information, a written statement from the next-of-kin or executor that the physician had killed himself or herself was sufficient for classification as suicide. There were instances in which there was enough information available that we felt justified in calling the death a definite suicide even though the death certification and medical report indicated otherwise, e.g., when a newspaper article stated that a suicide note was found but the death certificate stated that the death was accidental; there were only 2 such circumstances (4% of suicides tabulated) among the deaths of women physicians.

When none of the above criteria were met but there was sufficient evidence that the deceased physician had been psychiatrically ill and had died under suspicious circumstances, we classified the death as "probable suicide." There were only 4 probable suicides of women physicians (8% of total tabulated), all were extremely likely to have been suicides, and the numbers did not affect any of the statistical differences calculated; therefore the 4 probable suicides were included with the 45 definite suicides.

Probabilities given in this report are based on either the standard error of the difference of means or chi square with Yates correction. Differences at the .02 level of statistical confidence are considered significant.

RESULTS

In the five years between May 22, 1967, and May 30, 1972, the deaths of 16,722 physicians (16,063 male, 659 female) were reported by *JAMA* obituary; 2,008 (1,916 male, 92 female) were reported to the AMA too late for obituary. The total number of deaths ascertained by the AMA during this five-year period, then, was 18,730 (17,979 male and 751 female) and a significant percent (10.7% male, 12.3% female, 10.7% total) never appeared in the *JAMA* obituary columns. Fully 20% of the suicides of women physicians were among those 12.3% not appearing in a *JAMA* obituary.

The crude annual death rate was 1,261 per 100,000 per year (3,596 per year per 285,131) for men and 623 (150 per year per 24,088) for women physicians (on December 31, 1969, the midpoint of this study, there were 24,088 women physicians in the United States and its possessions [2]).

The percentage of these 751 deaths of women physicians due to suicide was 6.52; these 49 suicides gave a crude annual death rate by suicide of 40.7 per 100,000 for the five-year period; the death rate by suicide of white women over age 25 for this same period was 11.4 per 100,000 ($p<.0001$) (3).

In comparison, 543 of 17,979 male physician deaths by suicide gave a percentage of 3.02 and a crude annual death rate of 38.1 per 100,000.

The age at which women physicians committed suicide ranged from 25 to 79. The median was 47 years and the mean was 47.8±2.0 (SEM); clearly, the majority of these premature deaths occurred during what should be years of productive professional life.

Drug overdose is used significantly more frequently by women physicians for committing suicide than by the general U.S. female population matched by age and race (71.1% compared to 45.7%), and there is a corresponding lower frequency of gunshot suicide among women physicians ($p<.001$). This increased tendency to use drugs rather than guns is also seen regularly among male physician suicides.

Five of the 45 women physicians who definitely committed suicide were certified medical specialists (one each in ophthalmology, pathology, pediatrics, preventive medicine, and radiology). Practice preference grouping showed 15 in family practice; 8 in residency training; 5 in internal medicine; 4 each in pediatrics and psychiatry; 3 in pathology; 2 in preventive medicine; and 1 each in anesthesiology, obstetrics and gynecology, ophthalmology, and radiology. Since family practice was a residual category no special meaning can be attached to the apparent clustering; otherwise, the numbers in each category were too small to determine anything other than suicide is a cause of death for some women physicians in all types of practice. Apparently it is not the type of practice but the type of woman that determines whether or not she commits suicide. Of the 8 women in training who committed suicide, 2 each were in psychiatry and internal medicine, and 1 each were in pathology, radiology, pediatrics, and obstetrics-gynecology.

DISCUSSION

The rate of suicide among U.S. women physicians in our study does not differ from that of Craig and Pitts's study (1). In their study, 17 of 297 (5.72%) consecutive deaths of women physicians were definite suicides. In this study, 49 of 751 (6.52%) such deaths were suicides. Such close agreement and the constancy of annual occurrence demonstrate that there is a specific process that results in a suicide rate for women physicians approximately four times that of the general population of U.S. white women over age 25. It is about twice that for divorced women over age 70, the demographic group of women in the general population with the highest known suicide rate.

If this high suicide rate for women physicians is constant, it must, then, be due to a specific process, or more than one process, constantly related. In Robins and associates' study (4) of individuals who committed

suicide, 75% were diagnosed, according to strict criteria, as having either primary affective disorder or alcoholism. The remainder had probable affective disorder or probable alcoholism (as a result of insufficient data for strict diagnostic tests). Therefore, since it appears that nearly all individuals who commit suicide are either primary affectively disordered or alcoholic (4) it is highly likely that women physicians who commit suicide have one or both of these conditions. Pitts and Winokur (5) concluded that suicide is the cause of death in 16% (10% of women, 25% of men) of deaths of persons with primary affective disorder. They also concluded that in those alcoholic individuals who commit suicide, alcoholism is a symptomatic consequence of primary affective disorder. If Pitts and Winokur's conclusions are correct then nearly all suicides can be considered the outcome of primary affective disorders, and a morbid risk figure for primary affective disorder for any population can be calculated from the percentage of deaths due to suicide. Craig and Pitts (1) calculated the morbid risk for primary affective disorder to be 10.6% for male and 57.2% for female physicians. The morbid risk for primary affective disorder in women physicians calculated from our current data is 65.2%: 6.52%, the suicide rate among deaths of women physicians, divided by 10%, the suicide rate among deaths of women diagnosed as having primary affective disorder (1, 5). We predict that structured interview studies of women physicians will show that a very high proportion (nearly two-thirds) have primary affective disorder.

We must conclude that there is (or has been) some association between affective disorder and selection of a medical education and career by U.S. women. The rapidly increasing number of women in U.S. medical schools and medicine (2), consequent to the encouragement of women to apply for admittance, may mean that the morbid risk factor for primary affective disorder in women physicians is also changing. The current larger sample of women medical students may be from a different subpopulation of U.S. women, still intelligent but perhaps less laden with primary affective disorder. Those who undertake surveys of suicide and affective disorder among U.S. women physicians must at least consider the rapidly increasing number of women physicians and the resulting skewed age distribution curve.

Steppacher and Mausner (6) also concluded that women physicians have a higher suicide rate than men physicians. They discussed in detail the reliability and validity of suicide data for various populations and emphasized that women psychologists and women chemists also have higher suicide rates than their male peers. We have been unable to locate any other studies of suicide among women physicians.

Whatever the true reasons for the unexpected high suicide rate among women physicians, it is apparently a genuine finding and of heuristic importance. Systematic studies of women physicians using structured interviews and specific diagnostic criteria, with comparisons to other groups of professional women and to less specially educated women, are needed next—not unsupported speculations about "role strain" or pleas to examine the mental content of the physician who commits suicide.

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An Attempt to Objectify the Therapeutic Process: A Working Model

BY LAILA KARME, M.D., PH.D., SELMA COOPERMAN, M.D., AND THOMAS PRESTON, M.D.

On the basis of their observations in daily psychoanalytic work the authors developed five objective criteria for "bad" analytic hours. These criteria involve affect, intellectualization, isolation, lack of feedback, and dissatisfaction. The authors developed a formulation to help them preconsciously recognize the presence of these factors during therapeutic work and found that it was helpful in turning a potentially bad hour into a productive one.

THERE IS A GROWING CONCERN with objectivity in psychoanalysis and psychotherapy, especially in these days of peer review and third-party payment. The work described in this report began with an attempt to define, describe, and research the "stalemated analysis." We chose to concentrate only on neurotic patients and on established analyses. Established analyses were arbitrarily defined as those with a minimum of 100 hours, using standard analytic technique, in which both the working alliance and the transference neurosis were well established. We made these choices to avoid the complex issues involved in the opening phase of the analysis as well as the issue of analyzability. We felt that a reasonable approach would be to begin by studying the analytic material microscopically during limited time periods and by isolating specific aspects. It is not possible to observe everything that occurs in the clinical situation, nor is it desirable to do so for scientific purposes; features or variables that are relevant to the questions at hand must be selected.

In an attempt to clarify the cognitive processes involved in "working through," Horowitz (1) presented material from a single session of psychoanalytically oriented psychotherapy. He presented a method of microanalysis that focused on cognitive maneuvers which gradually led to the patient's conscious experience of initially warded-off ideas and feelings, permitting assimilation and working through. Horowitz concluded, "Minute clarifications, if multiplied, can lead to a theory of the process of change in psychotherapy. In turn, such knowledge would lead to improvement in technique."

We used a series of three to four sessions to establish criteria for a "bad" hour or a group of related hours. We maintained the common usage of describing the hours as bad or good with the ultimate goal of translating this vague and unscientific terminology into more precise, definable concepts. We selected hours we "felt" to be bad, the session immediately preceding such hours, and one or two sessions immediately following such hours. We assumed that frequent repetition of bad hours or very extended periods of them would lead to a lack of progress and, if not corrected, eventually a stalemated analysis.

What one observes in the analytic or therapeutic hour is words, intonations, postures, and gestures. The rest is inferred. Affects are inferred because we can measure or observe only their physiological components. For example, we can observe the patient's tears, but to assume the meaning of such tears is an inference. When asked about the affect accompanying the tears, the patient's answer may be that he or she is sad, happy, frustrated, angry, or regretful or even that he or she doesn't really know.

The therapist, no matter how experienced or neutral, is a *participant*-observer and as such is subject to distortions and countertransference feelings. We therefore attempted to subject the recorded observations of each of the analysts to thorough investigation and analysis. For recording, we selected the commonly used method of process notes taken during the session. This method is more reliable than notes taken after a session due to the obvious increased potential for distortion and omission, and in spite of its limitations it has certain advantages. It allows for recording the therapist's feelings, thoughts, and impressions immediately as they occur, which would not be possible in taped sessions. It is also not time-consuming or cumbersome. However, it would be desirable to use our model on tape-recorded or, preferably, videotaped sessions for the purpose of validation.

After each of the three participants had presented what they felt to be bad hours, we attempted to do the following: 1) achieve consensual validation that the

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hour was bad, 2) translate the feeling that the hour was bad into definable concepts in support of this feeling, and 3) search for the common denominators in such hours. Criteria that were not consistently found in most bad hours were rejected.

CRITERIA FOR "BAD" HOURS

After we had evaluated 60 sessions it became apparent that five criteria were consistently present in "bad" hours. Any one of these served as an alerting signal, but at least three produced a "bad" hour. Although we do not claim that these are the only valid criteria for bad hours, this working model proved highly reliable for us.

Affect

According to the criterion of affect the patient either displays no affect or, in spite of the therapeutic work, experiences no change in his or her predominant affect over the series of related hours. Another important manifestation is the lack of resonance, reverberations, or empathy in the therapist to the affect displayed by the patient. Aside from the possibility of countertransference, this probably reflects failure of communication because communication is achieved through the evocative power of language. In other words, for the communication to be truly understood one must evoke similar experiences in the receiver. As Peterfreund (2) stated, "Empathy bridges the gap between word and thing representations."

Intellectualization

Too much intellectualization by the patient or by the therapist or both is another criterion for a bad hour. This is somewhat related to the first criterion, but not completely so. For example, some hours showed affective productions by the patient with resonance in the therapist, but the interpretations were formulated in language that was too intellectualized to produce any emotional impact.

Isolation

The criterion of isolation involves a lack of continuity in the productions from the previous hours and, even more importantly, no carry-through into the following ones.

Feedback

Lack of feedback to the therapist's interpretations is another criterion. By feedback we mean something beyond merely accepting or rejecting the interpretation—for example, the emergence of relevant new or supportive material, error-correction, or finer adjustments (explicit or implicit), alteration of the patient's affect, or expansion of his or her awareness. After all, an interpretation is a tentative hypothesis; without the patient's feedback and freedom to correct, refine, revise, accept, or even reject the therapist's interpretation, the analysis or therapy cannot progress successfully.

Dissatisfaction

The final criterion for a bad hour is dissatisfaction, especially on the therapist's part. Dissatisfaction on the patient's part, although valid at times, did not prove to be one of the common denominators.

EVALUATING THE SESSIONS

Having defined these criteria, we devised a shorthand formulation for evaluating our sessions. We imprinted the following formulation on our minds:

1. Affect—none, no change, or no resonance
2. Intellectualization—patient, therapist, or both
3. Isolation
4. No feedback
5. Dissatisfaction

Our purpose in imprinting these concepts (visualized as a box with the criteria listed) was to ensure that the process of evaluation occurred on a preconscious level during the sessions and did not interfere with the free-floating attention of the therapist.

More bad hours and sessions surrounding bad hours were recorded and presented to test this formulation. As a control we analyzed sessions or series of related sessions that we felt to be "good" hours. We did not find at least three of the above criteria in the "good" hours. In the process of doing all this, an interesting phenomenon occurred. Hours that started as potentially bad hours were readily converted into good ones by correcting what was considered faulty. For example, an hour that proceeded for a while with a mounting sense of dissatisfaction by the therapist led to his or her alertness to the possibility of the presence of some of the criteria described above. If the criteria were discernible, the therapist directed his or her interpretations to the specific problems.

For instance, if the therapist sensed the absence of affect or a lack of feedback he or she became aware of the possibility of a countertransference problem that was then identified and dealt with. Occasionally the correction was not possible during the same hour but was achieved in the following session.

CRITERIA FOR "GOOD" HOURS

We judged the hours to be good by the following criteria: 1) active participation by the patient and the emergence of new illuminating material, 2) a sense of cohesiveness and continuity between the sessions, 3) good emotional expression and some qualitative affective change in the patient with reverberations in the therapist, and 4) a sense of working together and satisfaction. We soon learned to use these criteria as tools for the production of good therapeutic work and for

effectively overcoming either resistance in the patient or countertransference problems in the therapist.

DISCUSSION

This system offers definable criteria and is testable for validity. It helps the therapist to become aware of countertransference issues early. It also prevents the therapist from becoming too active and intellectual or remaining totally passive and nonintervening when it is constructive to define the problem and tackle it actively. Activity does not mean talking a lot. Verbal activity is only one aspect of the therapist's mental activity. This mental activity obviously should go on all the time in an effort to understand the patient's verbal and nonverbal communication and the meaning of the unconscious communication, to define problem areas of resistance in the patient or of countertransference in himself or herself, and then to make timely, appropriate interventions. This system also sheds light on subtle resistance. It is certainly a practical model for psychotherapists who see patients once or twice a week in psychodynamic psychotherapy as well as for psychoanalysts. In addition, this work supports the centrality of affective communication to the therapeutic process.

We feel that this report has potential value in two areas: treatment (analytic or psychodynamic psychotherapy) and teaching and supervision of these treatment modalities. We also hope that other therapists will try this simple model in their own sessions with their patients and help validate (or invalidate) our results.[1] Inclusion of the experience of other therapists would certainly prevent the results from being the unique product of our group. With the notable exception of the work done by Horowitz (1) and Peterfreund (3), there is a paucity in the literature of attempts at objectifying the therapeutic process.

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[1]Analysts or therapists who are interested in testing this model by applying it to their own work are encouraged to contact one of us. Comments from clinicians and researchers would be appreciated.

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Chlorimipramine Therapy for Obsessive-Compulsive Neurosis

BY JAMBUR ANANTH, M.D., LESLIE SOLYOM, M.D., SHIRLEY BRYNTWICK, AND UMASHANKAR KRISHNAPPA

Twenty patients with a history of treatment-resistant obsessional neurosis underwent a 4-week clinical trial of chlorimipramine. Scores on the Psychiatric Questionnaire for Obsessive Compulsive Neurosis, the Leyton Obsessional Inventory, and 3 self-assessments indicated that although there was no significant change in obsessive ruminations, obsessive rituals, horrific temptations, or pervading doubt, there was a substantial, significant improvement in the obsessive symptom item and severity of obsessions, as well as anxiety, depression, and phobia. Side effects were mild. The authors believe that chlorimipramine is the most promising treatment for obsessive-compulsive neurosis.

SINCE OBSESSIVE-COMPULSIVE neurosis has proven to be treatment-resistant and there is no single widely accepted and effective mode of treatment, there is a need for research to develop a more adequate treatment mode. Open studies have suggested that a beneficial effect has been produced by tricyclics such as imipramine, amitriptyline, desipramine (1), and doxapine (2). It has been claimed that clorimipramine is effective (3, 4) or indeed may be a specific anti-obsessional treatment (5).

We did a four-week uncontrolled exploratory study of chlorimipramine, the subjects of which were 7 men and 13 women selected from inpatient and outpatient facilities on the basis of a diagnosis of obsessive-compulsive neurosis. The patients were aged 23–64 and had a history of treatment-resistant obsessional neurosis over a two-year period; all patients gave informed consent. Diagnoses were confirmed by a clinical interview, psychometric data from the Psychiatric Questionnaire for Obsessive Compulsive Neurosis (Solyom, unpublished) and the Leyton Obsessional Inventory (6). Pregnant women and patients with psychosis, epilepsy, organic brain syndrome, or acute physical illness were excluded.

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METHOD

After a one-week wash-out period chlorimipramine was administered on a fixed, changing dosage schedule, 100 mg/day during the first week, 150 mg/day during the second week, 225 mg/day during the third week, and 300 mg/day during the fourth week. The dosage was increased weekly unless side effects occurred or marked improvement in psychopathology was apparent.

Assessment procedures included completion of the Psychiatric Questionnaire for Obsessive Compulsive Neurosis and administration of the Leyton Obsessional Inventory before and at the end of one, two, and four weeks of the clinical trial. Self-assessments, which included the Social Adjustment Scale (7), Maudsley Personality Inventory (8), and the Self-Analysis Form (9), were completed by the patients before and after the clinical trial. A brief physical examination (pulse rate, blood pressure, and weight) and laboratory tests (hemoglobin white cell and differential counts, transaminase estimates, alkaline phosphatase, and urinalysis) were carried out before and after the study.

Eighteen of the 20 patients completed the entire study. Two patients discontinued their medication during the third week: a woman who experienced severe drowsiness and difficulty in micturition and a man who became constipated. Dosage had to be altered for 4 more patients because of side effects: during the second and third weeks of treatment 2 patients experienced hypotension manifesting as distressing dizziness, for which the dosage was reduced to 150 and 100 mg/day; during the last week 1 patient developed severe tremors after three days of 300 mg of the medication, necessitating a reduction in dosage to 225 mg; also during the fourth week, dosage was decreased to 100 mg/day for 1 patient because of nausea, dizziness, dry mouth, and sleepiness. In the case of 3 more patients, dosage was not increased beyond 150 mg/day because of improvement.

RESULTS

The mean scores on the Psychiatric Questionnaire for Obsessive Compulsive Neurosis are shown in table

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TABLE 1
Effect of Chlorimipramine on Mean Scores on the Psychiatric Questionnaire for Obsessive Compulsive Neurosis

Scale	Predrug	Week 1	Week 2	Week 4
Symptom	102	90	91	78[a]
Severity	302	239[b]	172[c]	133[c]
Depression	117	106	63[b]	64[a]
Anxiety	160	121[b]	76[b]	67[c]
Phobia	76	61	44	32

[a]$p<.05$, one tailed t test, df=19.
[b]$p<.01$.
[c]$p<.001$.

1. Of the 20 patients, 9 had improved scores on the obsessive symptom item, 10 did not change, and 1 deteriorated. There was no significant change in obsessive ruminations, obsessive rituals, horrific temptations, or pervading doubt. The score for severity of obsessions was improved for 19 patients and was unchanged for 1 patient at the end of the fourth week. Fourteen had improved depression scores, 2 were not changed, and 4 had deteriorated by the end of the study. On the anxiety item, 17 patients had improved, 1 did not change, and 2 deteriorated during the study. Thirteen patients had improved scores on the phobia scale and 7 were unchanged at the end of the fourth week.

The total neuroticism score on the Maudsley Personality Inventory decreased from 628 to 494 ($p<.02$), and the extraversion score increased from 349 to 424 (n.s.). The total raw score on the Self-Analysis Form decreased from 835 to 729 ($p<.01$). On the Social Adjustment Scale there was statistically significant improvement in adjustment at work ($p<.01$), leisure activity ($p<.01$), and social adjustment outside the family ($p<.01$).

The Leyton Obsessive Inventory measures five items: total number of obsessive symptoms, patient's resistance to symptoms, symptom interference with the patient's activities, number of obsessive traits, and the Cornell index, which indicates the degree of neurosis. Only 8 of the 20 patients completed the tests before and after the study. The resistance score decreased from 181 to 124 ($p<.01$, t test, df=7), the symptom score decreased from 180 to 140 ($p<.05$), the interference score decreased from 197 to 107 ($p<.01$), and the Cornell index decreased from 176 to 137 ($p<.05$). The change in trait score was not significant.

A total of 137 side effects—44 gastrointestinal, 27 central nervous system, 21 psychiatric, 20 autonomic, 16 extrapyramidal, and 7 others—were recorded during the course of the clinical trial. However, all of them were mild and some were transitory and needed no contra-active measures. Gastrointestinal, autonomic, central nervous system, and psychiatric side effects were all greatest during the first week but gradually dissipated, whereas the extrapyramidal symptoms, feeling high, increased appetite, and weight gain occurred only during the second half of the study.

CONCLUSIONS

In our study we noted a substantial decrease in the severity as well as the number of obsessive symptoms. Anxiety, depression, and phobia also improved significantly. Therefore, whether the improvement was due to primary antiobsessional effect or secondary to amelioration of anxiety or depression is debatable. However, while obsessional severity improved significantly by the end of the first week, significant improvement of depressive symptoms was noted only at the end of the second week. This may be indicative of an antiobsessive effect of the medication. Psychometric findings also specifically reflect the antiobsessive effect of the drug by a significant decrease in the symptom, interference, and resistance scores. Scores on self-ratings concur with the findings on psychometric and psychopathological assessments. Therefore, chlorimipramine appears to be the most promising medication for the treatment of obsessive-compulsive neurosis. We are currently exploring the antiobsessive potential of chlorimipramine in a double-blind comparative trial.

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Manic-Depressive Variant Syndrome of Childhood: A Preliminary Report

BY RICHARD E. DAVIS, M.D.

The author describes the specific diagnostic entity of manic-depressive variant syndrome of childhood and outlines the five essential diagnostic criteria (affective storms, family history of significant affective dysfunction, hyperactivity, chronically disturbed personal relationships, and absence of psychotic thought disorder) and the five secondary criteria (sleep disturbances, minimal brain dysfunction, abnormal EEG, enuresis, and neuropathology). In the four cases reported, the children, when properly diagnosed, responded well to lithium therapy. The author points out the need for further study of this syndrome. Precautions about the use of lithium in children under age 12 are crucial, as is the need for continuing psychotherapy for parents and child.

ALTHOUGH FEW in number, there are persistent reports in the literature of children and adolescents with major behavioral disturbances who improve greatly when adequate serum levels of lithium are obtained (1–8). Many of these children have been hyperactive and diagnosed as suffering from minimal brain dysfunction or the hyperkinetic syndrome (8). Yet there seems to be a group of factors setting apart a small but significant subgroup of hyperactive children, most notably their positive behavioral response to lithium carbonate, which several studies indicate is not an effective medication for the child accurately diagnosed as suffering from minimal brain dysfunction (9).

In a recent review of the literature on the use of lithium carbonate in children, Youngerman and Canino (1) noted, "In children and adolescents the published literature seems to suggest that aggressivity per se and hyperactivity per se are insufficient indications for the use of lithium carbonate. However, aggressive or hyperactive patients with a major affective component to their behavior may well warrant a trial of lithium carbonate."

In their recent comprehensive book on hyperactive children, Safer and Allen (10) commented, "In the next 10–20 years, as the HA (hyperactivity) becomes more thoroughly explored, a number of other hyperactive syndromes no doubt will be identified."

I propose that there is an identifiable syndrome suffered by a group of persons between approximately 6 and 16 years of age that is characterized by five primary and one or more of several secondary features. I have labeled this entity "manic-depressive variant syndrome of childhood" because its behavioral manifestations do not resemble manic or depressive episodes, but rather a variant of the manic or depressive symptomatology typical of adult life. For this reason the 1960 proposal by Anthony and Scott (11) that one of the essential criteria for diagnosing childhood manic-depressive illness is an "abnormal psychiatric state similar to the classical description" is not appropriate for the group of children identified here, since they do not display classic adult symptoms of mania or depression. The term "juvenile manic-depressive illness" also seems inappropriate for this group for the same reasons (8, 12).

I propose that these young persons can be distinguished from persons suffering from minimal brain dysfunction without such major affective outbursts and from those with childhood or adolescent schizophrenia, autism, and other neurological, medical, and psychiatric illnesses (see table 1) on the basis of five primary criteria (all are essential) and five secondary criteria (one or more are essential).

PRIMARY CRITERIA

Inclusive Criteria

1. *Affective storms.* An affective storm is defined here as a loss of emotional control of major disruptive force in a person under 16 years of age, the precipitating factor being grossly inadequate to explain the degree of emotional outburst. Such disruptive temper outbursts are more intense than the common emotional lability seen in children properly diagnosed as having minimal brain dysfunction. Such storms are transient, irregular, and occur over hours or minutes, not for weeks or months. They may even reach psychotic magnitude, but they are not accompanied by thought process disintegration between such episodes.

The following are typical quotations from the literature indicating the presence of such unusual destructive emotional outbursts in children who are respon-

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up care. He has since been removed from lithium therapy, but is continuing moderate doses of thioridazine. No major outbursts have been reported since lithium was discontinued.

Case 2. B, a 9-year-old boy, had had temper tantrums since age 1. He was disruptive at home, fought with his peers, had violent outbursts of temper over relatively minor frustrations, was having serious behavior problems at school, and had normal thought processes. He was reported to be hyperactive, although he could concentrate well at times. He had had restless sleep for years. The father reported that two uncles were "emotionally ill"; one had shot himself, and both were reported to be alcoholic. The mother had been hospitalized once for depression and felt she had "cyclic" but not incapacitating depression. Previously the family had been in family psychotherapy and the boy in play therapy without major changes.

The boy was hospitalized and after negative findings in examinations for organic illness and renal, thyroid, and nervous system pathology, he was placed on lithium carbonate. Several temper outbursts had occurred during his first 2 weeks of hospitalization, one after a serum lithium level of 1.0 mEq/liter was first obtained. During the subsequent 6 months at home, however, he has had no affective storms, the only such 6-month period since age 1. He is maintained on serum lithium levels of 0.8–1.0 mEq/liter. His parents state that he is "a different boy," and there is a compatible family life for the first time.

Case 3. C, an 11-year-old boy, was seen because of severe recurrent temper tantrums, continuous unhappiness, labile mood, hyperactivity, and loss of emotional control since early childhood. He was a chronic restless sleeper. There was no loss of thought control. There were chronic family and sibling fights, and he had previously been reported to have a learning disability. The father reported a grandfather with a "terrible temper," an alcoholic aunt, and one aunt who had episodes of "being wound up day and night" and going on spending sprees.

Methylphenidate and other medications had been tried previously with minimal results. The boy was evaluated and a visual perceptual learning problem was identified; no organic impairment or illness was identified. He was placed on lithium carbonate and a serum level of 0.8–1.0 mEq/liter was maintained.

The boy's major affective outbursts have decreased markedly in frequency and have not reached the former lifelong levels reported by his parents. After 6 months of lithium treatment his parents noted "a remarkable and persistent improvement" in behavior and management.

Case 4. D, a 12-year-old boy, was evaluated because of unmanageable temper outbursts over relatively minor frustrations and because he was becoming physically abusive to his mother during such episodes. He had no friends, was reported hyperactive at school, and had been placed in a special learning center. His grades were poor, but he was intelligent and without thought process pathology.

The boy was adopted; the biological father was known by the adoption agency and was reported to have a violent temper. No further biological family history was available.

An evaluation for organic illness or impairment was conducted, with negative results. The boy was placed on lithium carbonate. Since maintaining serum levels of 0.8–1.0 mEq/liter, he has had no major outbursts during a 3-month period. He recently received the top student award for his group. His parents reported him "markedly improved."

CONCLUSIONS

In this paper I have described the specific diagnostic entity of manic-depressive variant syndrome of childhood. The term "affective storm" was coined and defined to describe a major symptom of the proposed syndrome. Such a diagnosis can be considered when the five primary criteria are present, as well as one or more of the secondary criteria. In the four cases presented, the children, when properly diagnosed, responded well to adequate serum lithium levels (0.8–1.2 mEq/liter). Although more than one disease entity in children may respond to the use of lithium carbonate, this particular syndrome seems to constitute a specific entity. Severe parent-child conflicts existed in all of the families but were most responsive to psychotherapy after control was established with lithium.

The proposal is a tentative and preliminary one and requires further careful delineation or disproof. Precautions about the use of lithium in children under age 12, warning of possible unknown long-range effects, parental education about lithium dosage and toxicity, and prior approval for its use are crucial, as is continuing counseling or psychotherapy for parents and child.

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The Elective Within the Required Psychiatric Clerkship

BY MARTIN D. PLUTZER, M.D.

Two important goals of a successful psychiatric clerkship are to maximize students' responsibility and flexibility and to provide knowledge and skills relevant to their future careers. The author describes an elective program within the required medical school psychiatric clerkship that meets these criteria. On the basis of feedback from both students and staff, the author concludes that the advantages of the elective program to students, patients, faculty, and the department far outweigh the disadvantages.

THERE APPEARS TO BE a consensus that a successful psychiatric clerkship for medical students should maximize student responsibility and autonomy. Another important goal is to help the students learn skills that are relevant to their future needs as nonpsychiatric physicians. Kasuboski and Marshall (1) demonstrated that students are capable of assuming major clinical responsibility. Similarly, Linet (2) reported favorably on active student involvement on an acute inpatient unit. Medical students have also been successfully used as therapists in outpatient clinics (3).

It is difficult to design a program that provides an optimal experience for all. Each student has differing conscious and unconscious motivations, expectations, and "resistances." Choice of specialty, capacity for empathy, and ability to deal with psychological matters comfortably may vary considerably among students. In addition, the availability of clinical opportunities is such that involvement in one area frequently precludes involvement in another; i.e., a student assigned to an inpatient unit may miss an outpatient experience and vice versa. In an attempt to meet the needs of the individual student, a traditional approach still used is to offer electives, usually following the required clerkship. Another approach, which emphasizes flexibility, has been to use track programs. Papernik and associates (4) described a program that gave students a choice of clinical assignments, which resulted in diminution in anxiety and improvement in motivation, attendance, and grades. Having a "dignified adult choice" appeared to be most important to students. This selection procedure was so successful it was subsequently adopted by all clinical clerkships throughout the medical school.

Sabshin (5) stated, "Periods of elective learning tend to be utilized in a superior fashion by most medical students who feel themselves responsible for their own voluntary choice." However, the traditional elective following the clerkship is often used mainly by students who are interested in becoming psychiatrists. For a variety of reasons, including limited time and motivation, the majority of students remain uninvolved, but it is these students, many of whom will become primary care physicians, who might benefit the most. To correct this inherent limitation in the use of electives, we have designed an innovative program of electives within the required clerkship at the Medical College of Pennsylvania.

THE PROGRAM

A 6-week psychiatric clerkship is required of all junior students at the medical college. The basic program involves students assuming varying amounts of responsibility for patient care with intensive experience on an acute medical college hospital ward, a more intermediate-care state hospital ward, or as part of the liaison-consultation team. All students rotate on call for 24-hour periods covering the emergency room and the general hospital. In addition, there are required lectures and predominantly clinical seminars.

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The elective program gives each group of approximately 16 juniors the choice of selecting one or more electives. Some students participate in two electives and others participate in none. Our current electives include working in the general psychiatry clinic, where, under close supervision, the student evaluates and provides supportive therapy or "crisis treatment" as indicated. A second elective is participation in a mother-child group as part of a parent education project to explain child behavior and development to mothers of young children. A third elective is active involvement with the alcoholism team, which includes evaluation and counseling of patients. A final choice is a small group seminar stressing reading, active participation, and sharing of clinical vignettes on such topics as the difficult and noncompliant medical patient. The total time for each elective, including preparation, varies from approximately 2 to 5 hours per week, and each may accommodate from 2 to 8 students. Selection is made on the first day of the rotation during orientation. The students are told that the electives are to help them pursue their own interests and learning within the clerkship and will require extra time and work.

DISCUSSION

Advantages of the Program

Electives meet the needs of the individual student. Students with varying interests and capabilities can place themselves where they feel most comfortable along an active-passive continuum. Even within the elective options there is a gradient from active involvement with patients in clinics to a more observational role in the mother-child group. There is freedom of choice with a resulting sense of autonomy and dignity. In addition, the program is relevant to the student's future specialty choice. A student interested in family practice may choose to improve his or her supportive psychotherapy skills, while a future pediatrician may be more attracted to the mother-child group. Finally, the student planning a nonpsychiatric career who would not normally take a psychiatry elective has an opportunity to do so.

Electives meet the needs of our patients. Our clinic experience suggests that a bright, interested, empathic, and well-supervised medical student therapist can be supportive and therapeutic. Paradoxically, some patients transferred to a more experienced resident following the student's departure may express disappointment at the more limited time available.

Electives meet the needs of individual faculty members. An elective with one or two students may be an ideal way of including part-time faculty in teaching. This may have the added benefit of tapping teaching talents and interests previously undiscovered and under used. Voluntary staff who are reluctant to provide a "service responsibility" by themselves are usually happy to supervise an interested student and see a patient with the student. The elective may also be a means of rejuvenating a less than optimal seminar. We found that a moderately successful required seminar was transformed into a highly successful one by restricting the size of the group and making it elective.

Finally, electives meet the needs of the psychiatry department. There is more efficient use of clinical material for teaching. Areas not realistic for large numbers of students are quite appropriate for a few. For example, a department faculty member doing consultation work outside the hospital may teach 1 or 2 students. Elective opportunities may be easily and flexibly added to the program as they become available.

Disadvantages of the Program

There may be scheduling conflicts in coordinating time spent on electives with required ward assignments, supervisory hours, and seminars. Our policy has been that clinical involvement has priority.

There may be initial faculty resistance to change. One colleague complained when his student's supervisory hour was in conflict with an elective alcohol seminar: "You've got them running around doing too many things. They'd be better off just sticking to their ward."

There may be student disappointment and rivalry because of the limited number of electives available and the small size of each group.

Student Response

Student response to the program has been overwhelmingly positive both to the idea of having electives within the clerkship and to the elective experience itself. There is often such a demand for electives that equitable selection during orientation is difficult. One student commented that the elective gave her a valuable outpatient experience in contrast to her inpatient ward assignment. Another wrote, "This elective is far and away the most interesting, exciting, dynamic, and informative first-hand experience of the rotation." Another student commented, "A very good opportunity to actively do, or at least attempt to do, some initial treatment of the type we are more likely to do in our careers." Reflecting on the burdens of the increased responsibility in the clinical elective, a student wrote, "The feeling of independence was scary. The feeling I am responsible for this patient is very different from other rotations."

CONCLUSIONS

Satisfying the criteria of increased responsibility, relevance, autonomy, and flexibility, our elective program within the required clerkship has proved popular and successful according to feedback from students and staff. The advantages far outweigh the disadvantages. There is overwhelming interest in pursuing elective options despite the extra time and effort they re-

quire. Approximately 75% of each junior student group takes one or more electives, even though only 5% to 10% express an interest in becoming psychiatrists. This response would seem to contradict the long-held popular notion that medical students consider psychiatry unimportant. It suggests that when given a choice of areas seen as relevant to their future careers, students are interested. Two important keys to success are adequate coordination of the program and sufficient supervision for the students. An overall advantage is that such an elective program provides a mechanism of flexibility for students, individual faculty members, and the department. Depending on interests and availability, the range of elective possibilities is vast.

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The Community Meeting: A Comparative Study

BY RONALD S. RUBIN, M.D.

In this study of two identical open wards, the author found that in ward 1's highly structured meetings a higher proportion of patients participated, and they distributed their participation in more direct proportion to the relative numbers of each present than in the open meetings on ward 2, where patients directed their participation disproportionately to staff. Staff on both wards disproportionately directed their participation to patients, but less so in the structured meetings. The implications of these observations are discussed in terms of the executive ego deficits of the patients involved and their need for structure in the external milieu in order to compensate for these deficits.

SINCE THE EARLY 1950s and the work of Maxwell Jones (1), the concept of the therapeutic milieu has become increasingly entrenched in institutional thinking and practice. One important element in this concept is the ward or community meeting which, from both the literature and personal experience, appears to be one of the very few common elements in all the rather diverse forms that therapeutic communities can take. In some cases the mere holding of community meetings appears to be seen as synonymous with actually having established a therapeutic community. One need not go to such extreme cases, however, to see how central to the therapeutic community concept the community meeting has become. Wilmer (2), for example, not only considers community meetings a requisite for therapeutic communities but also considers their frequency a measure for evaluating these communities. Vitale (3) reported that Maxwell Jones gave similar weight to the importance of community meetings for his own therapeutic communities, and MacDonald (4) felt that community meetings were so common that it was essential to include such meetings in the design of his control or traditional ward.

Despite their popularity, community meetings have not been widely studied. One important reason has been the absence of any simple and economic way of quantifying what occurs in these meetings. There have been some good descriptive studies, but only a few, such as those of Rapport (5), Roberts (6), Fairweather (7, 8), and Kellam and Chassam (9, 10), have provided the kind of data that would make it possible to compare one kind of meeting with another. In an earlier paper (11), Daniels and Rubin described a method of analysis applicable to any large group that used

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simple, objective, replicable measures. In the present study I applied this method of observation to the verbal behavior of both staff and patients in two very different kinds of community meetings.

METHOD

Setting and Subjects

The research was carried out on two identical open wards run by medical school psychiatric faculty and residents in a Veterans Administration hospital. Patients were admitted to either ward by random selection and represented a fairly typical Veterans Administration hospital population (8, 11–14). Most of the patients were in their late 30s, unmarried or divorced, high school graduates, with poor or worsening work histories. There was a wide range of psychiatric disorders; the most common ones were character disorder, alcoholism, and chronic schizophrenia. Patients had spent an average of $2^1/_2$ of the past 10 years in multiple psychiatric hospitalizations.

About one year before this study the staff and patients of ward 1 had established the basic outlines of a comprehensive milieu therapy program that stressed a high degree of patient involvement and responsibility. It was centered on the establishment of a ward-based employment service, a policy committee to suggest general ward aims and codes and specific means of achieving them, and an honors committee to enforce ward behavior codes. These three components were jointly run by staff and patients.

Ward Meetings

On both wards meetings were held in the day room, and all ward staff and patients were expected to attend. Meetings on ward 1 were more frequent; they initially were held 4 times a week but were reduced to 3 times a week halfway through the study because of conflicting time demands of other staff-patient task groups. On ward 2 meetings were held twice a week.

The meetings on ward 1 were quite structured. Attendance and minutes were taken by an elected patient secretary. There was a set agenda with minutes read from the preceding meeting, reports of the various committees, old business, and new business. Both staff and patients had to be recognized by an elected patient chairman before they could speak. The ward meeting was considered to be the focal point of ward life in which all major decisions were proposed, discussed, and voted on, information was shared, functioning of the various task groups was scrutinized, and individual patient (and sometimes staff) behavior was discussed. The meeting also served as an "appeals court" for patients who felt they had been unfairly dealt with by their ward administrator or the honors committee.

On ward 2 the meetings were much less structured. They were chaired by the chief ward physician, and there was no set agenda, minutes read, or formal attendance. The emphasis was on allowing an open setting in which patients and staff members could raise issues they considered of importance to the ward community.

TABLE 1
Characteristics of Meetings on Ward 1 and Ward 2

Group	Mean Number Attending	Percent Participating	Mean Number of Communications/ Meeting	Meeting Length (minutes) Mean	SD
Ward 1				71.7	24.2
Staff	9.2	69[a]	68.0		
Patients	20.0	62[a]	220.9		
Ward 2				31.5	4.6
Staff	8.9	43	43.3		
Patients	23.7	42	140.2		

[a]Significantly more than on ward 2 ($p=.001$, Student's t test, one-tailed).

Procedure

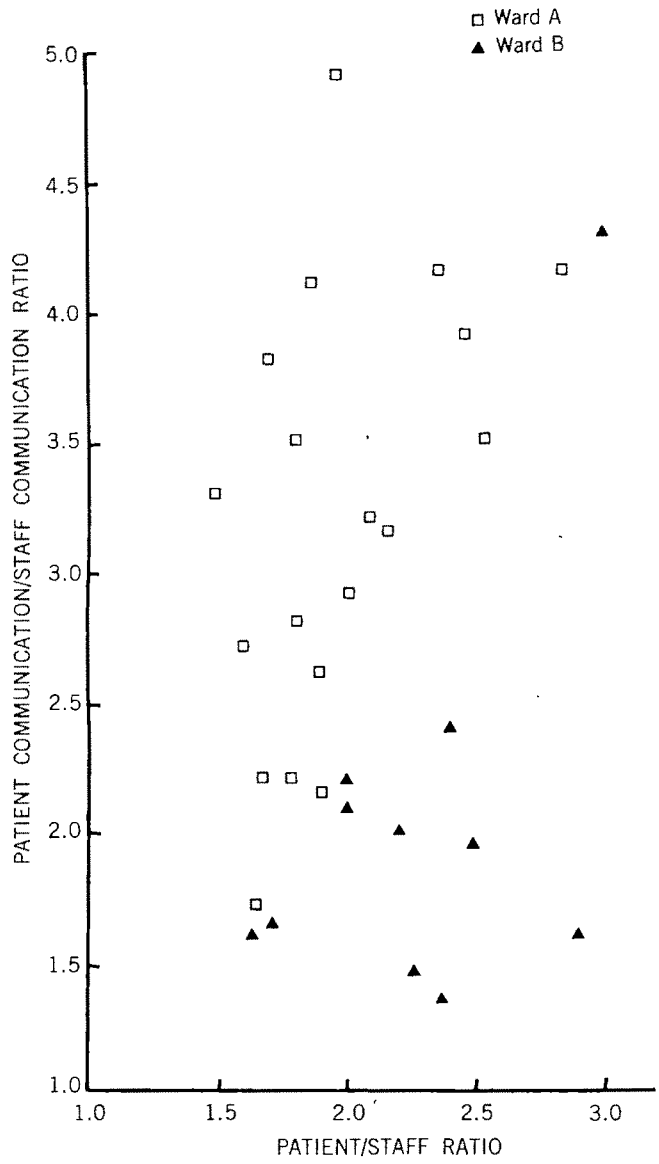
For 8 weeks a rater attended all community meetings on both wards. Each time a community member made a clearly audible verbal communication his identifying code was recorded, as well as the identifying code of the recipient of the communication (a person, several people, or the community as a whole). A new communication was scored each time the speaker changed the target or the topic of his speech.

RESULTS

The data in table 1 indicate that both meetings were roughly the same size in terms of attendance and proportion of staff to patients (about 1:3). Because of the greater number of patients, their communications predominated in both meetings. Both meetings were quite lively. Meetings on ward 1 were open-ended and lasted 30–168 minutes; meetings on ward 2 were scheduled to last 30 minutes but varied somewhat, with a range of 24–40 minutes. While attendance was roughly the same in both meetings, a significantly higher percent of patients and staff participated in ward 1 meetings (that is, made at least one verbal communication). Thus in the community meetings on ward 1 either there was something going on that elicited a far broader response than in the meetings on ward 2 or there were fewer barriers to participation.

Figure 1 presents information for each meeting, in each ward, on the ratio of the number of patients present divided by the number of staff present versus the ratio of the number of patient communications divided by the number of staff communications. This figure illustrates how verbally active the staff on each ward was as a function of how outnumbered they were by patients. In ward 1 meetings, as the patient/staff ratio increased, so did the ratio of patient communications

FIGURE 1
The Ratio of Staff to Patient Communications as a Function of the Proportion of Each Present



to staff communications as a direct positive linear function. However, for ward 2 meetings there is an almost horizontal line. In other words, no matter how outnumbered they were by patients, the staff on ward 1 did not change their verbal behavior, while on ward 2 the more outnumbered the staff were, the more each staff member talked. Meetings on ward 2 seemed to become increasingly disorganized as the patient/staff ratio increased and the staff felt constrained to intervene more in order to maintain some level of order. Because of the highly structured nature of the community meetings on ward 1, there was not the same tendency toward disorganization and patients and staff were therefore more consistent in their verbal behavior.

TABLE 2
Differential Distribution of Staff and Patient Participation

Direction of Communication	Percent of Staff	Percent of Patients
Ward 1		
To the community	33	26
To staff	12	27
To patients	55	47
Ward 2		
To the community	27	20
To staff	8	40
To patients	65	40

Table 2 presents data on the differential distribution of staff and patient participation, i.e., who talked to whom (to the community as a whole, a staff member, or a patient) and in what proportion. For example, if staff spoke to staff without a bias, the percent of total staff communications directed to other staff members compared with the percent of total staff communications directed to patients would be proportional to the relative number of patients and staff present. On ward 1, with an average of 20 patients to 9.2 staff (from table 1), this ratio should be 2.2:1. On ward 2 with an average of 23.7 patients to 8.9 staff, it should be 2.7:1. The actual ratio of staff/patient communications to staff/staff communications was 4.6:1 on ward 1 and 8.1:1 on ward 2. In other words, staff either differentially avoided speaking to each other or preferentially addressed themselves to patients, depending on how one chooses to look at it. This tendency was significantly more pronounced on ward 2 ($p=.005$, one-tailed Wilcoxon's rank sum test).

The importance of this finding is made clearer when one examines the patient participation pattern. As table 2 illustrates, patients on ward 1 distributed their participation to patients and staff in almost direct proportion to the relative numbers of each present (actual ratio=1.7:1). However, on ward 2, the participation focus appears to have been appropriated by the staff, and patients directed only about one-third of the expected proportion of their communications to other patients (actual ratio=1:1).

CONCLUSIONS

The data clearly demonstrate that participation was less role-stereotyped on ward 1. That is, staff and patients were less differentiated, as groups, by their observed patterns of participation. This was due in part to ward 1's ideology, which stressed concepts such as "democratization" and "egalitarianism," but more importantly to the fact that the structure of the setting facilitated the patients' ability to participate in a fuller and less stereotyped way in the shared life of the ward, i.e., more like "responsible members of the community" and less like "sick patients."

What I am suggesting is that it is not their diagnosed illness that prevents patients from participating in these meetings and causes their participation to be role-stereotyped, but rather the deficits in executive ego function that result from the diagnosed disorder and are often the actual reason the patient is being treated in an inpatient rather than an outpatient setting. I am referring to deficits in the ability to delay gratification, set priorities, tolerate frustration, screen out irrelevant inputs, reality test perceptions, moderate responses, and so forth. Such deficits can be chronic (e.g., severe character disorders, mental retardation, chronic schizophrenia) or transient (e.g., neurotic regressive states, manic or depressive episodes, acute schizophrenia). Finally, such ego deficits can be environmentally exacerbated or minimized. Large group meetings are potentially very disorganizing simply because many people are present and much is going on. In such a setting patients with the kinds of deficits in ego function I am describing will tend to either withdraw or become excited, leading staff to intervene in the first case to "bring the patient out" and in the second to "cool the patient off."

A study such as this clearly raises more questions than it answers. For example, is participation "therapeutic"? Can patients participate vicariously and thus get a great deal out of meetings in which they do not appear to be taking an active part? What about the question of quality as opposed to quantity of participation? Pending final answers, I would like to try to put these issues into some perspective. First, I should say that I do not believe that participation is therapeutic in the way that medication, for example, can be therapeutic, nor even in the somewhat less direct way that psychotherapy can be therapeutic. My working hypothesis is that being engaged in and by his or her human environment is likely to increase a patient's sense of self-worth, and, conversely, failure of engagement is likely to lead to a sense of isolation and futility.

As far as the question of vicarious participation is concerned, in the study mentioned earlier Daniels and Rubin (11) found that active participators did not necessarily feel more positive about community meetings than did less verbally active group members, which does seem to support the idea that patients can be both quiet and engaged. From my findings here I would suspect that the pertinent variable is whether patients can follow the proceedings. I hypothesize that given this population of patients and comparing relatively structured with relatively unstructured meetings, one would find not only a higher proportion of those present participating verbally but also vicariously in the more structured meetings. This hypothesis has the virtue of being relatively easily tested.

Dealing with the quality of participation is not quite so simple. When I initially thought of doing this study I had considered rating participants as to whether they facilitated or inhibited further group processes. A few experiments in this direction eventually convinced me that this was more easily thought of than executed. The problems are related to the subjective nature of the ratings that would have to be made in fast-moving meetings. One solution, of course, is to accept the subjectivity as a necessary aspect of the item being measured and settle for reducing the effect of individual bias by using multiple raters. Unfortunately, videotape is not particularly useful here since there are too many potential participators, which means that raters have to be physically present (although the disruptive effects of this could be minimized by having them behind a one-way screen). My hypothesis is that, given the comparison between less and more structured community meetings and given a population of patients deficient in executive ego function, one would note more evidence of personal disorganization, such as communications that were less lucid and less to the point, in the less structured meetings.

From observations over the past few years, it appears to me that the most common error in running community meetings is the attempt to make them into very large small group therapy sessions. I have not found that large group settings are an appropriate place in which to deal with intrapsychic issues. This often happens by default simply because small group therapy is the only approach to groups for which most of us are formally trained. In saying this, I am not suggesting formal training for community meetings but a more thoughtful approach to what can be usefully done with such a setting. For example, I have already mentioned the importance that I place on the patient's sense of engagement in his or her environment. It seems to me that the community meeting, like the old town meeting that it so much resembles, is an excellent place to establish such a sense of engagement, which I feel most reliably develops when patients have a real say in important decisions affecting their day-to-day lives on the ward, a setting in which they can exercise that say together as a community, and sufficient structure in that setting so that they can participate at their best to make the experience a positive one rather than yet another defeat.

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CLINICAL AND RESEARCH REPORTS

This section contains 1) new research findings, including preliminary data from pilot studies, either clinical or laboratory; 2) worthwhile replication studies; 3) case reports that describe a truly new syndrome or cast new light on established ones; and 4) case reports that indicate a new therapeutic procedure of potential value or call attention to adverse effects of drugs or previously unreported complications of therapeutic interventions. Program descriptions and literature reviews cannot be printed in this section. Criteria for format are listed in "Information for Contributors" in each issue; papers that do not adhere to these criteria will be returned to the author.

An Idiot Savant with Unusual Mechanical Ability

BY EDWARD HOFFMAN, PH.D., AND RUSSELL REEVES, SP.A.

The idiot savant phenomenon has continued to be an elusive subject in the fields of psychiatry and mental retardation. In recent years, there have been reports of isolated cases of otherwise typically mentally retarded or autistic persons with unusual abilities in calendar calculation (1, 2) or artistic expression (3, 4). Although there have been various explanations advanced for this condition, most viewpoints are now converging on the hypothesis proposed by Hoffman (2), i.e., that intense motivation, practice, and appropriate reinforcement seem to be necessary if not sufficient conditions for idiot savantism. Recent studies have supported Hoffman's contention that the retarded or autistic individual must have some minimum cognitive functioning, perhaps in the moderate or above range of intelligence, to exhibit the characteristics of the idiot savant.

The purpose of this article is to report a case in which unusual mechanical ability was shown by a moderately to mildly retarded institutionalized man.

Case Report

Mr. A was the oldest of several children born to a rural couple in the 1930s. According to the attending physician's report, the patient weighed 3.4 kg at birth, and the delivery was normal and spontaneous. However, there was no prenatal care and the birth occurred in a rural home that lacked sanitation. The child was bottle-fed, weaned at 18 months, toilet-trained at 30 months, and began to dress himself at 40 months. His home life was described as "meager" by the intake social worker.

Mr. A entered public school at age 5 and left at age 15. He did not learn to read or write and was unable to write his name without a model. He did not speak until age 10. His favorite activity in school was working with machinery. Mr. A's mother died when he was an adolescent. When he was 18, he moved in with an uncle and shortly thereafter contracted a virulent infection that permanently damaged his hearing in both ears.

At that time, he was placed in a state institution by his father, who said he was unable to care adequately for his son. Within weeks after the admission, the father died and a younger sibling became Mr. A's legal guardian. When he was admitted to the institution, Mr. A was found to have a performance IQ of 40 on the Arthur Point Scale of Performance (Form 2), indicating a mental age of 6 years, 5 months. He was diagnosed as having a "cerebral defect, congenital," with no further diagnostic details given.

Mr. A has been institutionalized continuously since the early 1950s. His family visits him less than once a year and remains opposed to his placement in a less restrictive setting. He currently attends a sheltered workshop daily and earns a modest sum for his work. He initiates only minimal social interaction there and at the institution. He wears a hearing aid, but audiometric diagnostic techniques have not fully determined how much of his hearing problem is physical and how much is behavioral. He has not learned to sign, despite repeated attempts over the years.

Current assessment data indicate that Mr. A functions in the mild to moderate range of mental retardation. Testing in 1977 revealed a deaf learning age of 9 years, 4 months, and a corresponding learning quotient of 62 on the Hiskey-Nebraska Test of Learning Aptitude, and a social age of 10.5 years and social quotient of 42 on the Vineland Test of Social Maturity. Periodic intelligence tests over the last 25 years have typically placed his intellectual functioning in the IQ range of 55–65, with far better performance than verbal scores. He can meet all of his self-care needs, tell time, and make change; however, he cannot read, nor can he write anything but his name.

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Since his admission to residential institutions, Mr. A has received much attention for his unusual mechanical abilities. Previous records note that he typically repaired clocks, electric hot plates, and bicycles of other residents or staff. He was able to disassemble and clean the cottage dishwasher. He ran the film projector and built lamps, requiring only that the appropriate materials and tools be made available to him.

Because of his extreme slowness and deliberateness, standardized tests for mechanical aptitude have proven unreliable and of questionable validity in assessing his abilities. Therefore, a naturalistic experimental assessment was done in 1978 to measure Mr. A's mechanical abilities. In one task, he was given a broken electric alarm clock to repair. Within an hour, he correctly traced the problem to a break in the wiring, at which point he shrugged and handed the clock back to the examiners. In the other task, he was given a 10-speed bicycle that was broken in several places to diagnose and, if possible, repair. Over two consecutive evenings, Mr. A successfully diagnosed all of the problems and indicated by gesture which tools he would need to repair the bicycle.

Mr. A has his own workbench and a set of power tools, and he engages in a variety of mechanical projects in the cottage. He repairs bicycles for other residents, constructs wood-paneled and mirrored coatracks for sale, and spends much time adding mirrors, lighting fixtures, and extra electrical connections to his own stereo equipment, lamps, and bicycle. His most recent project has been to connect the wiring of his stereo set, headphones, and room lamp with one switch beside his bed.

Mr. A tends to be a social isolate in the cottage and devotes almost all of his free time to his mechanical pursuits. He receives much praise and attention from other residents and staff members for his abilities and is certainly aware of his talents. Aside from his mechanical abilities, his behavior is quite typical for institutionalized individuals of his mental capacity.

Discussion

The available information suggests that Mr. A's condition fits the general paradigm advanced by several investigators of the idiot savant phenomenon. First, his intellectual performance lies within the proposed minimum range for idiot savantism (2). It appears increasingly unlikely in view of other case reports (1, 3, 4) that the condition can occur much below this intellectual range, and to date no such cases have been documented to our knowledge.

Second, Mr. A's case history also suggests that his mechanical abilities were strengthened by a great amount of motivation and practice, which began in his school-age years. In his current residential setting, staff members have shown him how to use various power tools, and he has achieved independence with these tools only after careful supervision. As mentioned earlier, he typically spends several hours each evening on his projects, working slowly and methodically. He prefers this self-initiated activity to group entertainment or outings.

Observations in the cottage have also indicated that he receives extensive social reinforcement from staff members, as well as other residents, for his mechanical pursuits. For instance, staff members proudly show even the most casual visitor to the cottage Mr. A's tools, materials, and completed electrical and mechanical projects, which fill his room and part of a nearby lounge area. From his satisfied smiles and low chuckles as he works, it seems clear that he also receives strong self-reinforcement for these mechanical pursuits.

While we are not arguing that the idiot savant condition is due entirely to the influence of external or environmental factors, this case supports the emerging paradigm for the phenomenon. That is, as Hoffman (2) and Morishima and Brown (3, 4) have argued, the necessary if not sufficient conditions associated with idiot savantism appear to be a minimal cognitive level of functioning, intense practice and motivation aided by a funneling of external stimuli, and strong reinforcement to develop and maintain the unusual ability. The other prerequisite seems to involve an idiosyncratic pattern of intellectual performance, evidenced in this case by Mr. A's aptitude and preference for mechanical tasks.

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The "Psychotic" Curse

BY ROBERT J. PARY, M.D.

Until recently profane speech from a patient on prolonged neuroleptics might have evoked little added concern. Usually the profanity was assumed to be related to the patient's worsening psychosis. A recent case report by Klawans (1), however, may necessitate clinicians modifying their approach to profanity in a person undergoing chronic neuroleptic treatment. Klawans reported the case of a 28-year-old woman who developed Gilles de la Tourette's syndrome after 6 years of chlorpromazine therapy.

Tourette's syndrome consists of facial and/or body tics associated with either inarticulations or coprolalia (2). Following is a case report of a woman who developed Tourette's syndrome after prolonged neuroleptic therapy.

Case Report

The patient was a 19-year-old woman whom I first examined in 1978, on her seventh psychiatric admission. She had religious delusions, auditory hallucinations, and an extremely flat affect. Physical examination revealed paroxysmal eye blinking associated with either episodic cursing or inarticulations. Grimacing facial features often accompanied the episodic outbursts. There were no signs of oral-buccal or masticatory tardive dyskinesia, nor were there involuntary movements of the extremities. The remainder of the examination was unremarkable.

A chart review revealed that the patient was first admitted in 1972 and had since then been treated with increasingly higher doses of neuroleptics. Beginning in 1977 she was treated with multiple daily neuroleptics. The dosages were equivalent to more than 2000 mg/day of chlorpromazine for 6 months, and for several weeks the dosage was over 4000 mg equivalents of chlorpromazine (3). During 2 of the 6 months of moderately steep neuroleptic dosages, 23 nursing shift notes recorded that the patient had been hissing. A chart review failed to document any serious medical or neurologic illness, nor did the review indicate any observations of facial or body tics before July 1978.

The patient was one of 11 children. Both of her parents and all of her siblings were living, and none showed any evidence of neurological or medical illness. The patient's mother and 1 brother had been hospitalized in the past for auditory hallucinations and persecutory delusions.

The patient was treated with haloperidol in increasing amounts to 100 mg/day. Initially there was little change in her delusions and hallucinations; however, her profanity diminished, although she still exhibited inarticulations associated with paroxysmal eye blinking. The haloperidol was gradually reduced to 20 mg/day due to symptoms of lethargy. EEG, ceruloplasmin, and cerebrospinal fluid, including titers for encephalitic antigens, were normal. On 20 mg/day of haloperidol at discharge, she still manifested minimal eye blinking associated with inarticulations. Her delusions were much less pervasive than when she was first admitted, and her hallucinatory material was greatly reduced.

Discussion

When the patient was first seen in July 1978 the cursing and facial tics received little extra attention. It was assumed that the tics were idiosyncratic mannerisms of her schizophrenic process. Ascher (4) remarked on the similarity of certain signs of Gilles de la Tourette's syndrome to those observed in schizophrenia; his statements took on new meaning as the frequency of the patient's tics and profanity decreased with increasing haloperidol, even though her delusional thinking and hallucinatory material remained unchanged. The response seemed quite similar to the response of early tardive dyskinesia to neuroleptics. In fact, a classification of complex dyskinesias by Crane (5) encompasses both paroxysmal eye blinking and respiratory dyskinesias, which may cause peculiar vocalizations or grunts.

It is possible that tardive dyskinesia could be manifested by a complex dyskinesia initially without signs of the more common oral-buccal or extremity dyskinesia. Stevens (6) presented a study showing that paroxysmal eye blinking may be the initial sign of tardive dyskinesia in the absence of oral-buccal or other dyskinesia.

A neuroleptic-induced Tourette's syndrome is compatible with the present knowledge about dopamine. Snyder (7) postulated that Tourette's syndrome is related to a dopamine receptor hypersensitivity. Tardive dyskinesia is also thought to be related to a dopamine receptor hypersensitivity after chronic, neuroleptic-induced dopamine receptor blockade (8). Klawans (1) discussed the possibility that rebound neuroleptic-induced dopamine hypersensitivity may be manifested as Gilles de la Tourette's syndrome.

To include Gilles de la Tourette's syndrome among the possible presentations of tardive dyskinesia may have pertinent clinical applications. Profanity or inarticulations in patients receiving chronic neuroleptic treatment will necessitate greater scrutiny. No longer can these signs be rubberstamped as part of the patient's psychosis unless the clinician also observes for muscular tics and considers the possibility that the profanity was induced by neuroleptics.

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Anxiety and Serum Prolactin

BY ROY J. MATHEW, M.D., BENG T. HO, PH.D., PATRICIA KRALIK, M.S., AND JAMES L. CLAGHORN, M.D.

The recent development of reliable radioimmunoassay methods for measuring serum prolactin has led to studies that indicate this hormone may respond rapidly and markedly to psychological influences in both man and animals. It is well established that stress can elevate prolactin in animals (1, 2) and a similar relationship has also been demonstrated in humans (3). However, the precise relationship between prolactin release and stress-related psychological areas such as anxiety is unclear (4).

The aim of the present study was to investigate the effect of anxiety and relaxation on serum prolactin in a group of subjects with chronic anxiety. A nonpharmacologic form of treatment was chosen to eliminate the confounding influence of psychotropic agents (5). Because hormone levels are influenced by several factors (sex, exercise, etc.), the patients were used as their own controls (5).

Method

The subjects were 11 outpatients (8 women and 3 men) aged 26 to 55 years who had generalized anxiety disorder according to Research Diagnostic Criteria (6). Patients with a history of alcoholism or drug abuse were excluded. All subjects, including those who were taking psychotropic medication at the time of screening, were required to undergo a minimum washout period of 1 week. A thorough physical examination and routine laboratory tests, including assays for stress-related hormones, T3, T4, and T7, were done to rule out concomitant physical illnesses. The patients were given firm instructions to avoid all forms of medication during their participation in the study. Pertinent background data were recorded during the second visit. Subjects were then requested to lie down and relax alone for 30 minutes in a quiet, semi-dark room. The first blood sample for prolactin assay was drawn immediately after the relaxation session. Blood was collected between 11 a.m. and 12 noon, when the endogenous prolactin levels were shown to be at their minimum. Subjects then filled out the State-Trait Anxiety Inventory (STAI).

The State-Trait Anxiety Inventory is composed of separate self-report scales for measuring two distinct types of anxiety. State anxiety is conceptualized as a transitory emotional state or condition characterized by 1) subjective, consciously perceived feelings of tension and apprehension, and 2) heightened autonomic nervous system activity. Trait anxiety refers to individual differences in tendencies to respond to situations perceived as threatening with increased state anxiety (7).

During the next 3–4 weeks the subjects received nine 30–40 minute relaxation training sessions. Four subjects received Jacobson's progressive relaxation training (8) and were given biofeedback-assisted relaxation training. The subjects were instructed to practice the relaxation exercises at home for 20 minutes a day. After the training was completed the subjects returned to the laboratory for final evaluation. The STAI was administered and a second blood sample drawn after the subjects relaxed in a quiet, semi-dark room for 30 minutes.

Serum was separated from 6–8 ml of venous blood within 2–3 hours after collection. Prolactin was determined by radioimmunoassay using a commercial reagent kit modified for liquid scintillation spectrometry and for increased sensitivity (method of Kralik and Ho, unpublished).

Results

The pre- and posttreatment STAI scores and prolactin levels were compared using paired t tests. There were significant reductions in both state anxiety ($t=3.16$, $p<.01$) and trait anxiety ($t=3.56$; $p<.005$).

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The change in serum prolactin level following treatment was insignificant and inconsistent (13.74 ng/ml±6.93 versus 13.12±7.40). Pearson product moment correlations showed no significant pre- or post-treatment relationships between serum prolactin and either state or trait anxiety.

Discussion

Although there were significant reductions in anxiety following relaxation therapy, no consistent correlations could be found between the prolactin and anxiety levels before and after treatment.

Stress has been shown to increase prolactin secretion (3). However, stress does not always cause anxiety and has been shown to induce more than one response (9). Our inability to find a relationship between serum prolactin and chronic anxiety and anxiety relief does not necessarily argue against a link between stress and this hormone.

It is important to note that the lack of a prolactin-anxiety relationship applies only in chronic anxiety. Since it has been established that endocrinologic changes seen in association with acute stress are different than those seen with chronic stress (10), it is likely that the endocrinology of acute anxiety is different from that of chronic anxiety.

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Haloperidol, Hyperthyroidism, and Sudden Death

BY MYRON F. WEINER, M.D.

Death due to aspiration asphyxia in patients treated with phenothiazines has been reported many times. The mechanisms postulated are regurgitation due to loss of control of esophageal sphincters and impairment of swallowing (1, 2). Moore and Book (3) cited a report of absent gag reflex in 40.3% of psychiatric inpatients treated with phenothiazines compared with 9% of controls. Solomon (4) reported on a patient treated with fluphenazine who had absent gag reflex and a bulbar palsy-like syndrome; he postulated that aspiration might result from the patient's inability to close the glottis or to create sufficient intrathoracic pressure to cough (4).

This case is to our knowledge the first report of death due to aspiration asphyxia in a patient treated with haloperidol, a nonphenothiazine antipsychotic drug.

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Case Report

Ms. A, a 43-year-old woman, was admitted to a psychiatric inpatient service because of bizarre behavior noted by her husband and family. She had become preoccupied with photographs of her dead son, talked as if he were alive, slept little, and was agitated and hostile. She was brought to the hospital when she expressed the feeling that people were trying to kill her.

On admission, Ms. A was confused, agitated, and talked almost continuously. She was oriented to place and person but thought the year was 1955. Her recall and recent memory were intact. She expressed ideas of reference and of extern control.

Her medical history (obtained after admission) incl treatment for pulmonary tuberculosis in 1965, hyster pingectomy in 1969, and heavy alcohol intake up years before admission. Ms. A had lost 22.7 kg in years before admission and had noted increased hai dry skin for about 4 years.

On admission, Ms. A's blood pressure was 12 piration was normal, and her pulse was 144, w lar beat heard. She had pharyngeal exudatio and questionable muscle weakness and wa

Ms. A was placed on haloperidol, 5–25

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tropine mesylate, 2 mg/day. By the 3rd hospital day she was still agitated but expressed no delusions, and her pressure of speech had diminished. On the 7th day she complained of sleepiness and difficulty breathing due to nasal stuffiness. Examination revealed a reddened pharynx with whitish exudate. Her respiratory rate was 25 and her apical pulse was 180 and slightly irregular.

On the 8th day a throat culture was reported to be negative. Ms. A complained of difficulty breathing due to nasal stuffiness but was "unwilling" to open her mouth to breathe. She had mild paucity of movement and was agitated and diaphoretic. Her blood pressure was 142/92, respirations were 28, and pulse 196.

On the 9th hospital day thyroid studies revealed T_3 uptake greater than 75% and T_4 of 24.9 μg/100 ml. An ECG showed atrial fibrillation. The thyroid was not markedly enlarged to palpation. She was transferred to the internal medicine service and was started on digoxin, 0.25 mg/day, propanolol, 160 mg/day, and phenobarbital, 60 mg b.i.d. Haloperidol and benztropine mesylate were discontinued.

On the 13th hospital day Ms. A refused to undergo a thyroid scan, stating that the orderly had come to take her to the morgue. Later in the day, after a nurse convinced her to have the scan, she fled from the nuclear medicine department after asking if the people dressed in street clothes (outpatients) had come to pick up deceased relatives. She was restarted on haloperidol, 10–15 mg/day, in addition to her other medications. She underwent a thyroid scan on the 15th day that showed 50% uptake and gland size 2–3 times normal. Thyroid antibodies were reported negative on the same day.

On the 16th hospital day Ms. A received a therapeutic dose of ^{131}I. At that time, she complained of increased salivation. On the 17th day nursing staff noted that she was constantly salivating and tremulous. She was anxious and demanding of attention on the 19th day. Her oropharynx was suctioned twice in the morning. She complained of difficulty in swallowing fluids and solid foods. At 4 p.m., her blood pressure was 190/100, pulse 130 and irregular, temperature 99°F, and she was restless. At 4:10 p.m., she had some ice cream and some orange juice; a little later she drank half an ounce of milk. Shortly after 7 p.m., Ms. A was found without respiration, with fixed pupils, and no detectable pulse. Creamy liquid was suctioned from the endotracheal tube that was inserted. Cardiopulmonary resuscitation was unsuccessful and the patient was pronounced dead at 8:28 p.m. An autopsy was not performed.

Discussion

Several reports in the literature indicate that hyperthyroidism enhances the neurotoxic effects of haloperidol.

Lake and Fann (5) reported a case in which there was a clear relationship of the hyperthyroid state to extrapyramidal side effects. Their patient developed tremor, muscle fasciculations, and pronounced rigidity. When he was taken off haloperidol and treated to a euthyroid state with propylthiouracil (PTU), his rigidity abated. After PTU was discontinued, he was restarted on haloperidol, 8 mg/day, for his psychotic behavior, and did well for 15 days. Two days after treatment with ^{131}I his hyperthyroid state returned, despite resumption of 600–800 mg/day of PTU, and was accompanied by a recrudescence of parkinson-like rigidity. Haloperidol was discontinued 5 days after radiotherapy and propanolol was started to reduce tachycardia. After clinical and laboratory evaluation indicated he was euthyroid, the patient was able to tolerate haloperidol, 8 mg/day, for another 45 days in the hospital without unusual reaction.

Yosselson and Kaplan (6) reported on a patient who had uncontrolled hyperthyroidism and had taken haloperidol, 4 mg/day, and who then developed severe weakness, inability to speak or walk, facial rigidity, and dryness of the lips and tongue.

Hamadah and Teggin (7) described a 74-year-old woman with unsuspected hyperthyroidism who developed boardlike rigidity, dyspnea, cyanosis, and inability to swallow fluids after taking haloperidol, 6 mg/day for 20 days.

Flaherty and Lahmeyer (8) suggested that a laryngeal-pharyngeal dystonia was the cause of respiratory distress seen in two presumably euthyroid patients treated with haloperidol. An absent gag reflex was noted in one patient. Both patients responded well to intravenous diphenhydramine and temporary maintenance on 2 mg/day of benztropine mesylate.

The patient we have described developed a bulbar palsy-like syndrome with difficulty swallowing, probably in connection with an exacerbation of her hyperthyroid state after a therapeutic dose of ^{131}I, as occurred with the patient described by Lake and Fann.

The increased risk of dystonic reactions suggests that haloperidol should be routinely accompanied by antiparkinsonian agents or that less potent dopamine blockers should be employed in treating psychotic hyperthyroid patients. The development of a severe extrapyramidal reaction to haloperidol may also suggest undiagnosed hyperthyroidism.

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Potentiation of Lithium by Tryptophan in a Patient with Bipolar Illness

BY GUY CHOUINARD, M.D., M.SC.(PHARMACOL.), BARRY D. JONES, M.D., SIMON N. YOUNG, PH.D., AND LAWRENCE ANNABLE, DIP.STAT.

The results of our recent double-blind controlled study of the action of tryptophan and imipramine in the treatment of newly admitted depressed patients led us to hypothesize that unipolar and bipolar patients differ in their response to tryptophan (1, 2). Bipolar patients and patients with acute mania seem to require higher doses of tryptophan than do unipolar patients. Two studies of acute manic patients showed moderate (3) or no (4) therapeutic effect with 6 g of tryptophan a day, but a third study achieved better results using an average dose of 9.6 g/day (5). In an unpublished pilot study we have obtained encouraging results from the use of 12 g/day of tryptophan in manic patients. If high doses of tryptophan are therapeutic in both manic and bipolar depressed patients, this drug might be useful in manic-depressive illness, especially for patients who do not respond satisfactorily to lithium. This paper is, to our knowledge, the first report of potentiation of lithium by tryptophan in a patient who responded poorly to lithium.

Case Report

Ms. A, a 46-year-old married woman, had no psychiatric history and was working successfully in an executive position when she experienced her first depressive episode at age 42. The episode was characterized by lack of energy, suicidal ideation, insomnia, weight loss, guilt feelings, and feelings of helplessness. These symptoms responded to amitriptyline, 50 mg p.o. b.i.d., but Ms. A stopped the medication because she became overactive and hostile, exhibited pressure of speech, and began spending inappropriate sums of money. Approximately 1 month after she stopped taking the antidepressant, she became extremely depressed and was admitted to the hospital with suicidal ideation. In the hospital she responded well to tricyclic antidepressants and was discharged, but 6 weeks later her hypomanic symptoms reappeared. Although the medication was continued, her depressive symptoms returned within 1 month and she was readmitted after she attempted suicide by overdose. She was given antidepressants, recovered, and was discharged.

At this point, Ms. A started a cyclic pattern of mood changes that usually consisted of a 2-week period of mania or hypomania, 2–3 days of normal mood, and then 2 weeks of depression (the depressed phase lasted up to 2 months at times). This cycle did not correspond to her menstrual cycle. During the depressed phase Ms. A was unable to do anything beyond the most basic necessities of caring for herself, and she spent most of her time in bed. It was necessary to discontinue antidepressants because they precipitated more severe manic periods and did not prevent the depressed phase.

At the age of 44, Ms. A was readmitted to the hospital in an attempt to stabilize her on lithium. Lithium carbonate, 300 mg p.o. q.i.d., lessened the severity of the manic phase, but the time course of the cycle remained unchanged. Approximately 3 months after she was discharged, perphenazine, 4 mg/day, was added to lessen the subjective anxiety that she experienced during both depressed and manic phases. Throughout the next year and a half, Ms. A continued to experience the cycle of depressive and manic periods and was unable to work despite serum lithium levels of approximately 1 mEq/liter. She lived with friends who cared for her during the recurrent depressed phases and prevented her from acting on her severe suicidal ideation.

Ms. A was referred to our pharmacology research unit for consultation concerning possible change in the treatment regimen, which consisted then of lithium carbonate, 300 mg p.o. q.i.d.; perphenazine, 4 mg/day p.o.; and flurazepam, 30 mg q.h.s. as needed for insomnia. Her medical history included a male first cousin on her father's side who had a history of bipolar affective illness. Physical examination and routine laboratory analysis were normal. There was no history of drug abuse or alcoholism.

It was decided to add L-tryptophan, 3 g p.o. b.i.d., and nicotinamide, 750 mg p.o. b.i.d., to her treatment regimen. Informed written consent was obtained from the patient. Nicotinamide was given with tryptophan in order to decrease the peripheral catabolism of tryptophan by tryptophan pyrrolase (6). She was rated on the Hamilton Rating Scale for Depression, the Beck Self-Rating Inventory for Depression, and a 5-item mania scale before treatment and at weekly intervals thereafter. At the same time intervals, blood was drawn for the measurement of plasma tryptophan and serum lithium. An ultrafiltrate of plasma, prepared as described previously (2), was used for the measurement of free (non-albumin-bound) plasma tryptophan. Tryptophan was measured by the method of Denckla and Dewey (7). On day 30 of the trial, the dosage was increased to 3 g q.i.d. of L-tryptophan and 1 g q.i.d. of nicotinamide. She tolerated this high dose very well. During the 8th week she discontinued flurazepam because her sleep had improved and at week 10 a marked reduction in anxiety enabled her to stop the perphenazine. She continued throughout the trial on lithium carbonate, 300 mg p.o. q.i.d.

Table 1 shows the results of all evaluations. It can be seen that during the study period Ms. A exhibited 2 complete cycles, the first ending on week 3 and the second ending on week 8. The depressed phase of cycle 2 was reduced in intensity on both the Beck and Hamilton scales as compared with the first cycle. The third cycle started on week 9 with a manic phase of slightly reduced intensity, but on week 10, when the patient's plasma tryptophan levels were higher than any level reached previously, she was considered by

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TABLE 1
Weekly Measurements of Psychopathology, Plasma Tryptophan, and Serum Lithium

Week	Hamilton Depression Scale[a]	Beck Inventory	Mania Scale[a]	Plasma Tryptophan (mg/ml) Total	Plasma Tryptophan (mg/ml) Free	Serum Lithium (mEq/liter)
0	5	4	8	9.4	2.3	1.17
1	3	3	2	12.9	3.2	0.86
2	39	41	0	32.7	10.4	1.00
3	37	42	0	67.6	21.8	0.89
4	4	5	8	22.9	9.0	0.91
5	3	3	4	98.4	24.4	1.17
6	3	3	4	19.8	4.1	0.87
7	30	33	0	16.4	3.8	0.95
8	22	36	0	76.7	23.5	1.00
9	2	3	6	31.0	7.8	0.96
10	2	3	0	110.8	51.6	0.87
11	2	3	0	104.2	36.0	0.91
12	2	3	0	52.7	10.4	0.96
13	4	3	0	142.0	63.2	0.39
14	2	3	0	137.0	47.6	0.87

[a]Total scores.

both herself and two psychiatrists to be normal. At the time of writing she has remained normal for sixteen weeks. The only item on both the Beck and Hamilton scales that remains elevated is sexual dysfunction, consisting of a loss of libido. The patient has not yet resumed her normal sex life.

Discussion

Although lithium is usually an efficacious treatment for bipolar affective illness, there remains a small percentage of patients who do not respond satisfactorily. Incomplete responders to lithium therapy are often given additional medications such as neuroleptics or tricyclic antidepressants, but such drug combinations have met with little success. In the case described here, the patient had been treated with tricyclics, lithium alone, and subsequently with lithium in combination with a neuroleptic. These treatment regimens had not adequately controlled her manic or depressive symptoms or altered the cycle's length. However, the addition of tryptophan-nicotinamide to lithium resulted in an almost complete remission of symptoms. The first effect was seen 7 weeks after addition of tryptophan-nicotinamide, with a reduction in the severity of the depressed phase. After 10 weeks the patient entered her first extended period of normality since the illness began 4 years ago.

Lithium reduced the severity of both manic and depressed episodes, but the cycle itself continued. The addition of tryptophan-nicotinamide resulted in a further reduction in the severity of symptoms and a return to normality. However, we have clinical and biochemical evidence from studies in progress which indicates that the cycle continues but does not induce symptoms in the patient. For example, Ms. A reports greater difficulty in sleeping at the time of the expected switchover. This suggests that tryptophan acts in a similar way to lithium and raises the question of whether tryptophan potentiates the action of lithium or has a therapeutic effect of its own. Whatever tryptophan's mode of action, this case is consistent with our hypothesis (1, 2) that only high doses of tryptophan are of therapeutic value in bipolar illness, whereas lower doses are required in unipolar illness. The fact that tryptophan may have some antimanic action in acute mania when given at high doses (unpublished data) also supports the idea that tryptophan when given in combination with lithium may be an effective and nontoxic prophylactic agent for use in manic-depressive illness.

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Binge Eating Associated with Oral Contraceptives

BY RICHARD A. MOSKOVITZ, M.D., AND AURORA LINGAO, M.D.

Binge eating has been described recently (1–4) as a distinct clinical syndrome with possible neurophysiologic correlates. It is characterized by sudden, uncontrollable episodes of excessive eating, usually ending in physical distress. It may be followed by self-induced vomiting and usually leads to feelings of remorse or self-contempt. Although similar behavior occurs in some patients with anorexia nervosa, the relationship of the binge-eating syndrome to anorexia nervosa is unclear (5).

The following case, a typical example of this syndrome, was marked by a dramatic remission of symptoms following discontinuation of an oral contraceptive. It is to our knowledge the first reported case correlating clinical change in symptoms with changes in the EEG.

Case Report

A 25-year-old single woman was referred for compulsive eating of 3 years' duration. Since age 21 she had experienced abrupt episodic eating binges characterized by a driven, ego-alien feeling. She could identify no precipitating circumstances or emotions. The episodes lasted from 15 minutes to several hours and occurred in clusters over 2 to 5 days. The clusters were spaced from several days to several weeks apart, alternating with periods of strict dieting. She was 152.4 cm tall and maintained her weight at 47.7–48.6 kg despite acute fluctuations of 1.8–2.3 kg during binges.

The binges generally occurred in solitude and secrecy. They tended to occur in the evening but could occur at any time of day. The patient would rapidly consume large amounts of food, mostly sweets and starches but also leftovers and other ready-to-eat fare. Each episode would end with marked abdominal distress or sometimes sleep but no vomiting. After each binge, she felt helpless, disgusted, and guilty. Depression accompanied each cluster of binge eating, with insomnia, feelings of defectiveness, and fear of losing control. The frequency and intensity of the binges fluctuated. She could recall no binge-free interval longer than 1 month since the onset of the episodes.

Aside from infrequent headaches, which were rarely severe, she had no other major psychiatric or neurologic symptoms. The patient appeared to function normally in other respects. The only remarkable aspect of her medical history was the use of oral contraceptives continually since age 19. She had taken one combination pill for about 2 years and changed to another, which contained 1 mg of norethindrone and .05 mg of mestranol, when she was 21.

Neurologic examination was unremarkable. Thyroid studies and a glucose tolerance test done before referral were normal. Her initial EEG was remarkable for bifrontal, rhythmic slow and sharp activity. A follow-up EEG 1 month later was similarly abnormal. A CT scan showed left-sided punctate basal ganglia calcification of undetermined significance.

After 8 months of further evaluation and psychotherapy, the patient accepted our recommendation to discontinue her birth control pills on a trial basis and was fitted with an IUD. She reported a gradual improvement in her symptoms over the next 2½ months. By the end of this time she noted a marked change, with only 1 episode of overeating during the following 3 months. This episode, on Thanksgiving, was not accompanied by the usual drivenness and emotional sequelae. Her mood and self-esteem improved with the abatement of symptoms, and her weight stabilized at 45.4 kg. A follow-up EEG 5 months after she stopped taking the oral contraceptive showed marked reduction of epileptiform activity and was only marginally abnormal.

Comment

Several reports (1–4, 6) have linked binge eating with abnormal EEGs. The significance of small sharp spikes and 14- and 6-per-second positive spikes, which account for the majority of these abnormalities, has been debated. Two groups of researchers (1, 3) have reported encouraging results from treating binge eaters with phenytoin. Rau and Green (4) have reported a correlation between an inventory of soft neurologic signs, including EEG abnormalities, and clinical response to phenytoin.

Hypothalamic dysfunction has been suggested (1–3) as a possible etiology for the binge-eating syndrome. Either an inhibitory effect on the ventromedial satiety center or an excitatory effect on the lateral appetitive center of the hypothalamus could lead to hyperphagic behavior.

Since oral contraceptives exert at least part of their effect at the hypothalamic level (7), they might influence the emergence of binge eating. The almost exclusive occurrence of this syndrome in females, primarily of child-bearing age, further suggests a hormonal effect, although psychodynamic explanations have also been offered (5). These considerations led us to request that our patient stop her medication.

We postulate that the marked improvement in our patient's symptoms and the associated improvement in her EEG resulted directly from her discontinuing the use of birth control pills. These assumptions are limited by our omission of blind technique and our failure to rechallenge the patient with her medication. Nevertheless, the subacute time course of her im-

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provement suggests that this was not a placebo effect.

This case provides further evidence to support a neurophysiologic mechanism for the binge-eating syndrome. It particularly suggests a role for disordered hypothalamic function. Since our patient's symptoms were correlated with an epileptiform EEG pattern, we further suggest that her symptoms resulted from overexcitability of the lateral hypothalamus.

The epidemiology of binge eating raises the question of its frequency of association with oral contraceptive use. While none of the authors cited above indicate which of their patients were using these drugs, we would expect, according to chance alone, that some were. It would be interesting to see whether reanalysis of their data shows correlations between oral contraceptive use and either EEG abnormalities or positive response to phenytoin.

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CT Scan Variants and Genetic Heterogeneity in Schizophrenia

BY ROBBIE CAMPBELL, M.D., PETER HAYS, M.B., DON B. RUSSELL, M.D., AND DAVID J. ZACKS, M.B.

Although it is well known that genetic factors are important in the etiology of functional psychoses, it is also clear that a specific overt genetic predisposition is not always necessary for their emergence. Dalen (1) demonstrated two sets of etiologic factors in a series of manic patients, one set mainly genetic and the other associated with EEG anomalies and brain damage. Subsequently, Hays (2) drew similar conclusions from a series of bipolar manic-depressive patients and later, on the basis of a series of schizophrenic patients, put forward a similar hypothesis about two separate sets of etiologic factors in schizophrenia (3).

Like EEGs, CT scans are painless and safe, and have been reported to show abnormalities more often among schizophrenics than among controls (4). CT scans are therefore suitable for testing the above etiologic hypothesis.

Method

The subjects were 35 outpatients who had been described as schizophrenic by another consultant and by the psychiatrist who proposed them for the study. None of the subjects had epilepsy, neurologic syndromes, or a history of alcoholism, drug psychosis, or more than trivial exposure to illicit drugs other than cannabis. None was adopted. All subjects had been treated with phenothiazines or similar psychotropic drugs.

One of us (R.C. or P.H.) obtained from each patient a family history that covered all first- and second-degree relatives; most histories were supplemented by relatives' accounts. Patients were then divided into two groups; those with and those without a family history of major functional psychosis.

In the course of a separate investigation of the natural classification of the schizophrenias (5), 18 of these patients had had detailed recording of symptoms at the height of the syndrome. Of these 18 subjects, 16 had thought disorders, 19 had hallucinations (auditory in 12 cases), 11 had catatonic symptoms, 15 had delusions that were regarded as primary, and 7 had passivity phenomena. The 7 patients who did not have catatonic symptoms had auditory hallucinations. Of the 11 patients who did not have passivity phenomena, 9 had primary delusions, 1 had prominent catatonic symptoms, and 1 had a syndrome that consisted primarily of thought disorder, tactile hallucinations, visual misrecognition, and secondary delusions.

CT scans were performed using an EMI head scanning unit with a water bag. Six images (160 × 160 matrix) were obtained for each patient. The scans were angled 15° off the orbitomeatal line and a film recording and number printouts were obtained. No sedation or

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contrast enhancement was given. No scans were excluded as uninterpretable on the basis of excessive artifact.

The scans were interpreted independently, without knowledge of the patient's identity, by two neuroradiologists (D.B.R. and D.J.Z.). The patients' ages were disclosed in the second review of cases to aid in assessing the significance of cortical atrophy.

In the initial interpretation, any apparent or suspected variance from absolute normal was recorded. What is normal is still being determined for CT scans and some findings now considered normal may eventually prove to be abnormal. The application of currently accepted norms in the course of the second review caused several changes of category, always from "questionably abnormal" to "within normal limits."

After the second review, the patients were divided into roughly equal groups, one containing those with the most anomalies and the other, those with fewest. A comparison was then made with the genetically based division that had been done independently.

Results

Eighteen subjects had a family history of major functional psychosis in a first-degree relative. Mean ages were 37.3 for the total sample, 39.0 for those with a positive family history, 35.4 for those with a negative family history. The mean interval between the onset of symptoms and the date of the CT scan was 11.5 years overall, so the mean age of onset was approximately 26.

The two analyses of the CT scans did not reveal any reproducible finding common to all or most of the patients. The final interpretations indicated varying degrees of cortical atrophy in 6 of the 7 abnormal scans, a nonspecific finding with many possible causes. The patients with abnormal scans were an average of 6 years older than the normal group, but they had not received significantly more phenothiazines, although their illnesses were generally of greater duration. No other consistent abnormality was encountered in the CT scans. The size of the lateral ventricles was assessed according to the method of Huckman and associates (6). Huckman used approximately 20° angulation and we used approximately 15°, but this difference does not appreciably affect the measurement of the lateral ventricles. Two measurements were taken on two separate levels. Normal size (less than 15 mm by this computation) was only exceeded in one patient (15.5 mm).

Initially, the subjective interpretations seemed to support strongly the hypothesis that there would be more abnormalities among those with a negative family history, but after the results were checked and refined no significant correlation emerged.

Conclusions

Our finding of normal ventricles presumably means that we were looking at a different type of patient from those studied by Johnstone and associates (4). The cortical atrophy we noted may be caused by some feature of the disease process or its management, but on the basis of our findings it does not seem that phenothiazines are implicated.

Although no consistent abnormality was found in the scans of 35 schizophrenic patients and this procedure currently has no place in the diagnosis or management of schizophrenia, computerized tomography, increasingly informative as technical advances improve its resolution, remains of interest in the continuing search for organic features in schizophrenia. The hypothesis that organic findings would predominate in those patients with no family history of schizophrenia was not supported by our results.

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Lithium-Induced Thyrotoxicosis

BY VICTOR I. REUS, M.D., PHILLIP GOLD, M.D., AND ROBERT POST, M.D.

Lithium-induced hypothyroidism and goiter have been described by many investigators, but the actual incidence of such side effects is still open to debate (1). Considering that lithium has been used therapeutically in the treatment of thyrotoxicosis, it is somewhat surprising that lithium-precipitated thyrotoxicosis has also been reported (2, 3). This poses a diagnostic and treatment dilemma in psychiatry as any clinically observed change in behavior may reflect a complex integration of primary symptoms of affective illness, central and peripheral effects of increased thyroid function, and the direct and indirect effects of lithium carbonate.

In this article we describe a manic patient who developed thyrotoxicosis when given lithium carbonate during two hospital admissions. Changes in laboratory values of thyroid function are compared with blind ratings of behavior and the postulated hormonal effects on psychomotor activity are discussed.

Case Report

Ms. A, a 22-year-old woman who satisfied Research Diagnostic Criteria (4) for bipolar I affective illness, was admitted to a clinical research ward at the National Institute of Mental Health. She had a 6-year history of manic-depressive illness and had been hospitalized for acute manic episodes 5 times. Ms. A had been taking lithium carbonate, 1500 mg/day, for several weeks and was euthymic on admission. Triiodothyronine (T_3) and thyroxine (T_4) levels on two separate occasions during her first week in the hospital were within normal limits (80–170 ng/100 ml for T_3 and 4–11 μg/100 ml for T_4). Four days after lithium was discontinued the patient entered a classical manic episode characterized by absence of sleep, irritability, hyperactivity, and grandiosity. Repeat thyroid function determinations at this point showed a dramatic increase in T_4 levels (from 6.0 to 13.4 μg/100 ml). The dopamine antagonists pimozide and chlorpromazine were administered in independent sequential trials, without evidence of substantial therapeutic benefit, although weekly T_3 and T_4 values progressively declined to within a normal range.

Lithium carbonate (1500 mg/day, with plasma levels of .9–1.0 mEq/liter) was added after 3 weeks of phenothiazine treatment and resulted in a rapid decrease in mania. However, 6 days after lithium was started Ms. A's T_3 and T_4 levels increased markedly. These values continued to climb and reached peak levels 2 weeks later (T_3=284 ng/100 ml, T_4=16.5 μg/100 ml). Coincident with this increase in thyroid function the patient, after 2 weeks with no ratable mania, experienced a 17-day recurrence of manic behavior that was indistinguishable from her first episode in the hospital. This episode terminated with no specific adjustment in medication and was associated with a continuing decrease in clinical values of T_3 and T_4. Twenty-four hour radioiodine uptake done 2 weeks after peak T_3 and T_4 levels was approximately 1% but increased to 13% at the time of Ms. A's discharge 2 months later. Anti-thyroid antibodies were noted to be present. The patient was discharged on lithium carbonate and was euthyroid by laboratory evaluation at that time.

One year later Ms. A was readmitted because of a manic episode. Levels of T_3, T_4, and free T_4 (normal value=1.0–2.3 ng/100 ml) at the time of admission were within normal limits and remained so when evaluated at weekly intervals throughout clinical trials of low-dose piribedil and carbamazepine. Ms. A's thyroid-stimulating hormone (TSH) response to an infusion of thyrotropin-releasing hormone (TRH) in a drug-free period following admission was also within a normal range. Since the patient experienced only a partial therapeutic response to carbamazepine, she was again given lithium carbonate (1500 mg/day), which led to a rapid decrease in mania ratings. However, during the third week of lithium administration, Ms. A experienced a 5-day period of hypomanic activity characterized by increased motor and verbal activity and loud, intrusive behavior during ward meetings. T_3 and T_4 levels at this point again showed an increase over normal limits (T_3=232 ng/100 ml, T_4=12.3 μg/100 ml, free T_4=3.8 ng/100 ml). These values continued to rise for a week and then returned to normal limits. The patient's TSH response to infused TRH was severely blunted at this time.

Although no further observable mania was recorded, Ms. A continued to complain of anxiety, restlessness, increased perspiration, heat intolerance, and tremor. Thyroid ultrasound and scan showed homogeneous, well visualized symmetrical lobes, with no evidence of enlargement or solid or cystic nodules. Levels of plasma iodine, 24-hour urinary iodide, and thyroid-binding globulin were within normal limits. Endocrinologic consultation at this time suggested a diagnosis of subacute thyroiditis, but it was noted that certain aspects of the patient's symptom complex could be ascribed as well to a primary affective episode or to lithium carbonate therapy. No specific therapy was recommended because a gradual return to normal thyroid function was expected. This did in fact occur over the next several weeks and Ms. A was discharged in a euthymic and euthyroid state on lithium carbonate, 1500 mg/day.

Discussion

Many of the symptoms (5) of hyperthyroidism and its postulated underlying biochemical mechanisms (6, 7) may parallel those of mania. Although in general lithium has been shown to inhibit the release of thyroid hormone and to reduce the intrathyroid iodothyronine/iodothyrosine ratio, lithium-associated thyrotoxicosis

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When this work was done, Dr. Reus was Staff Psychiatrist, Biological Psychiatry Branch, National Institute of Mental Health, Bethesda, Md., where Dr. Post is Chief, Section on Psychobiology, and Dr. Gold is Chief, Unit on Neuroendocrinology, Clinical Psychobiology Branch. Dr. Reus is now Director of Clinical Research, Inpatient Treatment and Research Service, Langley Porter Neuropsychiatric Institute, University of California, San Francisco, School of Medicine, San Francisco, Calif. 94143.

may also occur. The nature of the phenomenon remains unclear, although Brownlie and associates (2) have suggested it may relate to an "escape" of thyroid hormone following expansion of the intrathyroid iodine pool. Alternatively, the increase in TRH and TSH release that occurs secondary to decreasing plasma levels of thyroxine may be associated with direct stimulant-like activity in brain (8). TRH has been shown to possess strong locomotor stimulant action when injected into the nucleus accumbens and appears, in both behavioral and biochemical paradigms, to act as an indirect dopamine releaser (9). The transient and circumscribed nature of the thyrotoxic episodes in this and previously reported cases suggests that the phenomenon is not a primary effect of lithium carbonate but is related rather to an overadaptive response to lithium's hypothyroid actions. Discontinuation of lithium carbonate during these episodes is therefore ill advised because it would be expected to lead to an acute exacerbation of the thyrotoxic state (10). Thyrotoxic "rebound" secondary to lithium withdrawal was demonstrated in the present case at the onset of the first admission and may have played a role in the precipitation of that manic episode.

It is important for clinicians to be aware that symptomatic changes in patients who have recently been given lithium carbonate or have had the drug discontinued may be related in part to alterations in thyroid metabolism. In initial lithium trials, increases in psychomotor activity and anxiety do not necessarily signify a failure in lithium response but may represent transient compensatory increases in thyroid activity.

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Toxic Psychosis with Cimetidine

BY C. CLIFTON BARNHART, M.D., AND CHARLES L. BOWDEN, M.D.

We have recently seen several patients referred for psychiatric consultation because of acute confusional psychoses that were temporally correlated with cimetidine use. Cimetidine, a recently introduced H_2-receptor antagonist, has quickly achieved broad use as an inhibitor of gastric acid secretion in peptic ulcer disease (1). Investigation before commercial release did not indicate any CNS effects, toxic or otherwise (2). Two examples of acute, severe confusional psychoses that started soon after cimetidine treatment are described below.

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Case Reports

Case 1. A 58-year-old man was hospitalized with abdominal pain that was thought due to cholecystitis. Laboratory studies were within normal limits, except for an elevated white count (12,800 cells/mm³). Two days after he was admitted the patient underwent a cholecystectomy. On the second postoperative day he was given 300 mg of I.V. cimetidine over a 6-hour period. Four hours after the infusion the patient became acutely confused, with visual hallucinations and bizarre speech. The next day he was given 100 mg of chlordiazepoxide, with no improvement in his mental symptoms. He continued to receive cimetidine orally, 300 mg q.i.d. The next day he was given 75 mg of magnesium pemoline orally, without improvement. Psychiatric consultation was then obtained. On examination he was confused and disoriented and had fluctuating levels of consciousness. Cimetidine and magnesium pemoline were discontinued, and within 24 hours the patient's mental status was entirely clear. His further postoperative course was uneventful.

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Case 2. A 58-year-old woman had a long history of chronic undifferentiated schizophrenia that had been well controlled with perphenazine, 24 mg/day, and amitriptyline, 150 mg/day. She was also taking Maalox and Donnatal for peptic ulcer disease. Symptoms of melena and coffee ground vomitus led to gastroscopy, which revealed an active pyloric ulcer. Cimetidine, 300 mg t.i.d., was started on an outpatient basis. On the second day, after the fourth dose of cimetidine, the patient became confused and disoriented. Although she did not take more medication, she remained confused throughout the day and that night her family found her wandering outside her home. Her psychiatrist was consulted and she was advised to discontinue all medications. Her confusion cleared within 24 hours after discontinuation of her medications. All medications except cimetidine were restarted and she had no further problems.

Discussion

Four physicians in England, where cimetidine has been marketed for a longer period of time, have published letters reporting CNS toxicity (3–6). Two additional cases have been reported in this country (7, 8). The symptoms and clinical course of the CNS toxicity are similar to this report. Within hours after drug institution or dosage increase, the patient becomes confused and disoriented, is often agitated and restless, has unintelligible or bizarre speech, and may have hallucinations, predominantly visual. Upon discontinuation of cimetidine the confusional state clears rapidly, and within 24 hours no symptoms remain. The patient is amnestic for the episode. No sequelae are apparent.

In most of the reported cases of cimetidine CNS toxicity, several identifiable factors are present:

1. *Coexisting psychiatric illness and psychotropic medications.* In addition to the patient described in case 2, we have seen a similar psychosis in a unipolar depressed patient who was taking tricyclic antidepressants. Whether it is the psychiatric disorder or, more likely, the psychotropic medication that increases vulnerability is undetermined. In all cases, the psychotropic agent had strong central anticholinergic effects that can cause a toxic psychosis.

2. *Old age.* All the patients we have seen have been at least 56 years old. Confusional psychoses have been reported in three patients over the age of 70 (5, 8). This apparent age-associated risk could be related to higher peak blood and CNS drug levels or be secondary to impaired CNS function.

3. *Cerebral impairment.* Impaired brain oxygenation might increase the risk of toxicity from cimetidine. Marginal cerebrovascular supply is more common in the aged. One patient we observed had a mild preexisting organic brain syndrome. The patient described in case 1 developed symptoms in the immediate postoperative period, during which the effects of anesthesia or the cardiovascular stress of surgery could impair cerebral function.

4. *Overdose.* In one report, an otherwise healthy 25-year-old man developed a confusional psychosis after taking 12 g of cimetidine (3), and in another (4) two patients, 34 and 50 years of age, inadvertently took double the prescribed dosage and became acutely confused.

Comment

The rapid development of CNS confusion in certain circumstances after cimetidine use, the similarity of the clinical picture in the reported cases, and the rapid, complete clearing of symptoms after discontinuation of cimetidine suggest that the drug causally contributes to the clinical reaction described. The frequency of this reaction is presumably low but unknown. Since readily identifiable circumstances appear to be associated with a risk of CNS toxicity, it seems advisable for physicians to exercise particular caution when prescribing and monitoring cimetidine in high-risk situations. Psychiatrists should be aware of this possible contribution to organic brain syndrome. Further investigation is needed to determine the frequency and the pathophysiology of this effect.

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LETTERS TO THE EDITOR

This section contains reactions to Journal *articles, statements of opinion, comments on Association activities, requests for information, etc. Letters should be submitted in duplicate to the Editor, who makes all decisions regarding publication. Letters must be typed double spaced throughout and should not contain more than 500 words and 5 pertinent references. Criticisms of published articles will automatically be sent to the author(s) for response. Letters will be edited for clarity and conformance with* Journal *style. We regret that we cannot inform writers of the disposition of letters or return those which are not printed.*

Tardive Dyskinesia Not Associated with Neuroleptics

SIR: The most prominent hypothesis advanced to explain the pathophysiology of tardive dyskinesia suggests a hyperactivity of dopaminergic neurons in the basal ganglia that results from prolonged dopamine blockade induced by chronic neuroleptic treatment (1). We have seen two cases of buccolingual-masticatory syndrome after long-term use of the MAO inhibitor tranylcypromine given in conjunction with lithium carbonate to two patients with bipolar affective disorder. Neither of these patients had received neuroleptic medication.

Case 1. Ms. A, a divorced housewife, was referred for psychiatric assessment at the age of 62 because of depression and chronic back pain for which no physical cause was found. She had had a severe manic attack previously. The patient's only sister had a history of bipolar affective disorder that had responded to lithium prophylaxis.

Ms. A was started on 900 mg/day of lithium carbonate, resulting in a serum lithium level of 1.1 mEq/liter. She continued to be depressed, and antidepressant medication was added. The most successful antidepressant proved to be tranylcypromine, 40 mg/day, which she took for 3 years. During the latter part of this therapy, Ms. A began to develop involuntary chewing movements associated with the sensation of a phantom mass lodged against the upper palate just behind the maxillary central incisors. These symptoms did not cease when the tranylcypromine was stopped for 12 months and lithium carbonate was given alone.

Case 2. Mr. B, a 38-year-old married real estate agent, had been in good physical health before developing a manic-depressive disorder when he was in his 30s. During his hypomanic phase he was more effective, but his depressed episodes seriously impaired his functioning. The patient had been on 30 mg/day of tranylcypromine for 4 months before he was given 1200 mg/day of lithium carbonate simultaneously for prophylaxis (serum lithium=1.0 mEq/liter). Mr. B continued to take this drug combination over the next year and a half. During the reduction of the tranylcypromine dosage he began to complain of persistent involuntary chewing movements that had not ceased when the antidepressant was discontinued.

Tranylcypromine is said to exert its effect by inhibition of monoamine oxidase required for the catabolic oxidation of the putative catecholamine neurotransmitters norepinephrine and dopamine, as well as serotonin. In vitro acute studies have shown this compound to be inactive on [^{3}H]haloperidol and [^{3}H]dopamine binding associated with dopamine receptors in calf brain membranes (2). Lithium has been reported to produce cogwheel rigidity (3) and reinduce tardive dyskinesia in patients (4) and to inhibit norepinephrine and dopamine receptors in rats (5).

The above two cases suggest that chronic administration of tranylcypromine with lithium may result in dopamine receptor hypersensitivity or that there may be other mechanisms to explain tardive dyskinesia than those suggested for chronic neuroleptic treatment.

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Subjects Sought for Naltrexone Study

SIR: The National Institute on Drug Abuse is planning a study of the use of naltrexone, a narcotic antagonist, in the treatment of opiate abuse and addiction. One study protocol is planned for patients under the care of private practitioners. Physicians interested in possible participation are invited to write the coordinating body, Biometric Research Institute, Inc., 1010 Wisconsin Ave., N.W., Suite 505, Washington, D.C. 20007, for full details.

SAMUEL C. KAIM, M.D.
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Sex Roles and Group Therapy Dropouts

SIR: In "The Group Psychotherapy Dropout Phenomenon Revisited" (December 1978 issue), Leslie M. Lothstein,

Ph.D., wondered why so many men dropped out of his groups and attempted to explain the phenomenon on the basis of both patient hostility and group dynamics. However, an explanation based on the sociology of sex roles may be appropriate.

Three factors in masculine socialization may be important here: masculine inexpressiveness, the myth of masculine competence, and avoidance of intimacy with other men (1). A group situation challenges all of these. In group therapy, men are encouraged and expected to ventilate feelings with appropriate affect; this is something men all too often do not know how to do and thus find threatening. In addition, expression of feelings is considered to be a sign of weakness (2). Men are expected to be expert problem solvers and to be competent at all times; participation in group therapy is an admission of one's inability to solve personal problems, which is a major blow to masculine self-esteem. Finally, group therapy forces men to become emotionally intimate with other men, a particularly anxiety-provoking social situation that men are socialized to guard against. Thus, flight from group therapy may be a response to overwhelming anxiety and major threats to one's self-concept. As men use aggression to defend against anxiety, it is certainly possible to perceive these men as hostile.

Accepting the dropout phenomenon as necessary to the development of group cohesion, as suggested by Dr. Lothstein, may not be the solution to this problem. Rather, as I have outlined elsewhere (3), therapeutic interventions to confront and work with specific masculine issues are vital in preparing men for group psychotherapy.

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KENNETH SOLOMON, M.D.
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Dr. Lothstein Replies

SIR: In my paper I stressed that the white men who dropped out of treatment may have experienced a particular kind of stress early in group therapy which needs "to be addressed in a special way." Dr. Solomon offers an alternative explanation for our findings. He attributes the phenomenon to the sociology of sex roles versus patient hostility and group dynamics. He also seems to suggest that by attending to specific masculine issues, the dropout phenomenon can be reduced or eliminated. Although it is currently in vogue to explain away many social-psychological ills by resorting to sex role theory (1), such an enterprise, by overgeneralizing a point and ignoring group dynamics, grossly oversimplifies a complex human phenomenon.

The importance and complexity of preparing patients for group therapy are well established (2). Dr. Solomon's main point, that during the pregroup patient preparation the therapist(s) should pay special attention to how the sociology of male sex roles may make some men more vulnerable to dropping out of the group, is a worthy clinical addition to the group literature.

Dr. Solomon's theory, however, cannot account for why 50% of our male patients remained in the group or why about 20% of our female patients dropped out. Moreover, his theory has the disadvantage of ignoring individual differences and implying that women do not experience difficulties similar to those of male patients.

In our paper we suggested that not all groups would experience the same stresses for cohesiveness. Therefore, one would expect differences in the reasons for the dropout phenomenon in other groups that we noted in our student-led groups. Dr. Solomon seems to have ignored this point. While I agree with Dr. Solomon that the sociology of sex roles may play an important part in the dropout phenomenon, I disagree that this is the case for all patients. Furthermore, it may also play a role for some of the male therapists!

Entering group therapy can be quite stressful for some patients. The more we know about how we can facilitate a patient's entry into group therapy, the better able we will be to serve the mental health needs of our patients.

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Advisability of Combined Treatment

SIR: Although we were interested to learn of a patient who survived a doxepin overdose while taking a tranylcypromine-doxepin combination ("Tricyclic Overdose in a Patient Given Combined Tricyclic-MAOI Treatment" by Kerrin White, M.D., November 1978 issue), we question the use of an MAO inhibitor with a tricyclic at the stage in this patient's treatment when the combination was introduced. Dosage was not cited in the article, but we assume the patient described by Dr. White had failed to respond to therapeutic doses of chlorpromazine and amitriptyline. He was then started on the tranylcypromine-doxepin combination rather than a more conventional treatment regimen such as tranylcypromine alone (1), tranylcypromine combined with lithium (2), or ECT (3, 4). We can find no basis for such early administration of a regimen which, according to current opinion, should be reserved for refractory cases (5). MAOI-tricyclic combinations may well prove to have a role in the treatment of refractory depressions, but failure to respond to chlorpromazine-amitriptyline does not constitute treatment refractoriness.

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Dr. White Replies

SIR: Although the patient's history of inadequate response to chlorpromazine and amitriptyline did contribute to my decision to treat him with a tricyclic-MAOI combination, I consider such a history unnecessary to justify clinical use of the combination. Rather, I may start treatment with the combination in the hope of improving the likelihood or rapidity of therapeutic effect, particularly when I am faced with a negativistic patient who is unlikely to cooperate with protracted treatment.

Controlled data bearing on these considerations are lacking. However, reviews (1-3) of uncontrolled clinical series suggest a higher frequency of improvement with combined tricyclic-MAOI treatment than with either medication used alone and indicate a total absence of dire consequences, given elementary precautions. The idea that combination treatment poses special risks seems to have arisen from the unhappy concordance of a few unusual cases with a biochemical theory explaining adverse reactions to combination treatment as another instance of the "tyramine reaction" with MAOIs; this idea then became widely accepted without further proof and has become, in Ayd's words, "one of the several myths or unscientific generalizations that plague contemporary psychopharmacotherapy" (4).

Rejecting the notion of the dangerousness of combined treatment as superstition, one may ask, "Why not prescribe the combination on a routine basis?"

Indeed, we need controlled data bearing on the efficacy as well as the safety of combination therapy for nonrefractory depression. I am currently engaged in an open, randomized pilot study comparing amitriptyline alone (up to 300 mg/day), tranylcypromine alone (up to 40 mg/day), and the combination of both in half dosage with depressed inpatients who have not necessarily been refractory to previous treatment. With results for only 21 available to date, I can say nothing about comparative efficacy, but I have seen no increased incidence or severity of side effects with the combination as compared with either drug used singly.

From a theoretic standpoint, one might even suggest that combining the drugs could improve safety, since tricyclics inhibit the tyramine-induced pressor response (5), considered the prototype of the hypertensive crises that have deterred many from prescribing MAOIs—although this may represent another overreaction to inadequate data.

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KERRIN WHITE, M.D.
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In Defense of the Internship Year

SIR: In "The Internship Year: A Negative View" (October 1978 issue) Seymour L. Halleck, M.D., stated that there are basically two arguments for the internship: 1) "There is something about the development of a sense of physicianship during the internship year," and 2) "An expanded knowledge of nonpsychiatric medicine is absolutely essential" if a psychiatrist is to function as a consultant to his colleagues.

I disagree with his view that these reasons are not valid. Dr. Halleck criticized the first as vague and the second as false. He stated that the "something" about the development of a physician which occurs during the internship year is "never clearly defined" and "almost mystical." He believes psychiatrists do not need to know a great deal of medicine to act as consultants because consultees "want a consultant who will assess the degree of psychopathology" and "rarely will they welcome a consultant who spends a great deal of time discussing medical issues about which they already possess superior knowledge."

A sense of physicianship is neither vague nor mystical, but blends confidence, humility, and perseverance. A physician needs the confidence to accept responsibility for clinical decisions, the humility to accept errors that may result in harm or even death to patients, and the perseverance to continually try to improve his skills. These qualities can only be acquired by the experience of being a physician. This experience is often painful. Because of the pain, clinical experience with the physically ill will often be avoided by those who need it the most. When the psychiatric resident begins his psychiatric work he may have great difficulty learning to be a physician. Unlike other physicians the psychiatrist can shield himself from taking personal responsibility for life and death decisions. As a resident he can refer serious problems to his supervisor, as a psychiatrist he can join a team of nonpsychiatrists who make clinical decisions democratically, or he can join the "retreat from patients" described by Kubie and become an administrator, teacher, or researcher (1).

The one area where the psychiatrist cannot avoid responsibility for clinical decisions is as a consultant to his colleagues. He must be able to integrate medical, psychological, and social data.

Unfortunately we cannot rely on our nonpsychiatric colleagues' undoubted superior knowledge of medical issues to be operative in the case of the patient they refer to us. Medical and surgical colleagues frequently miss medical diagnoses on patients they refer to psychiatrists (2). Saravey and Koran reported that physicians missed "obvious" organic disease in 4% of medical and surgical inpatients referred to them for psychiatric consultation (2). These authors point out that our nonpsychiatric colleagues tend to refer to psychiatrists those patients who upset them and therefore cause them to exercise poor medical judgment. My own experi-

ence at Kings County Hospital Center is similar (3). Meyer and Mendelson described a state of "active alienation" in the doctor-patient relationship in all inpatients referred to psychiatrists (4). This alienation causes poor clinical judgment.

As a psychiatric consultant, I have supervised the work of 150 second-year residents over the past decade. Between 25% and 30% had no internship. With 3 or 4 exceptions their work as consultants ranked in the bottom half of their group. About one-fourth of the total residency group was outstanding but only 2 of those without an internship qualified for this description. Karasu and associates reported similar findings (5).

Psychiatric residents without internships are rarely able to integrate medical, psychological, and social data to produce a treatment plan. Almost all show great difficulty in accepting responsibility for seriously ill patients and for advising colleagues. A few try to deal with the problem by avoiding patients. Most deal with their fears of responsibility by acting as a conduit for the opinions of their supervisor, who acts as an auxiliary professional ego. This meets the service needs of hospitals, but the internshipless psychiatrist never develops a professional ego of his own and never becomes a competent physician or an effective consultant.

Dr. Halleck is quite right that psychiatry departments should not be responsible for training in medicine, pediatrics, or family practice. Psychiatric residencies should return to a three-year program, accepting as residents only those who have completed at least one year of training in a medical, pediatric, or family practice program. He points out that "understaffed county hospitals in inner cities would welcome American graduates whether or not they are going into psychiatry." After completing such a year psychiatric residents will indeed have become physicians and will have the necessary foundation to become psychiatric specialists. Our patients would gain by the care of highly motivated physicians. Medical graduates who wish to avoid clinical experience might do better in a nonclinical specialty or one in which stressful decisions are not such a large part of patient care. Our specialty might gain both stature and recruits by a commitment to clinical excellence rather than by the commitment to filling residency programs at the cost of clinical excellence, which has caused the loss of both.

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LEWIS GLICKMAN, M.D.
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Dr. Halleck Replies

SIR: I agree that the decision to ask psychiatry departments to develop their own internships in primary care areas was unwise. I would certainly prefer a system in which residents were required to take a full year of a rotating internship or a primary medical care internship to the system we have now.

In other areas, Dr. Glickman and I disagree. I do not believe that physicians have a monopoly on developing an optimum blend of confidence, humility, and perseverance. Other professions such as the ministry, education, or law could make similar claims to having a unique capacity to develop such virtues. Dr. Glickman's blend of desirable qualities simply describes a competent professional who is also a decent human being. His argument does not clarify the mystical nature of "physicianship."

Dr. Glickman's observation that nonpsychiatric colleagues tend to miss diagnoses on patients they refer to psychiatrists does raise serious questions. It provides a rational argument for having psychiatrists know as much nonpsychiatric medicine as possible. But putting the burden on the medical expertise of the psychiatrist is only a palliative response to the problem. The real issue here is medical education. If nonpsychiatric physicians are so careless in examining psychiatric patients, this represents a terrible indictment of medical education. Training psychiatrists to compensate for the poor clinical attitudes of other physicians will not resolve this problem. Rather, we should be concerned with why we are graduating physicians who experience a state of "active alienation" from psychiatric patients.

Dr. Glickman provides some anecdotal data about the failures of residents without internships. I could provide more anecdotal data about their successes but this would not clarify anything. There are no scientific data in this area, and I would still be critical of making policy decisions without attempting to obtain such data. Dr. Glickman's contention that the "internshipless psychiatrist never develops a professional ego of his own and never becomes a competent physician or an effective consultant" may be a little overstated. We have now graduated hundreds of such individuals, many of whom have assumed positions of leadership in our profession. I pray that Dr. Glickman is wrong and that they are not all incompetent and ineffective.

Two years have now passed since I wrote my article. Although I have received considerable feedback which convinces me that I was right in being critical of the practical value of reinstating the internship year, I am no longer so committed to my theoretic critiques. It may reassure both Drs. Romano and Glickman to know that I do see a good many residency applicants who are uncertain as to their future identity as physicians and very much want a good internship. At the same time, however, I also see other applicants who do not seem to want or need an internship. If we ever get around to studying this issue rather than debating it, I suspect we would find that some applicants need an internship and some do not. Certainly the provision of a good internship year is essential for those applicants who feel they need it.

Further theoretic discussion of this issue without the availability of new data is wasteful. For better or worse, a fourth year of training is now required and there are no alternatives to concentrating on making it a good one. Our biggest problem is finding training sites for nonpsychiatric medical experience for our residents. I hope that we can stop arguing with one another and join forces in working toward persuading our medical schools to develop decent internship positions for psychiatric residents.

SEYMOUR L. HALLECK, M.D.
Chapel Hill, N.C.

Predicting Imipramine Response

SIR: The article by Frederic Quitkin, M.D., and associates on "Imipramine Response in Deluded Depressive Patients" (July 1978 issue) could be misleading. Many clinicians want to know if the occurrence of psychosis causes poor response to imipramine in depressed patients; the authors concluded that their data did not support this hypothesis. Although they cautioned against generalizing their data to other groups of deluded depressive patients, the general tone of the article did not follow this caution.

Several problems weaken the data. The Perceptual Distortion Scale used as one criterion to determine the presence of psychosis included the items "disorientation for people" and "disturbed by hostile impulses." Patients with a positive rating on any item were considered psychotic, but these items are not necessarily signs of psychosis. Another item was "implausible belief." This may have included only delusions, but since the severity of delusions was "generally rated as slight to mild," one cannot be convinced that this item included only definite delusions. Thus, the sample selected by the Perceptual Distortion Scale may have included persons who did not have true psychosis. If this was the case, then one would not expect to find differences in response to imipramine between the two groups ("psychotic" versus nonpsychotic).

The next set of criteria for psychosis seems more valid: the presence of separate psychopathologic features, i.e., conviction of sinfulness or persecution and belief in external control. Unfortunately, only one symptom yielded a treatment group with more than four patients. Such small sample sizes make it difficult to detect statistically even a large difference in response to treatment. Therefore, the authors presented a misleading conclusion in stating that no statistical difference was found in the response to imipramine on the basis of presence or absence of separate psychotic symptoms. I regret that hallucinations were not included in the analysis of separate symptoms; my own preliminary (and inconclusive) data suggest that hallucinations predict poor response to tricyclics.

Dr. Quitkin and associates stated that delusions and hallucinations were not prominent features in their patients and that these features were generally rated as slight to mild. This may explain the discrepancy between their results and those of other researchers, who have found that delusions predict a poor response to tricyclics (1–5). I agree that caution is required in generalizing the data to other groups of deluded depressed patients.

Many of the data presented by Dr. Quitkin and associates are provocative but weak. The stronger data seem to involve the "depressive" type of delusions, such as conviction of sinfulness. My own preliminary data suggest that depressive delusions (those of nihilism, self-deprecation, and somaticism) may respond to tricyclics, while other delusions may not respond. Therefore, the important issue for tricyclic response may not be delusions per se but depressive versus nondepressive delusions and hallucinations.

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RICHARD E. MINTER, M.D.
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Dr. Quitkin and Associates Reply

SIR: Dr. Minter's criticisms were met in our paper. First, our definition of "delusional" is a liberal one, and a different sample with more severe psychotic symptoms might have shown different results. Second, the lack of a statistically significant difference between the delusional and nondelusional group in this sample does not prove there is no difference. The possibility of a type II error is present, i.e., asserting there is no difference when in fact there is.

His suggestion that nondepressive delusions are particularly refractory to treatment is interesting but also requires systematic substantiation. A definitive answer will come only from further research. Our major point is that the present data are inconclusive and it is therefore premature to avoid a trial of a tricyclic antidepressant in a psychotic depressive. Most patients have a strong preference for drug treatment compared with ECT. Rejecting such preferences requires strong grounds.

Incidentally, Dr. Minter states that we had no more than 4 patients with any one psychotic symptom when in fact 11 imipramine-treated patients had conviction of sinfulness.

FREDERIC QUITKIN, M.D.
ARTHUR RIFKIN, M.D.
DONALD F. KLEIN, M.D.
New York, N.Y.

Warning Against Generic Substitutes

SIR: I would like to report a case in which substitution of the generic tricyclic for a brand name adversely affected a patient.

The patient, a 66-year-old woman, first became depressed 8 years ago. The depression was associated with diurnal mood variation, early morning awakening, anorexia, and weight loss. The patient responded well to a proprietary brand of imipramine. In January she became depressed again and resumed taking the proprietary brand of imipramine, to which she responded at a dosage of 150 mg/day. In October, the depression and vegetative signs reappeared. She had not altered the way she had been taking the medication, and we could not find any life event or anniversary that could explain the exacerbation of the depression. We increased the dosage of her medication to 200 mg and then to 250 mg/day over the course of 2 weeks, but she did not respond.

At this time I learned that the patient had not been getting the proprietary brand but a generic substitute. Instead of increasing the dosage I again prescribed 250 mg/day of the proprietary brand but this time ordered that there be no substitution. Within 3 days the patient's sleep began to improve and she felt less hopeless and less depressed. Two weeks later, all of her vegetative signs were in remission and her mood was "normal."

This patient's case demonstrates the possibility that generic substitutes are not always equivalent to the brand name product. Unfortunately, the testing and blood levels that would be necessary for rigorous proof of this assumption were not available in this case. I hope that such a study can be done.

SAMUEL I. MILES, M.D.
Los Angeles, Calif.

Neuroleptic-Potentiating Effect of Metyrosine

SIR: Metyrosine (α-methyl-*p*-tyrosine, AMPT), an inhibitor of tyrosine hydroxylase, decreases the synthesis of dopamine and, according to the dopamine hypothesis of schizophrenia, may be useful in treating schizophrenia. In addition, supplementing neuroleptic medications with metyrosine might allow a reduction in total neuroleptic dose, although earlier trials with this approach produced conflicting results (1, 2). We conducted an investigation to examine the antischizophrenic and extrapyramidal effects of metyrosine compared to haloperidol and placebo.

Twelve schizophrenic patients, ranging in age from 20 to 60 years and with a minimum of 2 years' illness duration, gave informed consent to participate in a double-blind crossover trial. After a gradual reduction in the previous neuroleptic dosage produced a clear exacerbation of psychotic symptoms, patients were given either metyrosine or haloperidol in randomized order for 4 weeks; doses were increased until the optimum effect was obtained. Placebo followed this active drug treatment until psychotic symptoms again intensified, at which time each patient received the alternate test drug for 4 weeks. Semi-structured interviews, recorded on videotape before and at the end of each treatment period, were assessed blindly by independent psychiatrists using the Brief Psychiatric Rating Scale (BPRS). Neurologic signs were documented on a specifically designed rating scale (3), and the patients' behavior was recorded by the nursing staff on the Nurses' Observation Scale for Inpatient Evaluation (NOSIE). Routine laboratory tests were monitored.

The initial neuroleptic dosage reduction was 50%–75% (mean=68%). Metyrosine dosage was 1.5–3.0 g/day (mean= 2.75 g/day) and haloperidol dosage 1.5–15 mg/day (mean= 5.0 mg/day). The BPRS and NOSIE scores were significantly reduced during treatment with both metyrosine ($p<.05$) and haloperidol ($p<.05$) compared to the respective placebo scores. There was no significant difference between metyrosine and haloperidol BPRS scores. The psychiatric state of 2 patients deteriorated during metyrosine treatment but improved when their previous neuroleptic dose was restarted. Parkinsonism was slightly but significantly increased during haloperidol administration compared to placebo ($p<.05$); a nonsignificant trend in the same direction was seen with metyrosine. There was no significant difference in parkinsonism or akathisia scores when metyrosine and haloperidol were compared. Correspondingly, the use of anticholinergic drugs during the two treatments was not significantly different. Neither metyrosine nor haloperidol induced abnormal laboratory values.

This study confirms the previous report that metyrosine has neuroleptic-potentiating effects (1). However, severe schizophrenic symptoms could not be managed satisfactorily with metyrosine. The modest antipsychotic effect and the potential for aggravating extrapyramidal symptoms suggest that the combination of metyrosine and neuroleptics does not offer a clinical advantage in the treatment of schizophrenia, although tyrosine hydroxylase inhibitors may still prove useful as a research tool in elucidating the pathogenesis of psychoses.

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JES GERLACH, M.D.
DANIEL E. CASEY, M.D.

Celiac-Type Diets in Schizophrenia

SIR: In "Another Look at Gluten in Schizophrenia" (November 1978 issue) James R. Rice, M.D., and associates reported that a schizophrenic patient who had been hospitalized for more than 13 years improved substantially on an 8-week experimental "gluten-free and milk-free diet" and was discharged. Another patient, hospitalized for 14 years, regressed when gluten was added to the hospital diet and improved considerably on the subsequent gluten-free and milk-free diet but was not discharged because she discontinued her diet when not supervised. The results in the other 14 chronic schizophrenics in the study were not discussed.

The authors state that their work did not duplicate the success reported by Dohan and Grasberger (1) and Singh and Kay (2). I would emphasize such duplication could not be expected because of crucial differences in patients, experimental design, and diet. My comments pertain only to the differences between their investigation and mine.

Dr. Rice and associates studied 16 chronic schizophrenics whose current hospitalizations ranged from 6 months to 10 years. My study included over 100 recently relapsed schizophrenics who were randomly assigned to control or experimental diets on the day of their admission to the hospital.

Although the diet used by the South Carolina investigators did not include obvious cereal grains or milk, it did contain some milk protein and wheat gluten. They used butter, and the bread was made with wheat starch that contained 0.2% gluten (personal communication). Symptomatic and morphologic effects of apparently small amounts of wheat gluten in celiac disease, which I have proposed as a possible model (perhaps genetically related) for studying the pathogenesis of schizophrenia, are well known. For example, Dissanayake and associates (3) have stated that for adult celiac patients as little as 0.5 g of wheat gluten a day is a "large amount" because even smaller doses prevented the return to near normal of the gluten-damaged intestinal mucosa.

My experiments were designed to eliminate all traces of glutens in all cereal grains because schizophrenia does occur (although possibly less frequently) in societies in which rice and/or other cereal grains (4) but little or no wheat, rye, or

barley (the grains omitted in the so-called gluten-free diet for celiacs) are eaten. All traces of milk were eliminated because milk sometimes worsens celiac disease. Furthermore, Zioudrou and associates (5) at the National Institute of Mental Health, stimulated by evidence relating cereals to schizophrenia (1, 2, 4), have reported naloxone-reversible endorphin ("exorphin") activity in peptic digests of milk casein and corn and barley glutens and, most notably, an exorphin peptide from wheat gluten that is about as potent as met-enkephalin. Other fractions from all tested glutens had a stimulating effect.

Dr. Rice and associates' study used only 8 weeks of diet, but patients with adult celiac disease, as well as the genetically related chronic disorder dermatitis herpetiformis, often do not show significant clinical improvement (which occurs in most but not all cases) for many months after a celiac-type diet is instituted. For this and other reasons I suggested in 1973 that an experimental therapeutic trial of diet in persistently symptomatic chronic schizophrenics should continue for 6 months to a year (1).

I thank the South Carolina investigators and dietitians for their interest in the food-derived peptide hypothesis of the etiology of schizophrenia and for their generosity in supplying me with the unpublished information I have cited above.

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F.C. DOHAN, M.D.
Philadelphia, Pa.

SIR: I read with interest the report by Dr. Rice and associates on gluten in schizophrenia. However, for reasons enumerated below, I found the study to be a less than satisfactory attempt at testing the "gluten hypothesis" or replicating our investigation (1–3).

First, if the continuous presence of a pathogenic factor has already resulted in a profound, long-lasting illness, there seems no reason to expect that increasing the quantity of that factor would have any further effect. Furthermore, for reasons such as the "ceiling effect" of ratings, any worsening of already severely ill patients may not even register. Therefore, the first part of their experiment, in which they added more gluten to a normal gluten-containing diet, was not a valid test of the hypothesis that cereal-grain gluten may be pathogenic in schizophrenics. It would have been much better to start the patients on an exclusion diet and then challenge them with the hypothesized pathogen. It would have been better still to use the ABA design we employed in order to permit control of the order effect of treatments (2, 4).

Second, the chances of proving any etiologic hypothesis in terms of treatment effects may be small if the test is conducted exclusively in patients with histories of long-standing intractable illness and institutionalization because of the likelihood that disease effects have become irreversible and the pathologic patterns of thinking and behavior have become habitual. This is not to say that malignant and chronicity-prone forms of schizophrenia should not be studied, but rather that they should be studied earlier in the course of illness to minimize the chances of erroneously rejecting a hypothesis because of the effects of chronicity. Thus, most of the patients in our study were acute or subacute, but 80% of them were of the poor prognosis or nuclear variety. Only 3 of the 14 patients tested were of the type involved in the investigation of Dr. Rice and associates.

Third, schizophrenia is recognized to be heterogeneous, so the results of any treatment depend on the degree to which patients responsive to that treatment are represented in the sample. Sample composition is a particularly critical issue in small studies. Dr. Rice and associates included schizo-affective patients, whom we excluded, and studied very few nonparanoid patients (hebephrenic and catatonic), who comprised 80% of our sample and accounted for most of the "gluten responders."

Considering these factors, which unduly increased the likelihood of type II error, one cannot regard this study as a fair test of the gluten hypothesis. Indeed, under the circumstances it is remarkable that a clear dietary effect was found in 2 of the 16 patients. This study does not warrant the authors' pessimism about the gluten hypothesis but should instead encourage further investigations with careful attention to the above-mentioned and other methodological issues (2–4).

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MAN MOHAN SINGH, M.D.
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Dr. Rice Replies

SIR: I wish to thank Dr. Dohan and Dr. Singh for their comments and critiques. I feel their observations should be kept in mind in future projects. Hindsight is often better than foresight. I hope my project will stimulate increased interest in the refinement of this subject.

JAMES R. RICE, M.D.
Columbia, S.C.

Applause for the End of a "Drought"

SIR: I read with great interest the article "Training Psychiatrists for Working with Blacks in Basic Residency Training Programs" (December 1978 issue) by Walter H. Bradshaw, Jr., M.D. This article was refreshing, particularly given a

three-year drought of articles about black mental health issues written by black authors. Could it be that the process of selecting this type of work for publication should be scrutinized?

This decline in the publication of work related to black mental health is apparent not only in the *Journal* but also at most recent annual meetings of the American Psychiatric Association. I long for the day when sessions on the subject of black mental health will be offered for continuing medical education credit; this would signal the importance of knowledge about black patients. Regrettably, the cause of black mental health has not been kept before our membership, and this has undoubtedly led to a complacency among black psychiatrists.

I would point out that the American Psychiatric Association has probably been the most sensitive and forceful medical specialty group in recognizing the health problems of minorities, including women and the elderly.

JAMES H. CARTER, M.D.
Durham, N.C.

A Question of Balance

SIR: "Genetic Counseling for Psychiatric Patients and Their Families" by Ming T. Tsuang, M.D., Ph.D. (December 1978 issue) admirably sets forth some of the central issues, as well as reviewing core information in this area. A good candidate for inclusion in the author's list would be epiloia (tuberous sclerosis), a disease that is not uncommon among institutionalized retarded persons and is probably transmitted in an irregular dominant mode (1). Despite the paucity of attention it has received in the psychiatric literature, tuberous sclerosis seems to have a relatively high frequency of associated psychopathology (2, 3).

This oversight is indicative of a broader problem relating to the role of psychiatry in mental retardation. A computerized search I conducted of the past 5 years' literature elicited fewer than 50 "hits" for the topic of combined disability, with less than 15 articles on combined mental retardation/psychiatric impairment in major English language psychiatric publications. For those who see this phenomenon in terms of economic determinism, this sad state of interest can be compared to the number of publications on drug-related topics. Is anyone listening?

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Possible Mechanism of Lithium's Effect in Bronchial Asthma

SIR: We would like to comment on the article "Coincidental Improvement in Asthma During Lithium Treatment" by Suhayl J. Nasr, M.D., and Robert W. Atkins, M.D. (September 1977 issue), who reported 2 cases of a prophylactic effect of lithium on bronchial asthma; and on a letter by Peter L. Putnam, M.D., who reported 2 additional cases of remarkable remission of asthma in patients started on lithium carbonate (letter to the editor, March 1978 issue).

Drs. Nasr and Atkins suggested several possible biochemical mechanisms for this effect. However, they did not mention that one of the leading theories of the biochemical defect that predisposes individuals to asthma suggests an unresponsive β-adrenergic receptor (or β-adrenergic adenylate cyclase) in the bronchi (1). Lithium has been shown to inhibit β-adrenergic adenylate cyclase in vivo in patients with therapeutic blood levels (2), and thus one might expect lithium therapy to aggravate, not ameliorate, asthma.

We wish to report recent data from our laboratory that might explain this paradox. In the cerebral cortex of rats treated for three weeks with lithium in food, β-adrenergic adenylate cyclase was found to be inhibited after noradrenaline stimulation. However, basal levels of cyclic AMP (in the absence of noradrenaline stimulation) were significantly increased (3). Thus lithium appeared to have a biphasic effect, activating adenylate cyclase when the enzyme is inactive and inhibiting the enzyme's full activation. These results would be consistent with lithium's prophylactic effects in both depression and mania.

We predict that chronic lithium therapy would prevent recurrent asthma but would reduce the effectiveness of epinephrine treatment in the acute asthmatic attack. This theory reconciles the β-adrenergic theory of asthma and the increasing evidence that lithium affects β-adrenergic adenylate cyclase.

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HAIM BRACHA
RICHARD EBSTEIN
ROBERT H. BELMAKER, M.D.
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The Medical Model Revisited: A Question of Citation

SIR: It is virtually axiomatic that good scholarship includes understanding and recognizing previous contributions, especially in any effort to set forth a new position.

In this respect, "The Illusion of Simplicity: The Medical Model Revisited" by Herbert Weiner, M.D. (July 1978 Supplement) is wanting as a piece of scholarship. The author did not mention the contributions of Miriam Siegler and Humphry Osmond, summarized in *Models of Madness, Models of Medicine* (1).

Siegler and Osmond (1) emphasize that the medical model includes a special encounter and relationship between physician and patient. The physician, acting powerfully and authoritatively (but not authoritarianly) confers the sick role on the patient. The patient with the sick role is excused from usual activity, responsibility, and behavior and is entitled to

the sympathy of other people and society, but is obliged to cooperate to get well. The doctor offers diagnosis, prognosis, therapy, and hope. Diseases are assumed to have natural causes if etiology is uncertain or unknown.

In light of Siegler and Osmond's statement, Dr. Weiner's error was to say nothing of that special encounter between doctor and patient. He explains the medical model merely as an outdated view of disease that should be expanded to incorporate observations and experimental data from diverse levels of organization, such as cultural, evolutionary, and molecular factors.

Weiner's views are reminiscent of those of Engel (2), who also did not acknowledge Siegler and Osmond. Curiously, both Weiner and Engel discuss and cite Ludwig (3) as the first author on their respective reference lists. Ludwig (3) discussed Siegler and Osmond with appropriate citation. Why did neither Weiner nor Engel pick up this important lead to the literature?

It may be a fitting irony that Dr. Weiner's article was published in the Festschrift for Francis J. Braceland, M.D., who is mentioned in Siegler and Osmond's acknowledgments (1, p. xi).

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Distinguishing Between Mood and Affect

SIR: "Mood and Affect: A Semantic Confusion" by Howard Owens, M.D., and Jerrold S. Maxmen, M.D. (January 1979 issue) helped to clarify this muddy area by recognizing the inferential nature of information about emotional states and offering clinical guidelines for more lucid descriptions of such states.

I would, however, question their conclusion that "perhaps the main distinction between mood and affect is that inferences about mood stem from present observations and past events . . . while inferences about affect usually pertain only to current observations." Custom and usage offer a more valuable distinction.

The common modifiers of the term "affect," e.g., shallow, constricted, monotonous, blunt(ed), flat(tened), (in)appropriate, of full range, suggest that we are describing not so much the emotional state per se as the subject's capacity to cope with that state, the extent of the subject's apparent awareness of his own emotions, and his ability to communicate them to others. When we use the term "affect" to describe the range, intensity, and appropriateness of the emotional state and how the subject communicates evidence of this state to the observer, we make a valuable distinction between "affect" and "mood."

Drs. Owens and Maxmen defined mood as "a disposition persisting over time" or as "a prolonged emotion," but we refer here not to conventional units of time but rather to persistence of the emotional state through periods when internal or external events would ordinarily be expected to alter that state. Thus we can describe in a brief mental status examination a mood that prevails throughout the interview despite the occurrence of events that invite change in the emotional state.

HERBERT BENGELSDORF, M.D.
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Drs. Owens and Maxmen Reply

SIR: There is clearly a difference between the experience of emotion and the manner of expression of emotion. We would reiterate, however, that the word "affect" is commonly used not merely to refer to manner of expression but rather in ways that are indistinguishable in meaning from mood. One often hears the statement, "The patient's predominant affect during the interview was depression." Such statements do refer to emotional state per se.

If we understand Dr. Bengelsdorf correctly, he is arguing that a mood is a persisting emotional state and that affect refers to alterations (or the lack of alteration) in the awareness, appropriateness, or expression of that mood. Our major contention has been that it is simply clearer to refer to such changes themselves as changes in expression, recognizing that even in a short interview a patient may show an array of complex, contradictory, or confusing emotions, sometimes in rapid sequence. A manic patient may be laughing and joking, then abruptly burst into tears; if such a patient is extremely labile we may be hard pressed to describe any persisting mood. A person in mourning may appear saddened, then begin to laugh at the recollection of an old family joke about the deceased. Clearly there is great variation in the range, intensity, and appropriateness of emotional expression. We continue to believe, however, that it adds little to label a person's sadness as mood and his laughter as affect.

How are we to distinguish between observations which tell us about mood and those which indicate affect? The distinction is deeply engrained, but in both instances we are really making inferences from the same array of observations.

HOWARD OWENS, M.D.
JERROLD S. MAXMEN, M.D.
White Plains, N.Y.

More on Mood and Affect

SIR: In "Mood and Affect: A Semantic Confusion" Howard Owens, M.D., and Jerrold S. Maxmen, M.D., stated that "inferences about mood stem from present observations and past events (and therefore belong in a patient's history rather than as part of his mental status examination), while inferences about affect usually pertain only to current observations." I was taught the same distinction, but by means of a simple, easily remembered analogy: mood is to affect as climate is to weather.

WILFRID L. PILETTE, M.D.
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BOOK REVIEWS

Controversy in Psychiatry, *edited by John Paul Brady, M.D., and H. Keith H. Brodie, M.D. Philadelphia, Pa., W.B. Saunders Co., 1978, 1,071 pp., $39.50.*

Disagreements on controversial issues are often published as an exchange of letters following an author's stated views in a scientific journal. Brady and Brodie chose controversial topics that are timely and important from a scientific, medical, or societal perspective. Another criterion for selection specified that reasoned arguments could be made from different vantage points. Two or more experts known to have a position on the issue prepared essays defending a point of view without benefit of seeing the opposite opinion. A brief summarizing commentary by one of the editors concludes each chapter.

There are 26 questions, such as, What is the future of psychiatry as a medical specialty? What is in the future of psychoanalysis? Is psychosurgery ethical and effective? Does ECT have a place in the therapy of schizophrenia? Are drugs more than palliative in the treatment of schizophrenia? Are CNS-activating drugs of value in the treatment of the hyperactive child? Is behavior therapy a fad or a new direction for psychiatry? Is there evidence for specificity in psychotherapy? Does psychotherapy alter the course of schizophrenia? What are the indications for group therapy? Should homosexuals adopt children? What are the medical hazards of marijuana? Should psychiatric patients ever be hospitalized involuntarily? Can informed consent be obtained from a psychiatric patient? Is community psychiatry a slogan or a new direction?

I found myself asking whether the topics selected were representative of major controversial issues in psychiatry today. Would the experts chosen be ones who would offer opinions I would value? Were the arguments offered well stated? Did the method of presentation add useful new information?

The topics selected are good ones. Perhaps one's own list of controversial issues could produce some suitable replacements for long-term drug therapy in the treatment of depression, the management of chronic alcoholism, biofeedback, or genetic counseling.

The essayists are well chosen and most are recognized experts in the area addressed. I would have selected someone other than the doctor from NIMH for a presentation on the future of psychiatry. Most are familiar with his attacks on psychiatry. Respect for the wisdom of an essayist strengthens the acceptance of an opinion. A discussion of involuntary admissions to mental hospitals and a focus on dangerousness might have been more convincing than the presentation by the familiar exponent of psychiatry as a myth. The commentary by one of the editors highlighting the major points made by the essayists and adding his own critical evaluation brought the differing viewpoints more clearly into focus.

In general the answers to the questions were well stated and backed up with supportive data. Some essayists did a superb job in providing a comprehensive overview of their topic. Particularly noteworthy was the chapter on management of alcoholism, which was an excellent summary of current research. The overview on drug therapy in schizophrenia and the review of the hyperactive child syndrome were outstanding.

Occasionally there was more detail than necessary to arrive at a reasoned position on an issue. It is a long book; it could have been a better one with pruning. The method of presentation consolidates in one place arguments for and against positions or issues recognized as controversial. It is useful in formulating one's own opinion.

It is a good book; I enjoyed reading it. It is highly recommended as a resource in the resolution of current controversial issues in psychiatry for psychiatrists and all mental health professionals as well as by those in administrative posts in the mental health system.

WALTER E. BARTON, M.D.
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Karen Horney: Gentle Rebel of Psychoanalysis, *by Jack L. Rubins. New York, N.Y., Dial Press (Dell Publishing), 1978, 350 pp., $9.95.*

Karen Horney is one of the great figures in the history of psychoanalysis and psychiatry. Her theory contributed a new dimension to our understanding of human nature. Jack Rubins, a long-time student of Horney's theory and a contributor to its development, has done us a fine service: he has brought together a mass of details about Horney's life as well as the cultural and historical conditions in which she lived. She lived in a time of vast change, encompassing World Wars I and II, world-wide depression, and the rise of Hitler. She immigrated to the United States at a time of changing values and growing awareness of the destructive capacity of man's technology as well as the creative investigation of the human mind uncovered by Sigmund Freud.

Rubins conveys how Horney's curiosity, vitality, and intellectual capability led her to an ever-widening search for answers to the problems she encountered in her life and in confrontations with her patients. His language and style make vivid the urgency Horney felt as she moved from the limited views of Freud into an awareness of the paradoxes opened by Kierkegaard, Tillich, and other philosophers, whom she read with care. Rubins implicitly shows how, in her clinical work and in her thinking, Horney was able to juxtapose differences without rushing to place them into some rigid framework. Like Freud, she put actual observation first and only later in her life did she begin to synthesize. Her freedom to observe allowed her a true rigorousness.

It is clear from this book that Horney's theories grew out of her need to comprehend the fragments of her life and her observations. Rubins shows how Horney came on the idea that the life process was a solution in itself. In *Our Inner Conflicts* and *Neurosis and Human Growth,* she began to detail overall forms of life as constellations of behavior that attempt to solve the essential problem of basic anxiety. She saw that the orientation of scientific psychoanalysis was primarily toward symptoms, that it was concerned with thought objects and such fragments as drives, and that the analyst

attempted to understand by connecting one symptom to another through cause and effect. Her revolutionary insight was to go beyond the obvious, the visible, and to look at the invisible, and to observe beyond particularities into the holistic workings of the personality. As she saw the use of solutions for deeper insecurity and the emergence of so-called drives and impulses from a ground of feeling, Horney gained more understanding of the whole structure of neurosis. She began to recognize the falseness of this structure and to ask what would produce a unified person—a person who is wholehearted and not fragmented into such parts as ego, id, and superego or idealized self and despised self.

These questions led her to Zen Buddhism, an interest that seemed to be a natural outgrowth of her life work. At the outset of her work, she made many different clinical observations. As Rubins shows, her early papers are detailed descriptions of different patients that contain an unusual understanding of each one. Although she developed a typology, she was careful to note that everyone exhibits something of every type. Only after seeing the diversity did she begin to ask how these pieces fit together to form a solution. Although she found many nodal points, such as the comprehensive pride system and the basic conflicts, it was clear to her that neurosis is ultimately a state of fragmentation. In Zen, she came into an arena that appreciated the true nature of this fragmentation and even offered a way to go beyond it. Horney saw that psychoanalysis, at least as developed by Freud, was involved in the fragmentation itself and that a more comprehensive, holistic therapy was needed.

Although Rubins' biography is excellent, it lacks that ingredient which would have left us feeling that we understood what Horney went through. One can sympathize with the problems Rubins faced in writing about Karen Horney: she seems elusive, secretive, and closed about her personal struggle; but we yearn to know more of what went into her creative processes. Her theory differed fundamentally from other work in that she appreciated the fact that neurotic process was a transformation from a healthy growth via deeper basic anxiety into compulsive, driven behavior.

While Freud and the mainstream of psychoanalysis were caught in a mechanical paradigm in which quantitative distinctions were made, Horney saw the qualitative distinctions between health and illness. What was it in Horney's life or spirit that gave her this insight? She was always more related to artists and philosophers than to other theorists, and these affinities seem to lie at the core of her understanding. Perhaps it was the relationship with her mother that was the basis of her deep faith in growth. That relationship seems to have contributed to her sensitivity to the problems of women. Unfortunately, Rubins does not help us with these kinds of questions, nor does he clarify what went into the inductive leaps Horney made when she developed her unique concept of the real self. However, Rubins goes a long way toward bringing this important person into focus and titillating our appetite to know more about Karen Horney.

DAVID SHAINBERG, M.D.
New York, N.Y.

Edward Jarvis and the Medical World of Nineteenth-Century America, *by Gerald N. Grob. Knoxville, Tenn., University of Tennessee Press, 1978, 287 pp., $14.00.*

Gerald Grob, professor of history at Rutgers University, belongs to that group of intellectual and social historians who have brought to the history of psychiatry a new rigor and enthusiasm by applying the tools and insights of social and intellectual historians to the history of American psychiatry. Among his books are *Mental Institutions in America: Social Policy to 1875* (1) and *The State and the Mentally Ill* (2).

Edward Jarvis (1803–1884), a leading physician, psychiatrist, and statistician of Massachusetts, presented his "A Report on Insanity" to the Massachusetts Legislature in 1855. It was the most significant statistical social survey of mental illness in the nineteenth century and became the prototype for later investigations. Although the dust jacket of this book states that he was "one of those responsible for reshaping the profession," Jarvis was never in the mainstream of American psychiatry. He was passed over for the medical superintendency of McLean Hospital, Worcester State Hospital, and Northhampton State Hospital.

After graduation from Harvard College and Harvard Medical School, Jarvis began his career in general practice. He became interested in mental disease and later devoted himself exclusively to that specialty, in which he soon acquired a national reputation. He achieved his greatest fame in the study and collection of medical statistics, particularly those concerned with insanity, and he served as an influential adviser to the federal census officials from 1850 to 1870. During his entire career as a psychiatrist, he treated inpatients in his own home.

Grob has done the best he could with a rather dull and wooden figure. His scholarship is meticulous, and the social and intellectual background of Jarvis' period is beautifully drawn. Grob's presentation elicits little empathy for the hair-splitting, rigid, sanctimonious character of Edward Jarvis, but the book is a superb biographical account. Grob magnificently reveals the dilemma of nineteenth-century American psychiatry—caught between the specific germ theory, which was transforming the medical profession to the search for specific causes of disease, and psychiatric practice and philosophy, which were geared toward holistic preventive medicine.

The book is well designed, is on good paper, has notes at the end of each chapter, and has an adequate index. This book will appeal to historians of psychiatry and to those practicing psychiatrists who wish to know about their intellectual roots.

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G. EDMUND GIFFORD, JR., M.D.
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Jungian Psychotherapy: A Study in Analytical Psychology, *by Michael Fordham. New York, N.Y., John Wiley & Sons, 1978, 175 pp., no price listed.*

This is the first in a series of books edited by Prof. Boris Semeonoff of Edinburgh University on methods in psychotherapy. The selection of Michael Fordham to present Jungian theory and method is a fortunate choice. Fordham is well known, articulate, and thoughtful. He has presented

Jungian thought extensively through the *Journal of Analytical Psychology* as well as in books and other writings. He is an editor of the Bollingen edition of Jung's collected works.

Jung, like Freud, lived a long and continuously productive life. His interests changed over the years, and the many organizations deriving from his thought have varied greatly in their emphasis. Dr. Fordham says that the London school has been brought "much nearer to psychoanalysis" and "in particular the Kleinian school . . . somewhat to the consternation of the more conservative members." Fordham's particular interest in the Kleinian school stems from his early career as a child psychiatrist and his observation that Jung was not especially interested and did not write much about the development of children, whereas Melanie Klein's career was built on her imaginative and creative ideas about childhood.

The first section of the book is devoted to a condensed exposition of Jung's theories. The rest describes Dr. Fordham's own modifications of traditional Jungian methods, his underlying theory, and his technique. Traditional analytical psychologists stress the processes of amplification and education with extensive development of mythological and historical themes, rather than the more specific analysis of the patient's personal unconscious, past experience, current problems, and current symptoms. A long-time criticism of Jung's method is that it does not deal effectively with aggression. Fordham recounts one tale in which Jung dramatically suppressed aggression and commented that then the analysis could proceed. Fordham sets limits to the amount of physical interaction and acting-out that he allows—more than Jung would tolerate. Fordham's description of a patient's behavior (painting his face and neck red, biting him) would not be acceptable to some analytical psychologists.

Fordham also does not like the recording of dreams, a characteristic tool of Jungians. He feels that by the time they get to the analytic hour dreams may be cold, and the patient may be in quite a different place psychologically. Furthermore, he discounts the usefulness of Jung's widely used typology as a therapeutic tool. He grants the typology a place in theory and research but believes that it is not as fixed as many Jungians think and may thus be misleading in its explication of human interaction.

In the last two-thirds of the book Fordham describes his own techniques and methods and the theory behind them, contrasting his approach with Jung's. Fordham stresses regression, transference, and countertransference more than is usual in Jungian writings. Traditionally, the Jungian analyst sits facing the patient. In an atmosphere of shared dialectic two people seek an answer. It was Jung's practice to see patients not more often than twice a week—often less—except in times of crisis. He took long, previously announced vacations and expected his patients to do the same. He frequently recommended places for them to go and hoped they would enjoy themselves, as he expected to.

Clearly, Jung's patients were not as sick as Fordham's. Jung was frankly afraid of "latent psychosis" and would terminate analysis if he smelled one in the offing. Many of his patients were in the second half of life, were intellectual, had had reasonable success, and had intact egos. Many were in mid-life crisis, and depression was a leading feature. Fordham, on the other hand, treats patients with character disorders and a "selected number of psychoses." He sees them three, four, or more times a week, and he uses the couch. In contrast to Jung's attitude toward vacations, Fordham fears that taking long weekends or holidays may cause his patients to fall apart.

Fordham discusses regression, transference, countertransference, and interpretation. He discusses the transference neurosis, a term not used by Jungians, and the importance of its resolution. He recognizes and discusses the archetypal elements in transference. Jung was not much concerned with resolution and interpretation of the transference, except when it was interfering with analysis, as in the case of the "inflated transference." Jung's patients were not as dependent or as regressed as Fordham's, and he felt generally that the transference would take care of itself and was not doing any harm anyway. In fact, he thought the transference might build a bridge to another relationship. In line with these differences, Fordham underplays symbol formation and its use in individuation and the amplifcation education technique in general. He says that placing stress here emphasizes the analyst's interest and ignores the resolution of the patient's conflicts in the transference neurosis. He tends "to replace them with a kind of religion in which the study of symbolic material takes first place."

Parts of this book have been published elsewhere, and thus there is a certain amount of repetition. The book constitutes a useful and clearly stated exposition of the position, theory, and practice of a well-known and experienced analyst of the London school.

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To Be an Invalid: The Illness of Charles Darwin, *by Ralph Colp, Jr., M.D. Chicago, Ill., University of Chicago Press, 1977, 276 pp., $17.50.*

Charles Darwin's chronic and disabling illness has perplexed a succession of scholars, beginning with the great evolutionist himself, who at the age of 16 started to observe his own symptoms while practicing medicine under his father's supervision. The elder Dr. Darwin for many years advised and treated his ailing son for a host of complaints, including abdominal distress, gaseous vomiting, skin eruptions, anxiety, and depression. During his voyage on the *Beagle* Darwin was often severely seasick, and later he often became ill as a result of "thinking evolutionary thoughts," experiencing "mental pain . . . when he searched for a plausible cause for evolution."

Darwin feared that marriage would interfere with his work, and he dreaded pregnancies (but he married his cousin Emma, a devoted and caretaking woman who bore him 10 children). By the age of 33 Darwin was so debilitated that he could only take short walks, and he moved to the country hoping to improve his health. After his father died, he transferred his medical dependence to Dr. James Gully, who started him on a lengthy hydrotherapeutic regimen. He later leaned heavily on a number of quite prominent British physicians, including Queen Victoria's personal doctor, Sir William Jenner.

Only during his middle years was Darwin able to write the books that made him famous. Worried about the hostility his evolutionary theories might evoke from religious leaders, he developed what Dr. Colp describes as a "severely negative and masochistic identity." Darwin tolerated criticism poorly, noted himself to be "burning with indignation" when disagreed with, and became seriously depressed during his 50s. But as he gained recognition in his 60s there was a gradual improvement in health, and he died rather peacefully at the age of 73 of coronary artery disease.

Dr. Colp's masterful analysis of Darwin's invalidism is divided into two sections. The first is a detailed description of all his various symptoms and treatments, and the second is a dispassionate review of the many theories that have been proposed to explain the illness. These include possible metabolic diseases (gout, hypoglycemia, porphyria), infections (Chagas' disease, malaria, amebiasis), anatomic problems (hiatus hernia, eye-strain), toxicity (arsenic poisoning, allergy to pigeons), and psychological factors (overwork, unconscious hostility toward his father, bereavement). Curiously, Dr. Colp, a psychiatrist, says nothing about Darwin's childhood development (the history begins when Darwin is in his teens), nor does he comment on the fact that no data are available about Darwin's sexual life. Nearly half of the book is devoted to Dr. Colp's sources of information, including the extensive memoranda kept by Darwin and his family about illnesses and medications. Darwin carried on a lifelong, probably psychotherapeutic correspondence with his best friend, Dr. Joseph Hooker, and Colp relies heavily on these letters.

The book is extremely well organized, with copious footnotes, and should serve as an antidote to some of the more speculative "psychobiologies" (1) that lately have been published about famous people. Indeed, Dr. Colp's intellectual honesty reminds one of Darwin's own scientific style, in that he comes to the modest conclusion that "it may be impossible to ever be certain about the causation of his illness." Nevertheless, Colp believes that "psychological stresses were the most probable cause."

This book can be recommended not only for its scholarship but also for the basic message that great suffering is not incompatible with extraordinary achievement. Taking issue with Sir George Pickering's view of Darwin's illness as a desirable part of Darwin's creativity (2), Dr. Colp finds that Darwin was creative in spite of his illness, not because of it.

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PETER F. OSTWALD, M.D.
San Francisco, Calif.

Hypnosis at Its Bicentennial: Selected Papers, *edited by Fred H. Frankel, M.B.Ch.B., D.P.M., and Harold S. Zamansky, Ph.D. New York, N.Y., Plenum Press, 1978, 301 pp., $27.50.*

These papers were presented during the Seventh International Congress of Hypnosis and Psychosomatic Medicine in Philadelphia in 1976. About 600 medical hypnotists from 19 countries shared their ideas; from among these, only 29 contributions have been selected for publication in this book. *Hypnosis at Its Bicentennial* represents the best possible introduction to the variety of interests, theories, methodologies, and practices of modern hypnosis. The book has been scrupulously edited by an astute clinician, Fred Frankel, and an enlightened experimental psychologist, Harold Zamansky. It contains E.R. Hilgard's latest comprehensive concept, neodissociation, and K.S. Bowers' superb theoretical formulations of absorption and dissociation. There are sections on the history and theory of hypnosis, experimental thought and methodology, and clinical studies on the efficacy of hypnosis in treating migraine, on a burn unit, and in alleviating problems in cancer, anxiety, asthma, sexual dysfunction, pain, airplane phobia, and multiple personality.

In "What Did He (Bernheim) Say?" A.M. Weitzenhoffer clarifies Bernheim's often-quoted statement, "There is no hypnosis only suggestion," by emphasizing that Bernheim had also pointed out that hypnosis was a "suggestion state" and a specific "condition of enhanced suggestibility."

J. Knox and associates discuss "Hypnotic Analgesia vs. Acupuncture in High and Low Hypnotically Susceptible Subjects" and state that not only was hypnotic analgesia more effective than acupuncture in reducing pain but 4 out of 12 highly susceptible subjects reported virtual elimination of cold pressor pain while under hypnosis. They conclude that "the relationship between hypnotic susceptibility and response to acupuncture is at best weak." The problem of whether hypnosis is actually a psychophysiological state is discussed by V.E. Rozhnov of Russia, whose data indicate that "when the subject enters a deep stage of somnambulism, there occurs a shift of consciousness with accompanying changes in the decasecond rhythm of the brain's bioelectric activity. These decasecond rhythms . . . are related tc hypnotic suggestions." J.C. Ruch's paper on "Self Hypnosis with Implications for Other Self Control Procedures" includes the statement that "self hypnosis is as effective on both objective and subjective measures as heterohypnosis in an *experimental* situation."

E. Dengrove and A. Lazarus, writing on the topic of hypnosis and behavioral therapy, agree that these techniques can be combined to great advantage. D. Ewin's "Clinical Use of Hypnosis for Attenuation of Burn Depth" indicates that hypnosis may be a significant factor in rapid healing and resistance to infection.

For special commendation I would select the papers of D.R. Collinson of Australia on asthma and by J.H. Hilgard and A.H. Morgan on "The Treatment of Anxiety and Pain in Childhood Cancer Through Hypnosis."

An adequate index is provided. I highly recommend *Hypnosis at Its Bicentennial* to anyone who is interested in knowing more about the theoretical concepts, research aspects, and practical interests of modern hypnosis.

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Psychoanalysis, Creativity, and Literature: A French-American Inquiry, *edited by Alan Roland. New York, N.Y., Columbia University Press, 1978, 355 pp., $20.00.*

Surprisingly enough this symposium-produced book is aptly titled and smoothly written. It bears the mark of an expert editorial hand, and Roland's introduction nicely sets the scene. The translations are excellent, avoiding what Aring once called the sense of grazing on a tether.

The three indexes include "Literary Works," with 28 references, and "Subject Index," with 19 references to "desire" and 13 references to poetic matters. Perhaps this is the French influence; certainly French psychoanalysis has become closely associated with philosophy, linguistics, literature, and structural anthropology. American psychoanalysis is also tied to the humanities, and one of the four sections of the book is all-American: the section on creativity, in which we are treated to Albert Rothenberg's groundbreaking creativity research.

This book is a particular pleasure for a reviewer because it has so much on words, language, and writing. André Green tells us of the power of writing to promote affects, a power so great that we sometimes prefer the affects generated by writing to those of life. He says language has pressure and that words, sentences, and style have irradiation and recoil effects. Green sees the writer standing on the fringes of the crowd, unnoticed, keeping to himself, forgetting and forgotten, inscribing a sign on a surface for someone absent.

We meet the unique and typically French phenomenon, Jacques Lacan, a man with the secret of arousing people and passion and a person of such influence that French psychoanalysis has been accused of becoming populated by "little Lacanians." Lacanian concerns are the relation of the unconscious and linguistic structure, "listening without knowing" (approaching the patient's associations without preconceived categories), and the need to be in touch with the patient's unconscious desire. The concern of others is Lacan's five-minute analytic sessions.

In the section on sociological perspectives, psychoanalysis in France is described as having become a fashion—one in which the image of the analyst as *barbu avec lunettes* has changed so that those on the Left are the most rather than the least favorably disposed to it.

In the section on psychoanalysis proper, we are told that what is being played out in the unconscious is the life of desire, with each part of our bodies speaking in the forgotten language of love. The problem is, as the actor says in Bergman's *Scenes from a Marriage,* "We are illiterates when it comes to love."

In a paper on "The Disconnected Self" we are reminded that the psychoneurotic patient of Freud's time has almost disappeared from the consulting room. Patients come today because they are no longer willing or able to bear their crumbling self-esteem. We see the narcissistic personality disorders, and the term "alienation" is used for the malaise of our time, replacing the banal use of "neurotic."

The section on creativity echoes Owen Barfield, noting that words are signs of natural facts and that each is borrowed from something material, something appreciated by one of the five senses, in particular the sense of touch or feeling. The self selects key words and they shape further developments of the self.

In this section Rothenberg spells out first his "Janusian thinking": the simultaneous conception of opposite, contradictory ideas or images (i.e., symmetry) and, second, his "homospatial thinking": the conceiving of two or more discrete entities occupying the same space (i.e., unity). He tells us that the homospatial conception leads to integration, an essential feature of metaphor. He notes that metaphors occupy a central place in the creations of painters, writers, sculptors, architects, and other types of artists, as well as applied and theoretical scientists. Since neither the artist nor the scientist is aware of the structure of the thinking, ideas seem to come out of the blue, but the creator is not suffering from divine madness. He or she is a more sensitive and flexible thinker capable of using and blending modes of thought in unusual ways.

Another essay on creativity notes that the artist must wrest his or her uniqueness from the collectivity in which he finds himself, yet create a product in harmony with the culture. As Otto Rank pointed out, the artist must appoint himself as an artist. The experience of the therapist's belief in and respect for individuality creates the atmosphere of trust in which arises the will to change and create.

In a work on creation of plays—or any piece of writing, for that matter—it is hypothesized first that the play deals at some level of consciousness with the writer's feelings about his own creativity and, second, that in every play the cast of characters represents a projection of elements of the playwright's identity. It is suggested that study of an author's plays will consistently reveal an internal gallery of characters from his past.

The section on psychoanalysis and literature contains a piece on the third face of psychoanalysis: what is "out there" and its relation to what is "in here." This is the world of the self versus nonself, and "identity" is the key term. We read one another like music, hearing ourselves play our lives like variations on a melody, yet, as Freud said, our egos lose their boundaries when we fall in love or have other mystical experiences. This may happen when we become "absorbed" in literary or artistic works.

Another paper reviews ways psychoanalysts have explored literature. First was the elucidation of the universality of unconscious fantasies from psychosexual stages of development. Second was investigation of the unconscious motivation or psychopathology of a character. Third was relation of the hidden psychological meanings in a work of art to the author's life, e.g., Freud's work on Leonardo da Vinci and Kligerman's study of Pirandello and his *Six Characters in Search of an Author.*

An essay on narcissism quotes Proust on how people in love seek themselves in the objects of their desire. They endow the desired object with mystery and beauty that really flow from themselves. The self radiates enough energy to transfigure commonplace reality into its own image and turn it into poetry. Everybody believes that someone else possesses the self he wants to acquire; this is why everyone experiences desire.

A final note states that every successful cure implies integration of the subject's psychic bisexuality; inversely, every serious threat to sexual identity or integrity is likely to lead to a variety of disturbances to the point of psychosis. Belonging to a sex, it is said, constitutes one of the firmest kernels of personality coherence.

This book is well written, well translated, and wise. It is also entertaining, and the various "Literary Works" constitute a tasty, easily digestible mélange. It is recommended for psychiatrists as well as those readers interested in creativity and literature—the lovers of the Word.

MORGAN MARTIN, M.D.
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The Painted Message, *by Otto Billig and B.G. Burton-Bradley. Cambridge, Mass., Schenkman Publishing Co. (New York, N.Y., Halsted Press, John Wiley & Sons, distributor), 1978, 255 pp., $19.50.*

This book breaks new ground in anthropology, psychiatry, and "creativity" as reflected in the mentally disturbed patient. It takes a comprehensive and multidisciplinary approach that is original. It enables the reader to find entrance into the distorted world of the schizophrenic patient through the "painted message."

The "chronicity" of the schizophrenic patient originates, for the most part, from an inability of the therapist to establish, stimulate, and enhance nonverbal communication when verbal channels are blocked, disturbed, or meaningless. This is the principal idea of the book; it is well documented and exemplified by the clinical cases and the "paintings" of the

patients. The text is leavened by clinical cases, which skillfully complement the theory.

The authors focus on dynamic and developmental approaches in chapters titled "The Stage Is Set," "Creativity and Psychotic Disintegration," "Schizophrenic Expression and Western Art," and "Magical Thinking." They endorse the theory that the schizophrenic process is a regression to early stages of development in all functions of the mind, including communication and creativity. Therefore, the "art" of the schizophrenic patient shows and is typefied by fusion of ego boundaries, lack of self-differentiation, and disturbances in dealing with external and internal space. Furthermore, the paintings of schizophrenic patients are characterized by transparency, lack of or mixing of perspective (vertical, horizontal, and diagonal), presence of a "base line," *perspective tordue,* absence of "geschtalt," amoeboid and amorphic elements, and appearance of primordial universal symbols when the conventional ones have lost their meaning.

The symbolic language of the painting, like dreams, has to be understood and interpreted by the therapist to uncover the latent meaning obscured by the manifest one. This is based on the great similarity between spontaneous painting and dreaming. Furthermore, the authors argue—justifiably, in my opinion—that the painting has properties that are therapeutic and reconstructive and not only cathartic, as is usually presumed.

The chapters on "The Conception of Space" and "The Structure of Space" deal with the patient's perception of space. Looming in the background of the authors' comments are philosophical, historical, and biopsychosocial aspects of space. Their discussion of space pathology is a major contribution in this field. In some cases the pathology of space indicates the patient's relationship to external objects (object relationship), his or her sociability and integration with society, and his or her perception of the body image and the self and external world (depersonalization and derealization).

The chapters "Crosscultural Diagnosis in Psychiatry," "New Guinea and Its People," "Societal Significance of Tribal Art," and "Schizophrenic Expression and New Guinea Art" stress the universality of the basic psychopathology of the schizophrenic process and its creative expressions. At the same time the authors do not lose sight of the diversity and social-cultural-ethnic determination of secondary signs and symptomatology, or of their elaboration and expression. It is generally accepted, as the authors state, that "the regression to common universal patterns in severely disintegrated patients, regardless of cultural backgrounds, suggests a common basis for the development of personality structure and space concepts."

The book includes an ample appendix, a glossary of technical terms for nonprofessional readers, and extensive and suggestive references that are most useful for the casual as well as the professional reader. However, details are overabundant and at times repetitious.

Some of the issues raised in the book remain controversial and deserve further study and consideration; e.g., the differences between modern, primitive, tribal, and prehistoric art and "psychotic art" or artistry of the mentally ill. Another issue left unresolved is the impact of mental illness on the creativity of the artist. It is not at all obvious that creativity deteriorates after mental illness. Some changes do occur, but they are not always detrimental.

I strongly recommend this book to all who deal with mental health, creativity, art (history, theory, and critique), and education as a very important source of knowledge, experience, and guidance in their everyday practice.

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Psychopharmacology of Thiothixene, *by Thomas A. Ban, M.D. New York, N.Y., Raven Press, 1978, 236 pp., $12.50.*

Rather than review a category of psychotropic agents, the author has chosen to study one of the thiozanthene series in depth. His review of thiothixene is both thorough and exhaustive.

The book is organized so that the first eight chapters discuss basic topics such as the history, synthesis, metabolism, biochemistry, physiology, and pharmacology of the drug. The next three chapters deal with clinical use, including indications for administration, side effects, and comparison with inactive placebo and other drugs that have established therapeutic indications. Thus, it is possible for the clinician to ignore the first part of the book if he or she wishes and to obtain helpful data regarding clinical applications of the drug. On the other hand, those who wish to learn more about thiothixene will find everything they need to know in the earlier chapters. There is also an excellent bibliography at the end of the book.

There are many tables in this book, some of which are so detailed that they obfuscate rather than clarify. Nevertheless, this form of in-depth careful analysis of a single drug could become a model for future dissemination of information about psychotropic agents. One might legitimately question, however, whether thiothixene is of sufficient importance in relation to other psychotropic agents to merit this degree of detailed scholarship.

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Effective Ingredients of Successful Psychotherapy, *by Jerome D. Frank, Rudolf Hoehn-Saric, Stanley D. Imber, Bernard L. Liberman, and Anthony R. Stone. New York, N.Y., Brunner/Mazel, 1978, 212 pp., no price listed.*

The point of this book is that the effective ingredients of successful psychotherapy cut across all approaches and "schools" of treatment. In explicating the nature of this common denominator, Dr. Frank and his associates cite evidence from various medical specialties, including psychiatry, demonstrating the beneficial results when patients and therapists expect that the treatment will work. They quote Freud's assertion in 1905 (1) that "expectation . . . coloured by hope and faith . . . is an effective force with which we have to reckon . . . in *all* our attempts at treatment and cure."

This point is already part of the folk wisdom of psychotherapy for practitioners and researchers alike—in no small part the result of the widely published work of Frank and his group. Their work may be in the third phase of the evolution of a theory as William James is reputed to have described it: after first being attacked as absurd, then regarded as true but obvious and insignificant, it finally has reached the stage where it is regarded as so important that its adversaries claim it as their own.

In chapter seven, which "contains the authors' combined

reflections," they become more specific about the mode by which these expectations operate. They take the position that a broad range of symptoms, which may be either "exaggerations of commonly experienced states" or the traditional phobias and obsessions, are best viewed either as manifestations of "demoralization, or as self-defeating strategies to combat [demoralization] by eliciting supportive responses from important others." Their view is that "whatever their origin, symptoms can contribute to the patient's demoralization by diminishing coping capacity." Thus, they conclude that the "effectiveness of all forms of psychotherapy largely depends on their ability to restore morale through combating destructive emotions . . . and foster health-promoting ones. . . . This is also achieved by enhancing the patient's sense of mastery or control over himself and thereby [over] his environment."

The assumptions in this position are made explicit on page 171: "The thesis that emerges from our studies is that, although both patients and therapists assume that the patients come to psychotherapy primarily for relief of specific symptoms, the chief reason they do so is that they are demoralized." Demoralization is defined operationally by the authors as the perception of patients themselves, as well as by others, that they have failed to cope with some aspect of life through their own inadequacies. I might sharpen this definition on the basis of research carried out with Tobin (2, 3) by pointing out that shame at the inability to cope (which to us seems synonymous with feeling demoralized) is often heightened by the need for help from another person. Thus, we would see demoralization as an inevitable subjective emotional state occurring as a consequence of whatever "symptom" the patient seeks relief for. Frank and associates' phrase "combating destructive emotions" is really a metaphor for the rise in the patient's self-esteem when previously unacceptable emotions are expressed in the presence of an approving authority figure and found acceptable (2, p. 596). Thus, the mechanism of action of the therapy they describe is the identification with the therapist, who either does not have the same problems the patient does or reacts to them differently.

In chapter four the problems of long-term follow-up investigations of psychotherapy are discussed. The authors are not optimistic about psychotherapy's long-term effects, although it does serve to shorten the duration of a patient's distress and disability. One problem in evaluating such findings, as Frank himself points out, is the way in which concern about the long-term effects of psychotherapy may erroneously derive from the traditional medical model of "five-year cure" (p. 124). Thus, someone with gall bladder disease whose offending organ has been removed will presumably be symptom-free of that disease at the end of five years, but the kind of psychotherapy studied by Frank's group is primarily oriented toward the restoration of a symptom-and-shame-free equilibrium. They are not able to measure the extent to which their treatment provides the patient with the "tools" to maintain that equilibrium or even to raise it to a higher level of integration. This problem is also addressed in chapter one (p. 20): "Practitioners of long-term therapy such as psychoanalysis find that their patients take months or years to respond," in contrast to behavior therapists, hypnotherapists, and others. This seems to be a misunderstanding of the fact that acute symptom relief (whatever distress the patient seeks relief for now) usually occurs early during psychoanalysis, but that chronic symptoms, due to chronic intrapsychic conflict and arrested or deviant development, do not disappear easily. Thus, it may be that a form of psychotherapy specifically organized to treat chronic symptoms (e.g., psychoanalysis) might provide better data for long-term follow-ups. It should be noted that the indications and criteria for psychoanalysis have been spelled out recently in a form that also requires the psychoanalytic diagnostician to justify the choice of psychoanalysis over briefer, less expensive forms of psychotherapy (4).

In my opinion chapter three presents a series of distortions based on an initially accurate description of the ambiguous nature of psychoanalytic treatment as the patient experiences it. I think the authors draw unwarranted inferences from their description about the supposedly coercive characteristics of psychoanalysis, but the reader can readily correct these by referring to our work (2).

Chapter six describes the natural history of the Johns Hopkins Psychotherapy Research Unit—a group with a productive output of published papers and a record of effectively treating hundreds of outpatients over the 27-year span up to 1974. Anyone who has ever participated in clinical research with colleagues can appreciate the deeper implications of how the "place in the authorship credits shifted from straight alphabetizing, to coin-flipping, to fair and open discussions of who should occupy which place." The emergence of the "task-oriented social unit" that grew out of this method is alluded to, but unfortunately there is no full description of the process. From my own experience it is clear that the clinical researchers' relationship to their patient-subjects and the data generated by that relationship is importantly affected by the researchers' relations with each other, a fact not always appreciated in clinical research. Thus, the search for truth and mastery through understanding the data of human transactions is always extremely difficult. In response to the emotional pain of this task, there is a tendency among colleagues to regress in their relations with each other. Then the viability of the research group may depend on the members' ability to provide each other with emotional as well as cognitive support based on mutual awareness of and ability to deal openly with their various feelings. In this way the task-oriented, secondary-process quality of the work can be maintained.

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Brain Dysfunction in Aggressive Criminals, *by Russell R. Monroe. Lexington, Mass., Lexington Books (D.C. Heath and Co.), 1978, 211 pp., $17.95.*

Dr. Monroe has long been interested in the neurophysiological aspects of aggression. In a 1970 monograph, "Epi-

sodic Behavioral Disorders" (1), he proposed a classification system for aggressive and psychological variables. The current volume reports a prospective study to test his hypothesis on a group of individuals with known histories of uncontrolled aggression. Working with a prison population, a research team collected extensive neurological, psychological, demographic, and social data, which are presented at length. They further evaluated the effects of anticonvulsant medication over a short-range trial.

The researchers are to be commended for their scholarship and diligence in developing a classification system that supports Monroe's hypothesis. Of considerable interest is the high rate of brain dysfunction in the subject population (over 50%), even though dysfunction is defined rather broadly and includes many different diagnostic entities.

The significance of the work lies more in the implications of the specific findings than in the raw data. By attempting to establish a systematic approach to the study of aggression, the author demonstrates an unwillingness to accept a holistic approach to the subject. The study actually raises more questions than it answers, but it serves the function of stimulating further research. For instance, specific types of brain dysfunction need to be correlated to aggression, a better way of correcting dysfunctional elements needs to be developed, and dysfunction needs to be detected earlier to minimize psychosocial impairment.

For the casual reader, the book is often complex, technical, detailed, and not easily understood. Except for those who are actively engaged in similar research, it will probably have limited appeal. Nevertheless, the implications of this work are important, and I would encourage referral to several of Dr. Monroe's other publications on the subject.

REFERENCE

1. Monroe RR: Episodic Behavioral Disorders: A Psychodynamic and Neurophysiologic Analysis. Cambridge, Mass, Harvard University Press, 1970

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A Systems Approach to Alcohol Treatment, *by Frederick B. Glaser, M.D., Stephanie W. Greenberg, M.A., and Morris Barrett, M.P.H. Toronto, Ont., Canada, Addiction Research Foundation, 1978, 303 pp., $14.95 (paper).*

This presentation is a report of a multidisciplinary study of the resources available for the treatment of alcoholism in Pennsylvania in the calendar year 1973. The undertaking was motivated by a growing concern over the increasing complexities and cost of such services and an attempt to apply criteria by which the structure and functioning of such programs could be judged. On the basis of their study the authors conclude,

> As lengthy as it is . . . . this book really tells a rather simple tale. A group of interested people wanted to find out what was being done in terms of the treatment of alcoholism in Pennsylvania. There, they visited all the programs which were performing this service. They saw much that was good, but they found that there was no overall coherence or unified thrust to the effort. It was fragmented, entrepreneurial, redundant, and discontinuous.

The basis of this assessment and the approaches to their evaluation of the resources are documented in the presentation by an orderly detailed outline of the various treatment programs, including the criteria by which these have been judged, the involvement of other agencies in these treatment programs, and a plethora of figures, charts, tables, and appendixes. However, the final judgment of the authors may be questioned. The challenge arises from the uncertainties concerning the relationship of treatment facilities to outcome of treatment, in view of the limitations of our current knowledge of the nature of alcoholism and its cure.

Despite this criticism of the judgment concerning the merits of the different programs, the authors have done an excellent service in providing an extensive survey of alcoholism programs in a systematic manner. Their report is well organized, divided into the four sections of Background, Organization, Criteria and Objectives, and Conclusions. There is also a selected bibliography. The report includes historical notes that add to its depth. The authors are careful to point out that programs studied in 1973 may have changed. Nevertheless, the data base they have provided and carefully formulated offers an excellent guideline for those individuals concerned with assessing the treatment resources employed in the prevention, containment, and treatment of alcoholism.

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Drinking: Alcohol in American Society—Issues and Current Research, *edited by John A. Ewing, M.D., and Beatrice A. Rouse, M.Ed. Chicago, Ill., Nelson-Hall, 1978, 432 pp., $19.95; $8.95 (paper).*

This pleasant to read book is divided into five parts: Introduction and History, The Complications of Drinking, Psychosocial Aspects of Drinking, Social Policy and Drinking, and Summing Up. The chapters are written by 19 experts, many of whom are well known in the field of alcohology. The reader's interest is stimulated but not satisfied by this book. The discussion of medical complications of excessive drinking is inadequate. However, in the same section, the chapters by Ewing on social and psychiatric considerations of drinking are excellent, as is a provocative chapter titled "How Risky Is Drinking?"

An interesting study by Globetti on teen-age drinking in a rural high school in a dry Mississippi area is quite different from the usual urban studies. Globetti found less drinking, more guilt about it, and, for those who drank, more trouble related to drinking. Rouse and Ewing report on a 20-year follow-up study by Straus and Bacon of college drinkers that showed a drop in problem drinking by men from 42% to 17% but a rise in women from 11% to 12%.

A review of state alcohol control laws by Loeb leads him to the conclusion that wider legalization of liquor leads to greater per capita consumption, that government ownership of outlets reduces consumption, that more outlets mean more consumption, and that liquor-by-the-drink laws lead to greater consumption. The following chapter by Popham and associates makes the important argument that consumption is not distributed on a bimodal curve; i.e., alcoholics and other drinkers are not two groups: one is the continuation of the other. They believe this suggests a strategy that lower total consumption would reduce pathological drinking. One strategy they believe may have some merit is control of con-

sumption by pricing because data in Ontario and other places show that as the price of alcohol rises, consumption and death from cirrhosis drop.

These chapters are followed by a very insightful chapter by Room titled "Evaluating the Effect of Drinking Laws on Drinking." Room is not too impressed with Popham and associates' hypothesis, which, incidentally, has caused a great deal of public outcry from the "alcohol field" for fear it would suggest a return to a prohibitionist viewpoint. Room points out that, since 10% of the population drink 60% of the alcohol, changes in consumption rates and cirrhosis are naturally highly correlated because they are essentially the same thing. He discusses three theories about the possible effects of laws on drinking. The null hypothesis is that there is no relationship. The constant proportion theory is the one advanced by Popham and associates. The inoculation theory suggests we should teach healthy drinking from an early age. Room's chapter is the best of the book and is of great value to those involved with prevention strategies.

The book ends with a fine overview by Rouse and Ewing. An enticing little nugget they throw in is the finding that mice from a high-alcohol-consumption strain raised by mothers from a low-consumption strain drink half as much as their littermates raised by their natural mothers; mice from a low-consumption strain drink twice as much as their littermates raised by their natural mothers. The implications for genetic research of this observation are obvious.

This book has a number of good things in it for those trying to make sense out of this complex field; and this fact outweighs the weak chapters and the imbalance of space given to some subject matter.

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The Prevention of Sexual Disorders: Issues and Approaches, *edited by C. Brandon Qualls, John P. Wincze, and David H. Barlow. New York, N.Y., Plenum Press, 1978, 196 pp., $14.95.*

This book contains an interesting and provocative collection of papers that the editors have attempted to unify around a central theme of prevention. In the introductory chapter Dr. Qualls makes a careful effort to provide a background for thinking about these issues. He provides abundant evidence for the importance of approaching sexuality from a preventive perspective. In many of the chapters, however, prevention appears to be an afterthought, or even a contrivance. The problems of prevention are complex, and, as presented, they are often not well formulated or directly applicable to the specific chapter content.

The topics covered in this volume represent a wide range of interesting and important areas, including the role of cultural values, the child with cross-sex identity, sexual offenses, and the role of antiandrogens in the treatment of sex offenders. There is also some discussion of sex education, the role of pornography, and ethical issues. Some of the chapters are excellent and cover the material in a consolidated and thoughtful fashion; others sound more like unedited presentations from a symposium, thus detracting from the generally serious and well-modulated tone of the book. The final chapter, by R. Kolodney on ethical issues, provides a well-integrated view of the complexity of the problems faced in this field and would make an excellent stimulus for discussion. Although its flaws make the book less useful as a text, it can be recommended as a supplement to course material. The book is generally good reading and evokes critical thinking.

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Progress in Sexology: Selected Papers from the Proceedings of the 1976 International Congress of Sexology, *edited by Robert Gemme and Connie Christine Wheeler. New York, N.Y., Plenum Press, 1978, 593 pp., $32.50.*

This book consists of 113 papers representing the work of about 200 authors. It is divided into sections covering Sexual Differentiation and Dimorphism; Sexual Dysfunction; Sexual Response and Fantasy; Fertility, Infertility, Contraception; Anthropological and Sociological Studies; and Sex Education. It undoubtedly represents an enormous amount of work on the part of the editors. To get papers from so many authors and from such a diversity of points of view and countries of origin is a labor that cannot be underestimated. However, from the reader's point of view it is to little avail.

Most of the authors have inadequate space to develop their ideas in sufficient depth to make the book a good reference source. Some of the authors have been widely published and their work is therefore readily accessible. For instance, in his chapter on "Hormones and Sexual Behavior" Gunter Dorner cites himself 22 times. Other authors have little to say worth publishing. The fluctuations in style make the book a poor one to serve as an introduction to the general area of concern of "sexologists."

I think the editors deserve a great deal of praise for putting this book together and for the complicated obstacles they must have had to overcome to do this. However, I see no reason why anyone would want to buy or read the book.

HARVEY BLUESTONE, M.D.
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Human Growth and Development: Wolfson College Lectures 1978, *edited by Jerome S. Bruner and Alison Garton. New York, N.Y., Clarendon Press (Oxford University Press), 1978, 167 pp., $10.50; $5.50 (paper).*

This small volume is a collection of six lectures given at Wolfson College, University of Oxford, in 1976. Other than the common theme of early child development, there is no particular relationship among the subjects of the lectures. Therefore, I will mention the titles of the chapters to inform the reader of the varied contents. The first, "Social Development: A Biological Approach" by Prof. Robert Hinde, deals with issues that arose from the study of nonhuman primates and focuses primarily on mother-infant relationships. This is somewhat related to the second, by Dr. Michael Rutter, titled "Early Sources of Security and Competence." The latter addresses some of the same questions as they relate to human children and the social stresses that put them at risk. Rutter also discusses the ameliorating influences that allow children at risk to achieve emotional security and social competence.

The next two lectures have in common the topic of language acquisition. In "Learning How to Do Things with Words", Bruner stresses how the young child learns to use

language in the context of social living. He builds on the mother-child interaction from the earliest nonverbal communications, describing how higher-level verbal language develops out of the interaction. Perhaps because this was a lecture on words, I was struck with the author's extraordinarily long and complex sentences. The following chapter, "From Gesture to Word," is a discussion by Clark of language development as it relates to and depends on cognitive development. These two chapters are for students of linguistics.

In the fifth lecture, "New Currents in Genetic Epistemology and Developmental Psychology," Inhelder reviews some of Piaget's theories and discusses further work on cognitive development that is being done by the Geneva group. The final chapter, "Nursery Needs and Choices," is a more pragmatic discussion. Tizard comments on the history of nursery services, parental desires, controversies, and economic aspects of nursery services, as well as the well-being of children and of parents. He offers some practical recommendations on how services could be organized. This paper might interest a different group of readers than would the preceding papers.

Each of these lectures will appeal to a small group of highly specialized professionals; not necessarily the same group will be motivated to study each one. Those with a particular interest in early child development will find some common interests in all of them. Although it is a carefully written and informative book, it will not have much general appeal to the readership of the *Journal*.

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Marriage and Marital Therapy: Psychoanalytic, Behavioral and Systems Theory Perspectives, *edited by Thomas J. Paolino, Jr., M.D., and Barbara S. McCrady, Ph.D. New York, N.Y., Brunner/Mazel, 1978, 566 pp., $25.00.*

This book not only illuminates the field of marital therapy as it is currently practiced but is a valuable contribution to the field of psychotherapy in general. Marital therapy does not have a single theory and practice that is idiosyncratic for marital therapists. Therapists enter the marital therapy field after having been trained in various disciplines and in other forms of psychotherapy—usually individual therapy. They bring with them the theory and practice in which they have been trained and apply it to the treatment of marital couples. Thus, the current three dominant theoretical perspectives of psychotherapy in general (psychoanalysis, behavioral therapy, and systems theory) have become the dominant theoretical perspectives in the area of marital therapy. Clinical practice of marital therapy clarifies, refines, and develops these theoretical perspectives so that they become more meaningful to practitioners of individual, group, and family therapy.

This book serves the valuable function of clearly picturing the current status of these three very different theoretical perspectives and their applications in marital therapy. The editors have done a great service in gathering together outstanding theorists and clinicians to present their views in original, updated contributions. The effective clarifications of the theorists and clinicians help clinicians gather their data and plan their interventions in a scientifically effective manner. Thus, a chapter on a particular theoretical approach is followed by a chapter on its clinical application. It can be observed in the theoretical chapters that despite the author's personal preference, there is no one agreed on psychoanalytic, behavioral, or systems theory. There are differences in theory in each of the three theoretical approaches, and an attempt to integrate all three into one theory of human functioning lies far in the future.

One of the principal goals of the editors was to provide the reader with a choice of models that can be used alone or in combination with other perspectives so that the student, researcher, or clinician can better organize his or her observations. Thus, the editors espouse an eclectic approach in the finest sense of the term. Indeed, this is what happens in practice as one can observe in reading the clinical chapters. What is done in treatment often violates the attempted purity of the preceding theoretical chapter. This is understandable, since clinicians, who offer service and who are primarily caretakers, do what will help the patient improve even if it violates the purity of a particular theory.

The book consists of nine chapters by leaders in the areas in which they are writing. It opens with a chapter by James and Janice Prochaska, which serves as a backdrop for the theorists and therapists who follow. William Meissner then offers the first theoretical chapter on the conceptualization of marriage and family dynamics from a psychoanalytic perspective.

In the third chapter Carol Nadelson shows how a psychoanalytic marital therapist can remain sensitive to important transference issues without being unduly restricted in the therapeutic modalities that can be applied. Nadelson is to be applauded in that she allows the couples' particular problems and needs to dictate the form that therapy will take rather than having rigid theories restrict its format.

In chapter four Robert Weiss presents the conceptualization of marriage from a behavioral perspective and shows how the union of marriage and behaviorism is not as unlikely as it has been considered. The clinical chapter that follows Weiss's theoretical one is by K. Daniel O'Leary and Hillary Turkewitz, who focus on the treatment of marital disorders from a behavioral perspective. They report their recent research on comparing communications training and behavior change contracts to determine which approach is most effective in helping marital couples.

The systems theory approach is covered in a chapter by Peter Steinglass on the conceptualization of marriage from a systems theory perspective and a chapter by Carlos Sluzki on a treatment approach from a systems theory perspective. Recognition should be given to the fact that systems theory and therapy are the most challenging contributions that marital and family therapy have made to the mental health disciplines.

Neil S. Jacobson presents a review of the research of the effectiveness of marital therapy. This is a very important chapter because marital therapy will remain in a state of confusion and disorganization until valid research studies prove the effectiveness of the many techniques in the field.

The final chapter, "Contemporary Marital Therapies: A Critique and Comparative Analysis of Psychoanalytic, Behavioral and Systems Theory Approaches" by Alan Gurman, is worth the price of the book. He attempts a systematic comparative analysis of the three theoretical perspectives. Integration of any one of these three fields has not yet been achieved, so that integration of all three is in our present state of knowledge an insurmountable task, but we should continue to make such valiant efforts toward a future integrative theory of psychotherapy in general.

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This Month's Reviewers

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Books Received

This listing acknowledges the receipt of recent books. Books of particular interest to the readers of this journal will be reviewed as space permits, and copies of the reviews will be sent to the publishers. Books cannot be returned to the publishers.

Psychoanalysis of Drug Dependence: The Understanding and Treatment of a Particular Form of Pathological Narcissism, *by J. Winstead Adams, M.S.W., Ph.D. New York, N.Y., Grune & Stratton, 1978, 315 pp., $23.50.*

Rorschach's Test II. Gradients in Mental Disorder, 3rd ed. of Rorschach's Test: A Variety of Personality Pictures, Vol. II, *by Samuel J. Beck, Ph.D., and Anne G. Beck, M.A. New York, N.Y., Grune & Stratton, 1978, 394 pp., $18.50.*

Cognitive Growth and Development: Essays in Memory of Herbert G. Birch, *edited by Morton Bortner, Ph.D. New York, N.Y., Brunner/Mazel, 1979, 333 pp., $22.50.*

Dual-Career Couples, *edited by Jeff B. Bryson, Ph.D., and Rebecca Bryson, Ph.D. New York, N.Y., Human Sciences Press, 1978, 120 pp., $9.95.*

Training in Ambiguity: Learning Through Doing in a Mental Hospital, *by Rose Laub Coser. New York, N.Y., Free Press (Macmillan Publishing Co.), 1979, 215 pp., $13.95.*

Human Autoerotic Practices, *edited by Manfred F. DeMartino, M.A. New York, N.Y., Human Sciences Press, 1979, 366 pp., $16.95.*

Crazy for God, *by Christopher Edwards. Englewood Cliffs, N.J., Prentice-Hall, 1979, 233 pp., $8.95.*

The Wildcat Experiment: An Early Test of Supported Work in Drug Abuse Rehabilitation, *by Lucy N. Friedman. Rockville, Md., National Institute on Drug Abuse, 1978, 147 pp., no price listed (paper).*

Death and Dying: Challenge and Change, *edited by Robert Fulton, Eric Markusen, Greg Owen, and Jane L. Scheiber. Reading, Mass., Addison-Wesley Publishing Co., 1978, 475 pp., no price listed (paper).*

Treatment of Primitive Mental States, *by Peter Giovacchini, M.D. New York, N.Y., Jason Aronson, 1979, 529 pp. $25.00.*

Addiction Research and Treatment: Converging Trends, *edited by Edward L. Gottheil, A. Thomas McLellan, Keith A. Druley, and Arthur I. Alterman. New York, N.Y., Pergamon Press, 1979, 146 pp., $15.00.*

Alcoholism Rehabilitation: Methods and Experiences of Private Rehabilitative Centers, *edited by Vincent Groupé, Ph.D. New Brunswick, N.J., Rutgers Center of Alcohol Studies, 1978, 130 pp., $6.00 (paper).*

Emotions in Personality and Psychopathology, *edited by Carroll E. Izard. New York, N.Y., Plenum Press, 1979, 590 pp., $29.50.*

Clinician's Handbook of Childhood Psychopathology, *edited by Martin M. Josephson, M.D., and Robert T. Porter, M.D. New York, N.Y., Jason Aronson, 1979, 476 pp., $25.00.*

Maturation of the Therapeutic Community: An Organic Approach to Health and Mental Health, *by Maxwell Jones, M.D. New York, N.Y., Jason Aronson, 1979, 529 pp., pp., $5.95 (paper).*

Stress and the American Woman, *by Nora Scott Kinzer. Garden City, N.Y., Anchor Press/Doubleday, 1978, 262 pp., $8.95.*

Family Therapy and Evaluation Through Art, *by Hanna Yaxa Kwiatkowska, A.T.R. Springfield, Ill., Charles C Thomas, 1978, 270 pp., $21.75.*

A Source Document in Schizophrenia: Whoever Had Most Fish Would Be Lord and Master, *by Julius Laffal. Hope Valley, R.I., Gallery Press, 1979, 367 pp., $19.95.*

Six Lives/Six Deaths: Portraits from Modern Japan, *by Robert Jay Lifton, Shuichi Kato, and Michael R. Reich. New Haven, Conn., Yale University Press, 1979, 296 pp., $16.95.*

The Heroin Stimulus: Implications for a Theory of Addiction, *by Roger E. Meyer, M.D., and Steven M. Mirin, M.D. New York, N.Y., Plenum Press, 1979, 247 pp., $22.50.*

Ego Impaired Children Grow Up: Post Discharge Adjustment of Children in Residential Treatment, *by John B. Mordock, Ph.D. Rhinebeck, N.Y., Astor Home for Children, 1978, 244 pp., $5.00 (paper).*

Culture and Curing: Anthropological Perspectives on Traditional Medical Beliefs and Practices, *edited by Peter Morley and Roy Wallis. Pittsburgh, Pa., University of Pittsburgh Press, 1978, 190 pp., $14.95.*

Psychosexual Imperatives: Their Role in Identity Formation, *edited by Marie Coleman Nelson and Jean Ikenberry. New York, N.Y., Human Sciences Press, 1979, 388 pp., $19.95.*

The Search for the Self: Selected Writings of Heinz Kohut: 1950–1978, Vols. 1,2, *edited by Paul H. Ornstein. New York, N.Y., International Universities Press, 1978, 954 pp., $35.00.*

OFFICIAL ACTIONS

Position Statement: A Call to Action for the Chronic Mental Patient

This statement was approved by the Assembly at its October 1978 meeting and by the Board of Trustees at its December 1978 meeting upon recommendation of the Ad Hoc Committee on the Chronic Mental Patient. It was prepared by the Ad Hoc Committee on the Chronic Mental Patient.[1]

THERE IS no more urgent concern than the needs of the chronic mentally ill who suffer from severe, persistent, or recurrent mental illnesses with residual social and vocational disabilities. As a result of the deinstitutionalization programs of the past decade and the continuing growth of high-risk populations that generate chronic illness, the problems associated with the care of these patients constitute a national crisis. The Conference on the Chronic Mental Patient, sponsored by the American Psychiatric Association in collaboration with the President's Commission on Mental Health, was held in Washington, D.C., January 11–14, 1978, and addressed the striking inadequacy of care, treatment, and rehabilitation of this group, estimated to number over one million Americans.

The chronic mentally ill are of all ages, including children, and have a variety of diagnoses.[2] They may reside in community or institutional settings. Such patients must be distinguished from those individuals who may be receiving various forms of psychotherapy for mental disorders without attendant long-term disabilities. The chronically ill have a host of special and unique problems including extreme dependency needs, high vulnerability to stress, and difficulty coping with the demands of everyday living, resulting in difficulty securing adequate income and housing and holding down a job.

The term "chronic mentally ill patient" stigmatizes persons so designated and obscures their diversity and potential for improvement. It is not a desirable appellation because of its implication of hopelessness and progressive deterioration but has been used in this report because of its historical and current use in the literature and by the profession and because of its descriptive clarity. While these people have a chronic illness that requires medical and psychiatric attention over a long period of time and are, therefore, appropriately called patients, it is equally important to recognize them as persons with continuing disability. This disability concept carries the positive implication that a psychosocial rehabilitation approach should complement any treatment provided.

Successful programs for helping the chronically ill patient offer a continuum of residential and nonresidential services to ensure that care is tailored to meet individual needs and to provide easy access and reentry to services and responsiveness to crises. Such programs use the skills of persons with an interest in and knowledge about chronic mentally ill patients. They provide thorough monitoring and balance active outreach with the encouragement of self-sufficiency and independence. They also encourage interagency cooperation and referral and serve as patient advocates. They have effective vertical (e.g., up higher governmental levels) and horizontal (e.g., across to other community agencies) structures. Sensitivity to incremental degrees of progress, economic stability, accountability, and responsibility are also essential features of effective programs.

Obstacles to effective delivery of services to the chronically mentally ill are monumental. They include the attitudes of patients, families, communities, community leaders, and professionals; the lack of an integrated community support system; fragmentation of federal programs; the absence of unified funding; the failure to designate responsibility for treatment, care, and rehabilitation of the chronically ill patient; widespread discrimination in employment, ambulatory care funding, zoning, etc.; and conflicting and/or limiting federal and state regulations.

POLICY STATEMENT

To address the needs of the chronic mentally ill, a national public policy must be adopted. This policy must include the following:

1. *Public sensitivity and financial commitment to a system of opportunities and services.* A systematic approach to caring for the chronic mentally ill must include at a minimum active case-finding and outreach; 24-hour emergency and crisis stabilization services; functional evaluation; medical and psychiatric care; training in skills of everyday living; so-

[1]The Ad Hoc Committee on the Chronic Mental Patient included John A. Talbott, M.D., chairperson, James T. Barter, M.D., Maurice Laufer, M.D. (deceased), W. Walter Menninger, M.D., Arthur T. Meyerson, M.D., Mildred Mitchell-Bateman, M.D., Lucy Ozarin, M.D., John P. Spiegel, M.D., and Harold Visotsky; Dr. Richard Duke, Dr. Z. Erik Farag, Dr. Henry Foley, Dr. Eli Ginzberg, Dr. Sam Keith, Dr. David Mechanic, Ms. Judith Turner, and Ms. Jane Yohalem were consultants, and Donald Hammersley, M.D., and Sam Muszynski, M.S.W., represented APA staff.

[2]In this report, the population we are concerned with are primarily those suffering from major psychoses, e.g., chronic schizophrenia, chronic recurrent affective disorders, etc. For reasons of simplicity and expeditiousness, the term, as used in this report, does not include persons suffering from alcoholism or drug abuse or the mentally retarded.

cialization; an array of specialized living arrangements; subsistence, prevocational evaluation, clinical work adjustment programs, and subsidized transitional and permanent full- and part-time work opportunities that are meaningful and feasible; and assistance to families—all of which are monitored and managed in accordance with individual needs. In addition, provision should be made for indirect services such as community consultation, community education, community organization, and interagency collaboration. The system should recognize that some patients, while chronically disabled, are only partially disabled and can function in supportive situations. The system should be designed to promote growth and sustain functioning to the maximum degree feasible for each individual and should be directed toward patients who voluntarily request assistance.

2. *Designation of clear responsibility for providing services at appropriate levels of government.* The assurance of care, treatment, and rehabilitation of the chronically mentally ill is a national public health responsibility. Thus, every level of government bears some responsibility to assure adequate services to this population.

The federal government should have the responsibility for defining eligibility; identifying and assuring levels of benefits; funding services under national health insurance or categorical programs; establishing regulations ensuring access to services, quality care, and cost effectiveness; and monitoring program implementation. The state government should assume responsibility for statewide planning, approval of local plans consistent with that statewide plan, supplementary funding and benefits, standards, and program monitoring within the state. At the local level, appropriate organizational entities should be responsible for local planning and integration of services for the chronic mental patient, administering and/or managing those services either directly or by contract, and evaluating programs.

3. *Full civil rights for the chronically mentally ill.* There should be no discrimination against the mentally ill. The right to adequate treatment in the community and to confidentiality must be guaranteed. Chronic mentally ill patients should have full access to medical, legal, educational, vocational, occupational, and housing services and opportunities. These services and opportunities to the mentally ill should be provided in settings that allow the maximum independence consistent with the patient's needs.

4. *Reform of funding mechanisms.* These should be designed to remove incentives toward more restrictive forms of care, to remove discrimination against the chronically mentally ill, and to assure their access to health, human service, rehabilitation, and housing programs. Funding should also increase the availability of vitally needed services such as active outreach, crisis stabilization in the normal environment, diminution of symptomatic behavior, remediation of functional skills, meaningful daytime activities, long-term supportive work opportunities, and case management.

5. *The same policy and implementation requirements* for classes of service, levels of care, and accountability that are required of public and private, state and local health systems and facilities should be applied to programs run directly by the federal government (i.e., the Veterans Administration and Public Health Service systems).

6. *Social and cultural factors.* There should be an equitable allocation of mental health resources in the community to citizens from all social, economic, and racial (ethnic) backgrounds and population densities. All services delivered must be adapted to meet the cultural values and perceptions or needs of various ethnic, minority, and subcultural groups.

7. *Utilization of families.* Wherever possible, patients' families should be involved in their treatment and care rather than depending on more expensive and less caring substitutes. While women in the home have traditionally assumed the caretaking responsibility, changing roles suggest that their presence can no longer be taken for granted; there must be adequate financial, social, and mental health supports available when families assume such responsibility.

RECOMMENDATIONS

Psychiatry's Role in the Care of Chronic Mentally Ill Patients

Since care of the chronic mentally ill patient is a major health concern to the public, it is incumbent on psychiatrists and other physicians to take an active role in attending to the needs of this population. Even though psychosocial problems may predominate, the medical and psychiatric needs of the chronic mentally ill require vigilant monitoring. In addition, psychiatrists have a responsibility in the development of comprehensive services for the chronic mentally ill and should be involved at all levels of program planning, public education, training, and research related to preventive care and rehabilitative services.

The American Psychiatric Association should take the lead in undertaking programs to elevate the prestige and value of work with chronic mentally ill patients. Portions of the scientific programs of annual meetings, regional meetings, and district branch scientific meetings should be devoted to this population. Research should be sponsored and groups working with this population should be encouraged. APA should also take steps to encourage psychiatrists and others to monitor the quality of care administered to the chronic mentally ill patient population by their peers.

The prestige and status of psychiatrists who work in programs with chronic mentally ill patients will be enhanced by affiliation with medical school departments of psychiatry; teaching and/or clinical assignments at medical schools by psychiatrists who work with the chronic mentally ill; clinical and supervisory assignments by faculty of academic departments to programs for chronic mentally ill patients; continuing medical education programs held at the site of programs for chronic patients by medical schools and APA district branches; academic appointments for psychiatrists working in programs for the chronic mentally ill patient; and a referral system involving private psychiatrists, which will ensure continuity of care.

Community Education

1. All involved consumer, professional, paraprofessional, and governmental bodies should mount a coordinated education and lobbying program, using professional communication expertise (lobbying, marketing, community education) to inform the public about the chronic mentally ill and how to meet their needs.

2. Community education must be oriented toward increasing the visibility and status of programs directed to chronic mental patients.

3. A major effort should be undertaken to develop a constituency for the chronic mentally ill patient population.

4. District branches should make an effort to include the subject of care and treatment of the chronic mentally ill in both their community and scientific programs.

Research

There must be a continuing emphasis on research in the area of chronic mental illness, including epidemiology, etiology, therapy, outcome, and effective service delivery. In addition, new efforts should be undertaken to clarify the conditions under which family care is helpful or harmful; to ascertain the rate and nature of problems faced by the (current) deinstitutionalized population as compared with the (formerly) institutionalized chronically ill population; to study the criteria for deinstitutionalization and for various types of group maintenance, including continued hospitalization; to define and refine the tasks, skills, and process of case management; and to reexamine the issues relating to confidentiality. Uniform data collection regarding the size, composition, and service needs of the chronic population at the local level is needed to help identify special problems and needs and to improve program planning and monitoring. Another fruitful area for research is the prevention of chronicity, especially in children and adolescents as well as in the aging.

Training

1. Training programs should be expanded or established for persons, including family members, in the skills appropriate to the needs of the chronic mentally ill.
2. Current professional training programs, including psychiatric residency programs, should be modified and reoriented toward an interdisciplinary focus to enhance the capacity of professionals to treat and care for the chronic mentally ill patient. Persons who have been working in chronic care settings should be retrained to be able to function within a community/rehabilitation model, nursing homes, and geriatric facilities, as well as in programs that help patients in strength assessment and the acquisition of the skills of everyday living.
3. Funding is required to implement the above retraining provisions and provide incentives for state governments to carry out this statutory responsibility where necessary. Consideration should be given to mechanisms whereby psychiatric residents could "pay back" the money spent on their training by serving in shortage areas (e.g., state hospitals).
4. Psychiatric residency training programs should be encouraged to include training for more chronic mentally ill patients than the 10 specified in the residency training guidelines, as well as to include training in administration and planning. Consideration should also be given to a new subspecialty—rehabilitation psychiatry.
5. A program for volunteer case aides should be established to promote local volunteer mobilization around the chronic mentally ill patient.
6. Training programs should be established for medical students and primary care physicians, especially those working in emergency settings, to focus on the special treatment needs of patients with chronic mental illness, since these patients have a higher incidence of medical illness and are often resistant to medical care due to their mental disability. Such programs should include experience with ambulatory chronically mentally ill patients, with particular emphasis on appropriate and inappropriate psychopharmacological medications, the concomitant social and vocational disabilities, and the full array of ambulatory treatment programs necessary for chronically ill patients.
7. The establishment of guidelines for training and career development of psychiatrists involved with program planning for and treatment of chronic mentally ill patients should be encouraged. A study of psychiatrists currently working in this area may offer data relevant to the development of successful educational experiences and career pathways.

Continuity and Provision of Services

1. Barriers should be removed to assure chronic mentally ill patients access to a full range of medical, psychiatric, rehabilitative, income maintenance, social, employment, and related opportunities and services appropriate to their needs in the least restrictive setting.
2. The system of care should be continuous between institutions and local programs, and there should be well developed systems for interservice program referral. It is necessary to establish and support case management to enable the chronically ill patient to use and benefit from community resources and programs. Such management should be based on a comprehensive treatment and management plan; the patient, and if possible the family, should be involved in the planning and delineation of responsibilities. Before extensive programs of case management are undertaken, however, there is a need to define the role, responsibility, and function of care, fixing of responsibility, and linking of hospitals with community services. Interagency linkage should be encouraged through inducements and sanctions written into legislation, regulations, and procedures. Adequate resources should be provided for case management functions, and funding should allow for an adequate period of time for training staff and establishing information systems, etc., to phase in such a system.

Financial Needs

The financial recommendations that follow include consideration of cost savings resulting from the shift of chronically ill patients from higher cost institutional programs to lower cost community alternatives. Attention to the ways in which financing mechanisms perpetuate higher cost care can prevent escalating and outrageous costs for programs serving chronic mentally ill patients. Some evidence exists that high quality integrated programs based on a least restrictive but full service model are no more costly than state hospital incarceration.

1. Programmatic funds should, as a long-term goal, flow from the federal to the state level and be earmarked for the chronic mentally ill patient where possible. This includes monies currently administered in the Departments of Health, Education, and Welfare, Housing and Urban Development, Labor, etc. Thus, a specified share of welfare, housing, rehabilitation, health, and mental health dollars would be directed to this population either on a capitation or index of need basis. These monies would be allocated to local communities or agencies only if programs were accountable in relation to the chronic mentally ill patients' needs for service.
2. On the federal level, structures should be created to provide oversight, both by Congress and the executive branch, of legislation and regulations affecting the needs of chronic mentally ill patients. A comparable structure should be established on the state and local levels.
3. The Department of Health, Education, and Welfare should perform a national survey of Medicare and Medicaid eligibility requirements, benefit services, and reimbursement schedules. This survey would elucidate current inequities and help establish national parity.
4. Chronic mentally ill patients are entitled to full participation in the health care system. Medicare, Medicaid, and

future national health insurance should not single out the chronic mentally ill as a class or discriminate against them in any way. This is especially important regarding private psychiatric care, which is often less costly than that provided by institutions.

5. Medicare, Medicaid, and future national health insurance benefits should include a full range of inpatient, day treatment, and outpatient services encompassing periodic medical and psychological evaluation and treatment, resocialization, and rehabilitation. In all future funding, there should be differentiation between health services (e.g., evaluation, diagnosis, medical, and psychiatric treatment) and social and supportive services (e.g., escort services, housing, etc.).

6. Any future national health insurance should also include cost effective but positive financial incentives to encourage professionals to care for the chronic mentally ill patient, so that the existing disincentives to providing long-term care are reversed.

7. Financing of psychiatric and human services should be modified to remove fiscal disincentives (e.g., Medicare restrictions on ambulatory care) and unnecessarily restrictive or debilitating settings or forms of care, such as inpatient hospitals or nursing homes.

8. All federally funded comprehensive community mental health centers should be required to provide comprehensive services to the chronically ill mental patient as one of the mandated essential services.

9. A federal technical assistance program, along the model of the agricultural extension program, should be developed to help localities develop appropriate programs for chronic mentally ill patients.

10. Funding mechanisms should encourage states and localities to move individuals out of the human services system into mainstream community life through rehabilitative programs.

11. Priority should be given to proposed systems ensuring that money follows chronic mentally ill patients, either through a voucher system that would enable patients to buy any or all necessary services or by some other mechanism.

12. There is agreement on the following points regarding recommendation 13 in the Preliminary Report of the President's Commission on Mental Health (advocating establishment of a class of intermediate care facilities for mental patients under Medicaid): there is a current *shortage of federal and state funding* for community living arrangements for the mentally disabled; there is a need for a *continuum* of types of living arrangements, offering varying degrees of supervision and support; funding policies should promote a planned, accountable *system* of living arrangements within each state and local planning area; there is a critical need for improved methods to *link* special living arrangements with nonresidential treatment, rehabilitation, and support services; and it is vital to recognize that appropriate living arrangements are *necessary* but not sufficient in meeting the needs of the mentally disabled.

Based on these areas of agreement, it is recommended that additional resources for community living arrangements for the mentally disabled be made available through earmarking federal and state housing and social service funds.

With respect to the advisability of specific federal funds for intermediate care facilities for the mentally ill, while we support the intent of the proposal, we believe that no such facilities should be established, because specific federal funding for a particular class of facilities will result in overdevelopment of one type of residential arrangement at the expense of other types, it will detract from the availability of adequate resources for essential nonresidential rehabilitation and support services, it will interfere with developing flexible local systems based on community needs, and it will be more expensive than a policy that would limit use of medical funds to more narrowly defined medical needs and would support housing arrangements from nonmedical resources.

13. Provisions of Supplemental Security Income legislation and procedures should be modified to replace the current disincentives against patients' returning to productive employment with positive incentives; e.g., allowances should be made for patients' rehabilitation potential.

Administrative Issues

As a long-term goal, the federal government should take responsibility for leadership and advocacy of care for the chronic mentally ill patient; establish policy and ensure consistency in all relevant agency policies; set basic programmatic guidelines and regulations; establish minimal care and accountability standards; issue guidelines setting forth broad parameters for the ulitization of funds; provide strong incentives and bonuses for care of the chronic mentally ill patient in the community; stimulate collaboration among agencies involved in policy planning and program implementation; develop technical assistance and disseminate information concerning the chronic mentally ill patient; develop criteria for determination of local government's ability to assume planning, management, and service operation responsibility and to establish programs for those localities without sufficient capacity to provide training, assistance, and funding to attain an acceptable level; and provide assurances that any jurisdictional level that has oversight/coordination responsibilities (and has reduced or eliminated its service operations) maintains the necessary staff expertise to carry out its responsibilities in the areas of planning, licensure, etc.

State governments should carry out the leadership, patient advocacy, and planning functions on a statewide basis for distribution of federal monies; supplement federal funds with state monies; and designate local authorities to have programmatic responsibility. They should also establish and provide assurances that coordination mechanisms are in place and operating to ensure chronically ill patients' access to appropriate support programs, develop appropriate standards for programs on a state level, establish regulatory/guideline appeals mechanisms, provide services for specific populations when it is not feasible for any other entity to assume this function, and monitor local service operations.

Local authorities should designate specific local entities to perform program activities; coordinate the planning and provision of services; hold local entities accountable for these services; establish entitlement for chronic mentally ill patients to relevant support systems; ensure nondiscrimination; ensure maximum consumer (public and nonprofit) participation; and provide local entities with formal authority over support-system resources such as welfare, rehabilitation, etc., applicable to this population.

Local entities should be the final common pathway for program funding directed toward providing the chronic mentally ill with a holistic integrated program based on the least restrictive, rehabilitative model with appropriate medical-psychiatric input. Local entities eligible for designation as the authority responsible for chronic mentally ill patients should include both public and nonprofit facilities.

The immediate goals should include the following:

1. Oversight mechanisms should be established at the fed-

eral level; examples are a select committee in Congress comparable to the Select Committee on Aging, and an executive branch equivalent, which would oversee federal legislation and regulations applying to chronic mentally ill patients.

2. Each state mental health authority should designate a single person/office to assume primary responsibility for acting in behalf of, planning, and supporting services for chronic mentally ill patients. Such person/office should develop knowledge about all potential federal and state programs that may provide funding and/or services for chronic mentally ill patients and transmit that knowledge to appropriate mental health service providers. Further, that person/office should review legislation, appropriations, and rules and regulations and should serve as an advocate for policies that will enhance services for chronic mentally ill patients.

3. Each state should produce a plan which guarantees that the needs of the chronically ill population will be provided for. Such plans should fix responsibility within each local planning area with a single community agency that assumes the role of convener, catalyst, coordinator, community organizer, and advocate for meeting the full range of needs of chronic mentally ill patients. The type of agency that can best assume this role may vary from community to community, depending on what is available. In all cases, it is essential that such responsibility be clearly assigned and recognized.

4. Clinical integration should be done by the local area health or mental health planning body independent of any care delivery system of its own that might represent a competitive interest. This also applies on the state level.

5. Accountability is a critical element to assure that the services promised are actually delivered. Evaluation of these services must be consistent and equally applicable to all service providers. Efforts should be made to limit the costs and bureaucracy of the evaluation process—possibly by utilizing the Health Systems Agency structure or an equivalent—and to encourage a positive attitude in enforcing accountability; i.e., evaluators should be oriented toward helping recipients satisfy not only regulatory requirements but also toward improving services, in addition to identifying service deficiencies and threatening penalties. Affirmative approaches to quality of life and social and vocational disabilities should be a primary objective.

6. Rather than building a whole new network of programs and services, the emphasis should be on the development of staff and facilities for the chronic mentally ill patient, making use of existing functions and resources, including the family whenever possible, and restructuring and reordering such programs in ways that better meet the needs of the chronic mentally ill patient. The development of new approaches and capacity should be encouraged at the local level, and technical assistance should be provided to enable this.

7. States should be discouraged from developing new state-operated facilities for chronic mentally ill patients and should phase down present facilities over time. While states must assure an adequate supply of facilities to meet the needs of chronic mentally ill patients, in order to encourage the local development of programs these facilities should not be state operated.

8. The federal government should eliminate any state or regional options in the utilization of essential federal funds, e.g., Section 8 of the Housing Law of 1975, and establish mechanisms whereby states and localities may appeal restrictive regulations.

Civil Rights

There should be federal legislation or regulations to accomplish the following:

1. *Prohibit discrimination against chronic mentally ill patients in vocational rehabilitation, employment, and education.* Specifically, Title VII of the Civil Rights Act of 1964 should be amended to prohibit "unjustified" discrimination in employment on the basis of handicap. In addition, there should be vigorous enforcement of Sections 503 and 504 of the Rehabilitation Act of 1973, federal legislation to encourage the hiring of the mentally handicapped (either through bonuses or tax incentives), and assurance that the severely and chronically mentally disabled are served by vocational rehabilitation agencies. Also, the concept of equal job opportunity should be applied to women in both institutional and community rehabilitative and vocational programs.

2. *Prohibit discrimination against chronic mentally ill patients in housing.* Specifically, Title VIII, Fair Housing, of the Civil Rights Act of 1968, should be amended to prohibit discrimination in housing on the basis of handicap. The Department of Housing and Urban Development should promulgate regulations to encourage states and localities to allocate additional Section 196 funds to develop more group care facilities, and to make additional Section 8 rental assistance funds available to mentally disabled persons living in group homes.

Federal legislation should be enacted encouraging the private market to provide housing to the mentally handicapped and conditioning receipt of federal revenue sharing or other funds to states having a plan for the development of community care and community residencies for the mentally disabled.

3. *Endorse the right to adequate treatment for both voluntary and involuntary patients in the hospital and the community, in the least restrictive setting consistent with individual treatment needs.* This includes the establishment of a mechanism whereby the patient may object to any aspect of his/her treatment plan, including transfer to another facility or to the community.

4. *Protect confidentiality, while allowing access to relevant information for legitimate treatment, planning, and research needs.* Centralized records should contain the minimum amount of information needed to meet the patient's future treatment needs, with access to records limited to a "need to know" basis, and patients should have the right to consent to the release of particular items of information from their records for time-limited periods, revocable by the patient. Stringent protection should govern access to treatment records, and stringent criminal penalties should be mandated for misuse of information included in records. Insurance claims (private and governmental) should be reviewed by a claims review system in which physicians would review patients' records without their names attached.

5. *Develop and fund an advocacy system independent of service providers to help ensure the implementation of patients' rights.* This system should either be part of the protection and advocacy system created by the Developmental Disabilities Act or should be modeled on that system.

6. *Prohibit zoning discrimination against the mentally ill by requiring that receipt of revenue sharing, housing, and other federal funds be predicated on the absence of exclusionary zoning laws or regulations in an area.*

7. *Enact a "Bill of Rights" for mentally disabled persons residing in the community.*

Position Statement on Active Treatment

This statement was approved by the Assembly at its meeting in October 1978 and by the Board of Trustees at its meeting in December 1978. The statement was originated by the Council on Mental Health Services[1] and revised by a special Assembly task force.[2]

PSYCHIATRIC TREATMENT is a planned effort on behalf of persons defined either by themselves or by their community as mentally ill or emotionally disturbed and in need of treatment. The person directing it must be qualified by specialized education and training to evaluate and understand the totality of the biological, psychological, and social factors that play a part in such an illness. Treatment is provided through medical procedures designed to benefit the ill person.

1. Treatment may begin prior to the establishment of a final diagnosis. The process of evaluation is an act of treatment.

2. The standards used by a community to judge behavior may not always be in agreement with the standards leading to a diagnosis used by a psychiatrist to judge behavior. For example, some people judged by community standards to be "bad" rather than ill may suffer from a diagnosable mental illness. On the other hand, some persons whose behavior is identified as aberrant by a given community may be perceived by the psychiatrist as following an alternative lifestyle and not as suffering from an illness.

3. As in physical illness, an individual's subjective distress may in itself be sufficient justification for treatment.

4. Psychiatric disorders result from the complex interaction of physical, psychological, and social factors and treatment may be directed toward any or all three of these areas.

5. Treatment may include measures to maintain current functioning and prevent further deterioration as well as measures designed to improve or eliminate dysfunction.

6. A variety of professional disciplines may be involved in a treatment program. The extent and kind of participation of any practitioner in a specific treatment program should be determined by the person primarily responsible for providing treatment. The professional qualifications and ethics of the various disciplines are defined by each professional group within society's sanctions. No practitioner should be required to participate in a manner contrary to the ethic of his or her discipline.

7. A formal or informal treatment plan is an integral part of treatment. The plan should include the goals of treatment and problems that may be anticipated and should be revised when appropriate and indicated. Psychiatric treatment should be based on principles that can be explained and communicated during review by one's peers.

8. Providing a human environment for the care of persons in need is not equivalent to providing treatment. However, when the environment is carefully organized to respond in a therapeutic manner to patients' needs and behavior and is staffed and supervised by qualified members of appropriate professional disciplines, it is a form of treatment. Treatment of that kind is usually referred to as a therapeutic environment or milieu therapy.

9. Psychiatric treatment is the sum of the activities of a psychiatrically qualified physician in meeting the therapeutic needs of a patient, a family, or a (community) group. This may include the supervision of others who are providing treatment and for whose activities the psychiatrist accepts professional and legal responsibility.

[1]The Council on Mental Health Services included J.M. Stubblebine, M.D., chairperson, Mildred Mitchell-Bateman, M.D., Israel Zwerling, M.D., Hayden H. Donahue, M.D., Alan Elkins, M.D., Aubrey Dent, M.D. (observer-consultant), Jack Kremens, M.D. (Assembly liaison), Robert W. Gibson, M.D. (Board liaison), Donald G. Langsley, M.D. (Board liaison), Richard T. Rada, M.D. (consultant), and Donald Hammersley, M.D. (staff assigned).

[2]This task force included Warren S. Williams, M.D., chairperson, Roy M. Coleman, M.D., Lino Covi, M.D., and Roger Peele, M.D.

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Position Statement on Marijuana Laws

This statement was approved by the Assembly at its October 1978 meeting, by the Reference Committee at its November 1978 meeting, and by the Board of Trustees at its December 1978 meeting upon recommendation of the Council on Research and Development. It was prepared by the Council on Research and Development.[1]

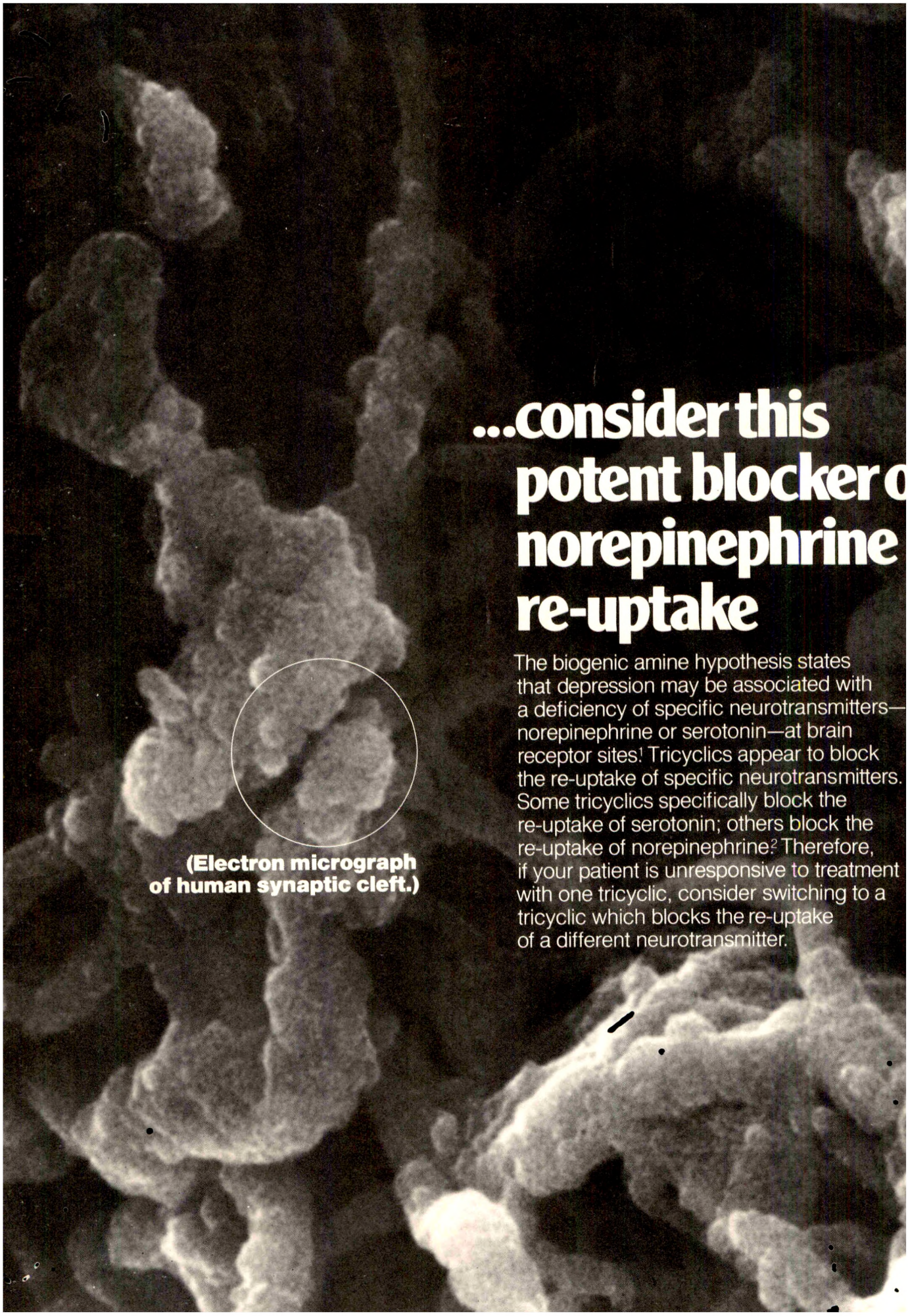
MARIJUANA IS now the third most popular recreational drug in the United States, after tobacco and alcohol. There appears to be no convincing evidence of serious lasting physical or psychological damage caused by moderate or intermittent use in healthy adults. Nonetheless, there have been more than 400,000 marijuana arrests annually for the last four years, at an estimated administrative cost of $600 million a year and an immeasurable cost in damage to the lives of many young people and their families. For the vast majority of users the main danger is being convicted of a crime. Thus, the present situation promotes disrespect for the law and has a destructive effect on efforts to present the dangers of drugs honestly to the public.

In 1972 the National Marijuana Commission concluded that "neither the marijuana user nor the drug itself can be said to constitute a danger to the public safety." It recommended the elimination of criminal penalties against the user. This policy, known as decriminalization, has been endorsed by a lengthening list of organizations, including the American Bar Association, the American Public Health Association, the National Education Association, the National Council of Churches, the Mental Health Association, and the governing board of the American Medical Association. President Carter supports decriminalization at the federal level, and 10 states have enacted decriminalization laws without producing any harmful effects or even any increase in marijuana use. The 1969 position statement of the American Psychiatric Association calls for "the cessation of disproportionate penalties for marijuana possession and use." Any criminal penalty for the possession of small amounts of marijuana for personal use can now be properly described as disproportionate, and decriminalization would fulfill the intent of the phrase used in 1969 as it has come to be interpreted by a large section of informed opinion. The Board of Trustees of the American Psychiatric Association therefore goes on record as supporting a policy of decriminalization.

This position statement is in no way to be construed as an endorsement of the social and recreational, particularly habitual, use of marijuana. Much research needs to be done concerning the nature of the action and effects, particularly long-term ones, of the drug, and until this knowledge is available, use of the drug must be considered potentially hazardous.

[1]The Council on Research and Development included Lester Grinspoon, M.D., chairperson, Charles Gaitz, M.D., Frank A. DeLeon-Jones, M.D., Edward Joel Sachar, M.D., John M. Davis, M.D., Joyce Lowinson, M.D. (observer-consultant), Leigh Roberts, M.D. (Assembly liaison), and Henry H. Work, M.D. (staff assigned).


When your patient is clinically depressed...
"...everything I saw was negative."



...consider this potent blocker o norepinephrine re-uptake

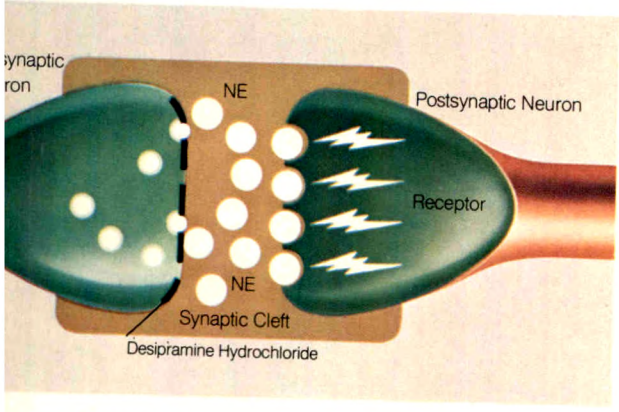
The biogenic amine hypothesis states that depression may be associated with a deficiency of specific neurotransmitters—norepinephrine or serotonin—at brain receptor sites.[1] Tricyclics appear to block the re-uptake of specific neurotransmitters. Some tricyclics specifically block the re-uptake of serotonin; others block the re-uptake of norepinephrine.[2] Therefore, if your patient is unresponsive to treatment with one tricyclic, consider switching to a tricyclic which blocks the re-uptake of a different neurotransmitter.

(Electron micrograph of human synaptic cleft.)

Norpramin®
desipramine hydrochloride ablets NF)
5, 50, 75, 100, 150 mg.

tent blocker norepinephrine re-uptake

cording to current theory[1,3] some cyclic antidepressants may fail in certain es of depression because they are not ficiently potent blockers of norepi-hrine re-uptake. Laboratory studies ve shown that desipramine is the most ent inhibitor of norepinephrine uptake.[4] Norpramin may work in some ients when other tricyclic antidepres-nts do not.



onsider it now, nsider it next.

Less anticholinergic activity

(See Warnings section.)

Studies in animals[5] and in normal human subjects[6,7] have shown that desipramine has significantly less anticholinergic activity than amitriptyline or doxepin. The single bond side chain is associated with lower anticholinergic activity than these other leading antidepressants. This may mean:

- less dry mouth
- less blurred vision
- less urinary retention

Helps patients remain calm but active

Norpramin does not usually inhibit normal activity although patients should be cautioned as to driving or operating machinery if drowsiness occurs.

In addition...

Begins to improve sleep patterns within one week in some patients[8]

- Norpramin helps relieve the sleep disturbances that often accompany depression.
- As the depression is relieved, the difficulty in falling asleep, restlessness, and early morning awakening diminish.

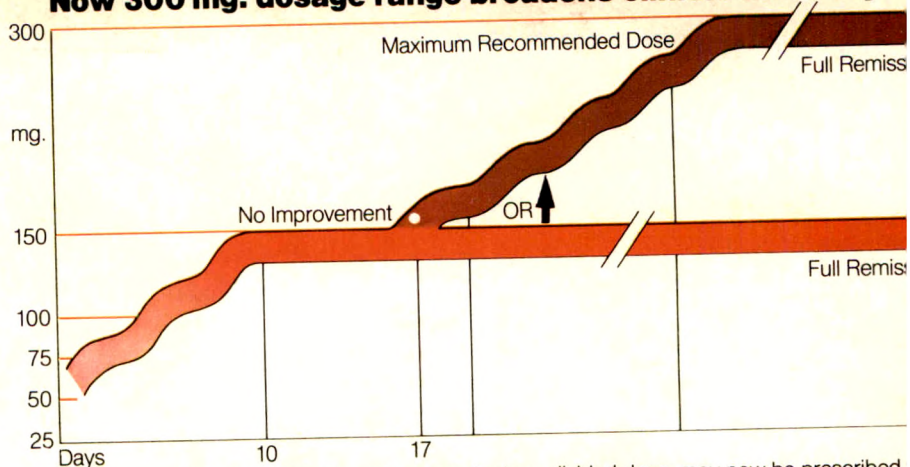
References:

1. Maas, J.W.: Biogenic amines and depression. Biochemical and pharmacological separation of two types of depression. Arch. Gen. Psychiat. 32:1357-1361, 1975. **2.** Mendels, J., Stern, S., and Frazer, A.: Dis. Nerv. Syst. 37:3, 1976. **3.** Davis, J.M.: Central biogenic amines and theories of depression and mania. In, Phenomenology and Treatment of Depression, W.E. Fann, I. Karacan, A.D. Pokorny, and R.L. Williams, Ed., New York, Spectrum Publications, Inc., 1977, pp. 17-32. **4.** Ross, S.B. and Renyi, A.L.: Tricyclic antidepressant agents. 1. Comparison of the inhibition of the uptake of ^{3}H-noradrenaline and ^{14}C-5-hydroxytryptamine in slices and crude synaptosome preparations of the midbrain-hypothalamus region of the rat brain. Acta Pharmacol. (Kobenhavn) 36:382-394, 1975. **5.** Snyder, S.H. and Yamamura, H.I.: Antidepressants and the muscarinic acetylcholine receptor. Arch. Gen. Psychiat. 34:236-239, 1977. **6.** Blackwell, B., Stefopoulos, A., Enders, P., Kuzma, R., and Adolphe, A.: The anticholinergic activity of two tricyclic antidepressants. Amer. J. Psychiat., 135:722-724, June 1978. **7.** Peterson, G.R., Hostetler, R.M., Blackwell, B., Kuzma, R., and Adolphe, A.B.: Effects of the tricyclic antidepressants desipramine and doxepin on anticholinergic and CNS activity in non-depressed volunteers. Neuroscience Abstr. 3:447, 1977. **8.** Zung, W.W.K.: The pharmacology of disordered sleep. A laboratory approach. J.A.M.A. 211:1532-1534, 1970.

(For prescribing information see next page.)

Merrell

Now 300 mg. dosage range broadens clinical flexibility



A maximum dosage of 300 mg. a day, as a single or divided dose, may now be prescribed Norpramin. Since inadequate dosage is thought to be one of the major causes of treatment failure, the new therapeutic range should prove clinically helpful. Plasma desipramine measurement would constitute the optimal guide to dosage monitoring.

Norpramin®
(desipramine hydrochloride tablets NF)

AVAILABLE ONLY ON PRESCRIPTION

Brief Summary

MECHANISM OF ACTION: Available evidence suggests that many depressions have a biochemical basis in the form of a relative deficiency of neurotransmitters such as norepinephrine and serotonin. Norepinephrine deficiency may be associated with relatively low urinary 3-methoxy-4-hydroxyphenyl glycol (MHPG) levels, while serotonin deficiencies may be associated with low spinal fluid levels of 5-hydroxyindolacetic acid.

While the precise mechanism of action of the tricyclic antidepressants is unknown, a leading theory suggests that they restore normal levels of neurotransmitters by blocking the re-uptake of these substances from the synapse in the central nervous system.

Evidence indicates that the secondary amine tricyclic antidepressants, including Norpramin, may have greater activity in blocking the re-uptake of norepinephrine. Tertiary amine tricyclic antidepressants, such as amitriptyline, may have greater effect on serotonin re-uptake.

Norpramin (desipramine hydrochloride) is not a monoamine oxidase (MAO) inhibitor and does not act primarily as a central nervous system stimulant. It has been found in some studies to have a more rapid onset of action than imipramine. Earliest therapeutic effects may occasionally be seen in 2 to 5 days, but full treatment benefit usually requires 2 to 3 weeks to obtain.

INDICATIONS: Norpramin (desipramine hydrochloride) is indicated for relief of symptoms in various depressive syndromes, especially endogenous depression.

CONTRAINDICATIONS: Desipramine hydrochloride should not be given in conjunction with, or within 2 weeks of, treatment with an MAO inhibitor drug; hyperpyretic crises, severe convulsions, and death have occurred in patients taking MAO inhibitors and tricyclic antidepressants. When Norpramin (desipramine hydrochloride) is substituted for an MAO inhibitor, at least 2 weeks should elapse between treatments. Norpramin should then be started cautiously and should be increased gradually.

The drug is contraindicated in the acute recovery period following myocardial infarction. It should not be used in those who have shown prior hypersensitivity to the drug. Cross sensitivity between this and other dibenzazepines is a possibility.

WARNINGS: 1. Extreme caution should be used when this drug is given in the following situations: a. In patients with cardiovascular disease, because of the possibility of conduction defects, arrhythmias, tachycardias, strokes, and acute myocardial infarction. b. In patients with a history of urinary retention or glaucoma, because of the anticholinergic properties of the drug. c. In patients with thyroid disease or those taking thyroid medication, because of the possibility of cardiovascular toxicity, including arrhythmias. d. In patients with a history of seizure disorder, because this drug has been shown to lower the seizure threshold. **2.** This drug is capable of blocking the antihypertensive effect of guanethidine and similarly acting compounds. **3.** USE IN PREGNANCY: Safe use of desipramine hydrochloride during pregnancy and lactation has not been established; therefore, if it is to be given to pregnant patients, nursing mothers, or women of childbearing potential, the possible benefits must be weighed against the possible hazards to mother and child. Animal reproductive studies have been inconclusive. **4.** USE IN CHILDREN: Norpramin (desipramine hydrochloride) is not recommended for use in children since safety and effectiveness in the pediatric age group have not been established. **5.** The patient should be cautioned that this drug may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery. **6.** In patients who may use alcohol excessively, it should be borne in mind that the potentiation may increase the danger inherent in any suicide attempt or overdosage.

PRECAUTIONS: 1. It is important that this drug be dispensed in the least possible quantities to depressed outpatients, since suicide has been accomplished with this class of drug. Ordinary prudence requires that children not have access to this drug or to potent drugs of any kind; if possible this drug should be dispensed in containers with child-resistant safety closures. Storage of this drug in the home must be supervised responsibly. **2.** If serious adverse effects occur, dosage should be reduced or treatment should be altered. **3.** Norpramin (desipramine hydrochloride) therapy in patients with manic-depressive illness may induce a hypomanic state after the depressive phase terminates. **4.** The drug may cause exacerbation of psychosis in schizophrenic patients. **5.** Close supervision and careful adjustment of dosage are required when this drug is given concomitantly with anticholinergic or sympathomimetic drugs. **6.** Patients should be warned that while taking this drug their response to alcoholic beverages may be exaggerated. **7.** Clinical experience in the concurrent administration of ECT and antidepressant drugs is limited. Thus, if such treatment is essential, the possibility of increased risk relative to benefits should be considered. **8.** The sedative effects of Norpramin and benzodiazepines (*e.g.*, chlordiazepoxide or diazepam) are additive. Both the sedative and anticholinergic effects of the major tranquilizers are additive to those of Norpramin. **9.** This drug should be discontinued as soon as possible prior to elective surgery because of the possible cardiovascular effects. Hypertensive episodes have been observed during surgery in patients taking desipramine hydrochloride. **10.** Both elevation and lowering of blood sugar levels have been reported. **11.** Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during therapy; the drug should be discontinued if there is evidence of pathologic neutrophil depression.

ADVERSE REACTIONS: Note: Included in the following listing are a few adverse reactions that have not been reported with this specific drug. However, the pharmacologic similarities among the tricyclic antidepressant drugs require that each of the reactions be considered when Norpramin (desipramine hydrochloride) is given.

Cardiovascular: hypotension, hypertension, tachycardia, palpitation, arrhythmias, heart block, myocardial infarction, stroke.

Psychiatric: confusional states (especially in the elderly) with hallucinations, disorientation, delusions, anxiety, restlessness, agitation; insomnia and nightmares; hypomania; exacerbation of psychosis.

Neurologic: numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures; alteration in EEG patterns; tinnitus.

Anticholinergic: dry mouth, and rarely associated sublingual adenitis; blurred vision, disturbance of accommodation, mydriasis, increased intraocular pressure; constipation, paralytic ileus; urinary retention, delayed micturition, dilatation of urinary tract.

Allergic: skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight), edema (of face and tongue or general), drug fever, cross sensitivity with other tricyclic drugs.

Hematologic: bone marrow depressions including agranulocytosis, eosinophilia, purpura, thrombocytopenia.

Gastrointestinal: anorexia, nausea and vomiting, epigastric distress, peculiar taste, abdominal cramps, diarrhea, stomatitis, black tongue.

Endocrine: gynecomastia in the male, breast enlarg ment and galactorrhea in the female; increased decreased libido, impotence, testicular swelling; elev tion or depression of blood sugar levels.

Other: jaundice (simulating obstructive), altered liv function; weight gain or loss; perspiration, flushi urinary frequency, nocturia; parotid swelling; drow ness, dizziness, weakness and fatigue, headache; a pecia.

Withdrawal Symptoms: Though not indicative of add tion, abrupt cessation of treatment after prolon therapy may produce nausea, headache, and malai

DOSAGE AND ADMINISTRATION: Not recommended for u in children. Lower dosages are recommended for elde patients and adolescents. Lower dosages are a recommended for outpatients compared to hospitali patients, who are closely supervised. Dosage should initiated at a low level and increased according clinical response and any evidence of intolerar Following remission, maintenance medication may required for a period of time and should be at the low dose that will maintain remission.

Usual Adult Dose: The usual adult dose is 100 to 200 per day. In more severely ill patients, dosage may further increased gradually to 300 mg./day if necess Dosages above 300 mg./day are not recommended.

Dosage should be initiated at a lower level increased according to tolerance and clinical respo

Treatment of patients requiring as much as 300 should generally be initiated in hospitals, where reg visits by the physician, skilled nursing care, and quent electrocardiograms (ECG's) are available.

The best available evidence of impending toxi from very high doses of Norpramin is prolongation o QRS or QT intervals on the ECG. Prolongation of th interval is also significant, but less closely correl with plasma levels. Clinical symptoms of intolera especially drowsiness, dizziness, and postural hypo sion, should also alert the physician to the nee reduction in dosage. Plasma desipramine measure would constitute the optimal guide to dosage mon ing.

Initial therapy may be administered in divided d or a single daily dose.

Maintenance therapy may be given on a once- schedule for patient convenience and compliance.

Adolescent and Geriatric Dose: The usual adolescen geriatric dose is 25 to 100 mg. daily.

Dosage should be initiated at a lower level increased according to tolerance and clinical resp to a usual maximum of 100 mg. daily. In more sever patients, dosage may be further increased to 150 day. Doses above 150 mg./day are not recommend these age groups.

Initial therapy may be administered in divided or a single daily dose.

Maintenance therapy may be given on a once- schedule for patient convenience and compliance.

OVERDOSAGE: See prescribing information for a di sion of symptoms and treatment of overdose.

Product Information as of April, 1978

Merrell

MERRELL-NATIONAL LABORATORIES
Division of Richardson-Merrell Inc.
Cincinnati, Ohio 45215, U.S.A.

8-3866 (

Stress control your patients can see and hear: GSR/Temp 2.

Extensive clinical papers attest to the value of biofeedback therapy as a natural alternative or adjunct in the treatment of anxiety/tension states. The advantages of the GSR/TEMP 2 system have been demonstrated in thousands of hospitals and educational facilities worldwide—enabling patients to learn a degree of voluntary control over tension related autonomic responses.

Both decreases in skin resistance as well as constriction of peripheral vasculature resulting in minor temperature changes are well recognized indicators of increases in both physical and mental stress. Only now have advances in electronic technology allowed the development of the first and only portable, inexpensive unit which is easy for the patient to adapt to and monitors these dual functions—the GSR/TEMP 2 *monitors both temperature and skin resistance!*

Temperature changes (of .05°C) are monitored through a thermistor probe which is placed on a finger. Skin Resistance is monitored through built in sensors or through remote electrodes. Feedback is either auditory from an internal speaker or earphone or visual from a meter.

The GSR/TEMP 2 is the *only* biofeedback system available in this lightweight form which incorporates both temperature and skin resistance as indicators of sympathetic nervous system activity.

The new GSR/TEMP 2 dual system is a complete unit including monitor, temperature sensor, earphone, read-out meter, remote monitoring electrodes, instruction cassette & record and manual. *It is the first time a compact and functional unit has been developed which is equally suitable for clinical or home use.*



15 Day Free Examination

Thought Technology Ltd., 2193 Clifton Ave., Montreal, Quebec, Canada H4A 2N5

Please send me _____ GSR 2 Kits at **$39.95**. each (plus $2.95 shipping and handling)

I prefer: ☐ Cassette, ☐ LP record.

I would also like the following optional equipment:

☐ Thermistor, $15.95, ☐ Meter, $12.95, ☐ Electrodes, $9.95, ☐ Carrying case, $12.95

Please send me instead _____ sets of the complete GSR/TEMP 2 Biofeedback System at $79.95 each (plus $3.95 shipping and handling).

I may return any of the equipment within 15 days for a complete refund. Full one year guarantee.

Residents of Quebec add 8% sales tax. Postage and handling outside North America: GSR2—$5.00; GSR/TEMP 2—$10.00. All U.S. orders sent from our N.Y. warehouse via U.P.S.

NAME _____

ADDRESS _____ APT. _____

CITY _____ STATE _____ ZIP _____

Method of payment:

☐ Check ☐ Money Order ☐ Master Charge ☐ Am. Ex. ☐ Visa ☐ Diners

Credit Card No. _____ Expiry Date _____

Signature _____

F

How to tell ATIVAN from other benzodiazepines.

Ativan is an effective, well-tolerated antianxiety agent. So are other benzodiazepines. But here are three ways Ativan stands out pharmacokinetically.*

1. Uncomplicated metabolic profile.

In contrast to most other benzodiazepines, the metabolic pathway of Ativan is straightforward. Ativan is active as given. Just one step from an active compound to an inactive one.

2. Steady-state levels achieved rapidly.

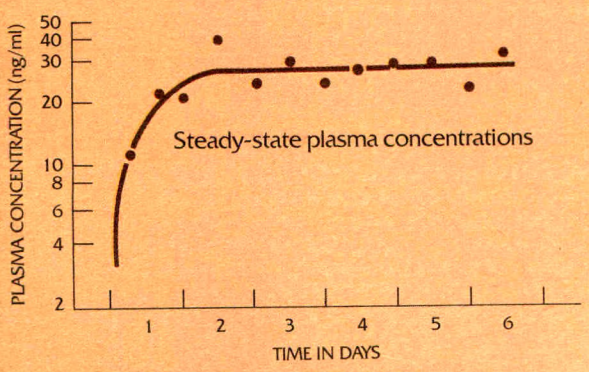
Most benzodiazepines take at least 7 to 10 days to reach steady-state serum levels. With Ativan, that equilibrium is reached in 2 to 3 days. A factor that can be important when you modify dosage schedules to meet individual needs.

3. No accumulation of multiple metabolites.

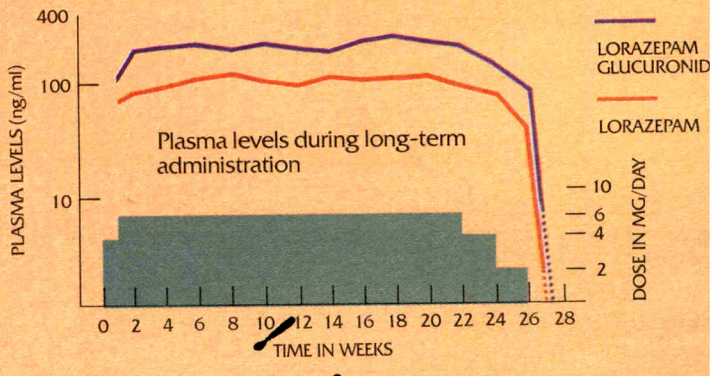
Because Ativan has no active metabolites to be eliminated at varying rates, steady-state levels are achieved predictably and smoothly. Six-month studies show no evidence of drug-accumulation above steady-state levels even at maximum daily dosages.

*The pharmacokinetic profile of a drug cannot, at present, be directly related to its therapeutic effectiveness.

Please refer to page after next for a summary of prescribing information.



Concentrations of lorazepam on repeated-dose regimen (single 1-mg. tablets given at 9 a.m., 3 p.m. and 9 p.m. daily for 6 days). A steady state is achieved after the second day, and is maintained thereafter.

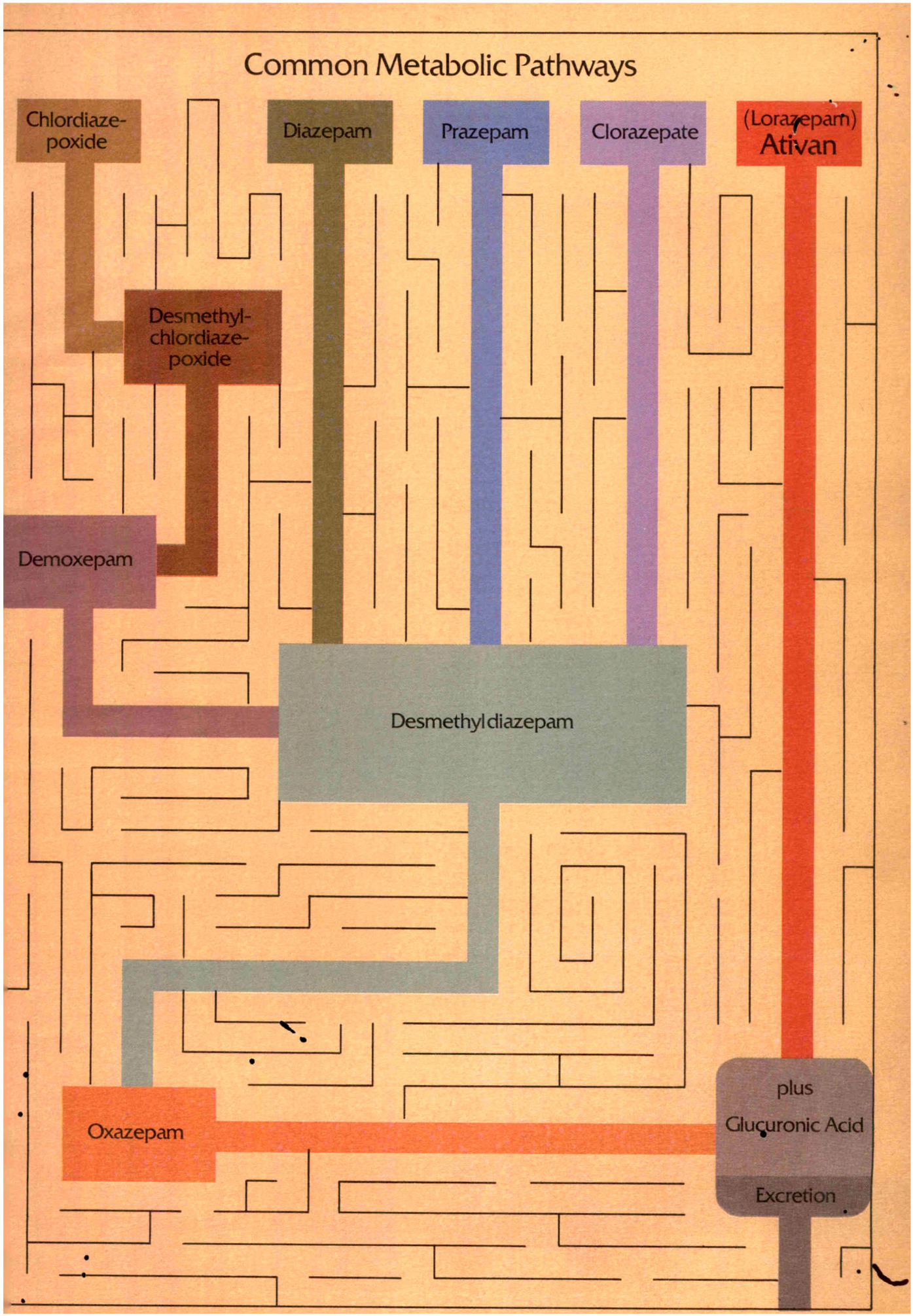


Lorazepam and lorazepam glucuronide plasma levels in ng/ml during chronic oral administration of the drug over a period of six months. Each point represents the mean of 8 subjects. The daily maintenance dose in milligrams per subject is given at weekly intervals.

ATIVAN® (LORAZEPAM) IV

The uncomplicated benzodiazepine

Wyet


Common Metabolic Pathways
Chlordiaze-poxide
Diazepam
Prazepam
Clorazepate
(Lorazepam)
Ativan
Desmethyl-chlordiaze-poxide
Demoxepam
Desmethyldiazepam
Oxazepam
plus
Glucuronic Acid
Excretion

(LORAZEPAM)®

ATIVAN®

prescribing information

Contraindications: Ativan is contraindicated in patients with known sensitivity to the benzodiazepines or with acute narrow-angle glaucoma.

Warnings: Ativan is not recommended for use in patients with a primary depressive disorder or psychosis. As with all patients on CNS-acting drugs, patients receiving lorazepam should be warned not to operate dangerous machinery or motor vehicles and that their tolerance for alcohol and other CNS depressants will be diminished.

Physical and Psychological Dependence: Withdrawal symptoms similar in character to those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepine drugs. These symptoms include convulsions, tremor, abdominal and muscle cramps, vomiting and sweating. Addiction-prone individuals, such as drug addicts and alcoholics, should be under careful surveillance when receiving benzodiazepines because of the predisposition of such patients to habituation and dependence.

Precautions: In patients with depression accompanying anxiety, a possibility for suicide should be borne in mind.

For elderly or debilitated patients, the initial daily dosage should not exceed 2 mg in order to avoid oversedation.

Ativan dosage should be terminated gradually since abrupt withdrawal of any antianxiety agent may result in symptoms similar to those for which patients are being treated: anxiety, agitation, irritability, tension, insomnia and occasional convulsions.

The usual precautions for treating patients with impaired renal or hepatic function should be observed.

In patients where gastrointestinal or cardiovascular disorders coexist with anxiety, it should be noted that lorazepam has not been shown to be of significant benefit in treating the gastrointestinal or cardiovascular component.

Esophageal dilation occurred in rats treated with lorazepam for more than one year at 6 mg/kg/day. The no-effect dose was 1.25 mg/kg/day (approximately 6 times the maximum human therapeutic dose of 10 mg per day). The effect was reversible only when the treatment was withdrawn within two months of first observation of the phenomenon. The clinical significance of this is unknown. However, use of lorazepam for prolonged periods and in geriatric patients requires caution, and there should be frequent monitoring for symptoms of upper G.I. disease.

Safety and effectiveness of Ativan in children of less than 12 years have not been established.

Essential Laboratory Tests: Some patients on Ativan have developed leukopenia and some have had elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function tests are recommended for patients on long-term therapy.

Clinically Significant Drug Interactions: The benzodiazepines including Ativan produce CNS depressant effects when administered with such medications as barbiturates or alcohol.

Carcinogenesis and Mutagenesis: No evidence of carcinogenic potential emerged in rats during an 18-month study with Ativan. No studies regarding mutagenesis have been performed.

Pregnancy: Reproductive studies in animals were performed in mice, rats, and two strains of rabbits. Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastroschisis, malformed skull and microphthalmia) were seen in drug-treated rabbits without relationship to dosage. Although all of these anomalies were not present in the concurrent control group, they have been reported to occur randomly in historical controls. At doses of 40 mg/kg and higher, there was evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lower doses.

The clinical significance of the above findings is not known. However, an increased risk of congenital malformations associated with the use of minor tranquilizers (chlordiazepoxide, diazepam and meprobamate) during the first trimester of pregnancy has been suggested in several studies. Because the use of these drugs is rarely a matter of urgency, the use of lorazepam during this period should almost always be avoided. The possibility that a woman of child-bearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant, they should communicate with their physician about the desirability of discontinuing the drug.

In humans, blood levels obtained from umbilical cord blood indicate placental transfer of lorazepam and lorazepam glucuronide.

Nursing Mothers: It is not known whether oral lorazepam is excreted in human milk like the other benzodiazepine tranquilizers. As a general rule, nursing should not be undertaken while a patient is on a drug since many drugs are excreted in human milk.

Adverse Reactions: Adverse reactions, if they occur, are usually observed at the beginning of therapy and generally disappear on continued medication or upon decreasing the dose. In a sample of about 3,500 anxious patients, the most frequent adverse reaction to Ativan is sedation (15.9%), followed by dizziness (6.9%), weakness (4.2%) and unsteadiness (3.4%). Less frequent adverse reactions are disorientation, depression, nausea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, eye function disturbance, together with various gastrointestinal symptoms and autonomic manifestations. The incidence of sedation and unsteadiness increased with age.

Small decreases in blood pressure have been noted but are not clinically significant, probably being related to the relief of anxiety produced by Ativan.

Overdosage: In the management of overdosage with any drug, it should be borne in mind that multiple agents may have been taken.

Manifestations of Ativan overdosage include somnolence, confusion and coma. Induced vomiting and/or gastric lavage should be undertaken followed by general supportive care, monitoring of vital signs and close observation of the patient. Hypotension, though unlikely, usually may be controlled with Levarterenol Bitartrate Injection, U.S.P. Caffeine and Sodium Benzoate Injection, U.S.P. may be used to counteract CNS depressant effects. The usefulness of dialysis has not been determined.

Dosage and Administration: Ativan is administered orally. For optimal results, dose, frequency of administration and duration of therapy should be individualized according to patient response. To facilitate this, scored 1.0 and 2.0 mg tablets are available.

The usual range is 2 to 6 mg/day given in divided doses, the largest dose being taken before bedtime, but the daily dosage may vary from 1 to 10 mg/day. For anxiety, most patients require an initial dose of 2 to 3 mg/day given b.i.d. or t.i.d.

For insomnia due to anxiety or transient situational stress, a single daily dose of 2 to 4 mg may be given, usually at bedtime.

For elderly or debilitated patients, an initial dosage of 1 to 2 mg/day in divided doses is recommended, to be adjusted as needed and tolerated.

The dosage of Ativan should be increased gradually when needed to help avoid adverse effects. When higher dosage is indicated, the evening dose should be increased before the daytime doses.

How Supplied: Ativan (lorazepam) is available in scored 1.0 and 2.0 mg tablets in bottles of 100.

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A highly effective, low-dose, non-accumulating antianxiety agent

Wyeth Laboratories
Philadelphia, Pa. 191(

MEDICAL DIRECTOR OF ALCOHOLISM THERAPY PROGRAM

The Department of Substance Abuse of Henry Ford Hospital, a comprehensive health care delivery system that integrates clinical and basic scientific research and medical education with innovative health services in Detroit and its surrounding communities, is seeking a Medical Director for its Alcoholism Therapy Program. Duties will include supervising the medical care of a 14-bed inpatient unit at the main hospital and assisting in the development for a plan for a new suburban 50-bed residential and outpatient facility and providing inservice education and research. We offer a competitive salary and liberal fringe benefits. Interested parties should send their curriculum vitae, in strict confidence, to:

Bernard Schuman, M.D.
Chairperson, Search Committee
Department of Substance Abuse
2799 West Grand Blvd. — Office A573W
Detroit, Michigan 48202

Director of Adolescent Psychiatry

343 bed suburban Detroit area hospital has a full-time staff vacancy for the newly created position of Director of the Adolescent Psychiatric Program. A new 40-bed inpatient unit is under construction to accommodate this program scheduled for opening in October, 1979.

The Hospital presently has an inpatient unit of 38 beds for adults and adolescents, a partial hospitalization (day care) program, an outpatient psychiatric clinic and a Psychology Department.

An excellent total compensation program has been designed for this position including negotiable salary and comprehensive benefit and retirement programs.

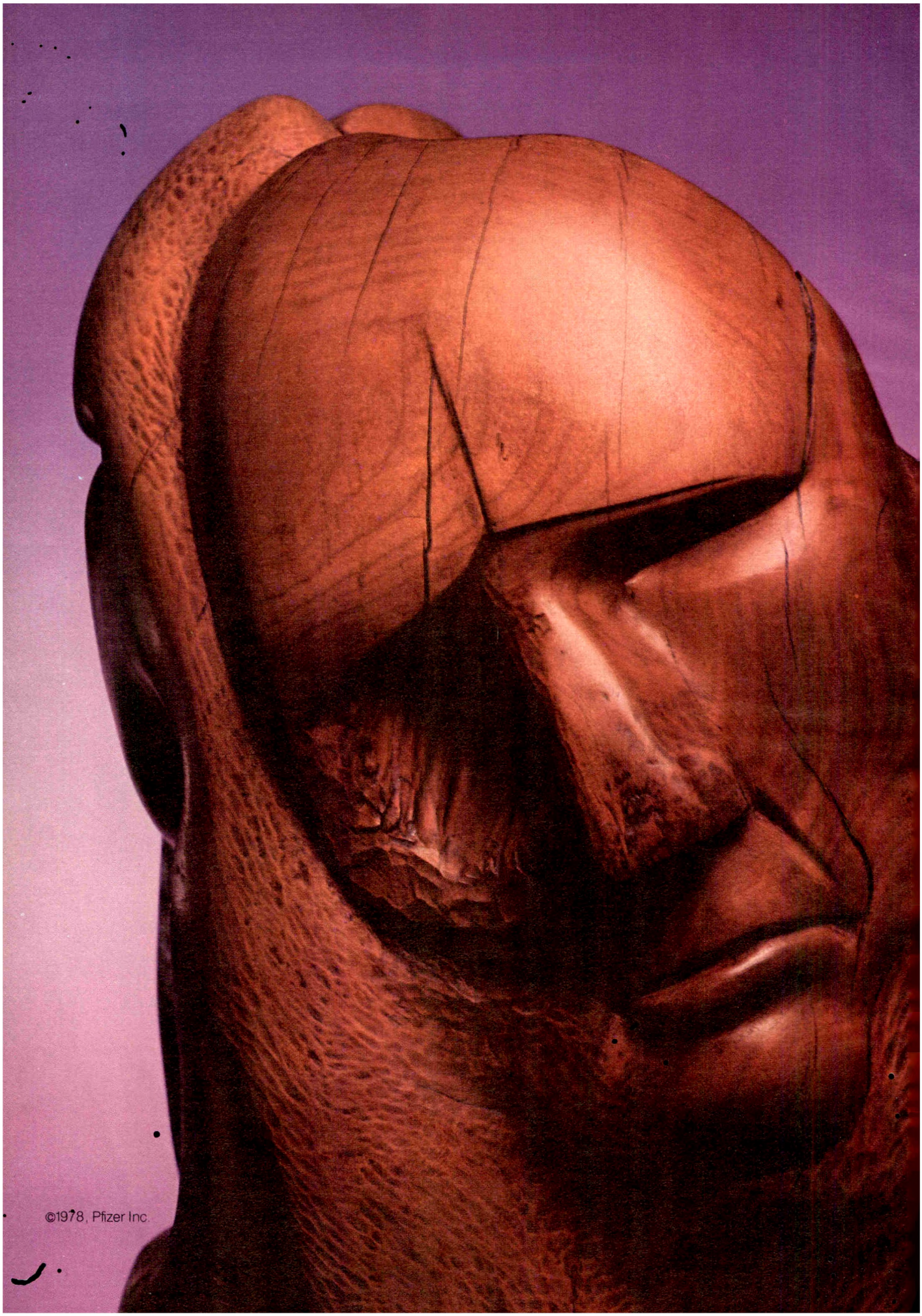
Interested board certified Psychiatrists (Adolescent and Child Psychiatry) wishing to apply for the position should submit their curriculum vitae in confidence or call collect if they so desire. Contact BRUCE L. NELSON, VICE PRESIDENT FOR MEDICAL SERVICES, WYANDOTTE GENERAL HOSPITAL, 2333 BIDDLE AVENUE, WYANDOTTE, MI, 48192. PHONE (313) 284-2400, ext. 2504.

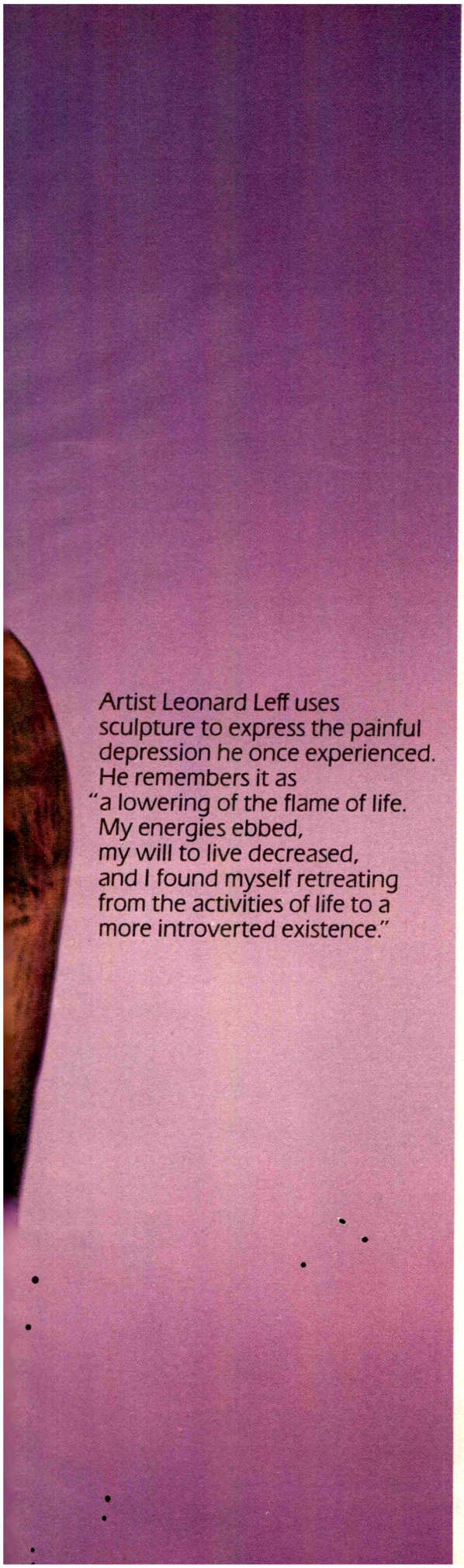
NATIONAL PSYCHIATRIC DIRECTOR

Internationally based rehabilitation and health planning agency in New York City seeks upwardly mobile, flexible and creative Board eligible (or certified) **psychiatrist** for high level administrative position. Candidates must be able to travel. Duties include supervision of 500 in-resident patients in 5 states, program design, and the articulation of health care issues for purposes of social change. Salary is $50,000 plus fringe. We are interested in hiring for this position as quickly as possible. Please send resume to Ms. Shirley Lewkowitz, Odyssey Institute, Mabon Building 13, Wards Island, N.Y., N.Y. 10035.

FACULTY PSYCHIATRIST

Department of Psychiatry, University of Utah College of Medicine. Inpatient work with general psychiatric and forensic population. Twenty percent free time for research. Primary location at Utah State Hospital, one hours drive from University. Salary to start: $45,468 board eligible; $48,731 board certified. Plus approx. $2,200 per year deposited in retirement fund. Plus large benefit package. Spectacular mountain country with year-round recreation, within one hour of four major ski resorts. Near two major universities. Apply to David Tomb, M.D., University of Utah College of Medicine, Salt Lake City, UT 84132


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Artist Leonard Leff uses sculpture to express the painful depression he once experienced. He remembers it as "a lowering of the flame of life. My energies ebbed, my will to live decreased, and I found myself retreating from the activities of life to a more introverted existence."

WHEN DEPRESSION EXPRESSES ITSELF

SINEQUAN®
(DOXEPIN HCl)

ANTIDEPRESSANT EFFECTIVENESS with convenient once-a-day *h.s.* dosage*

150-MG CAPSULE†

Also available in:
100-mg, 75-mg, 50-mg, 25-mg, 10-mg CAPSULES and ORAL CONCENTRATE, 10 mg/ml, in 120-ml (4-oz) bottles

*The total daily dosage of Sinequan, up to 150 mg, may be administered on a once-a-day schedule without loss of effectiveness.

† The 150-mg capsule strength is intended for maintenance therapy only and is not recommended for initiation of treatment.

See Brief Summary on next page for information on contraindications, warnings, precautions and adverse reactions.

ANTIDEPRESSANT EFFECTIVENESS

SINEQUAN® (DOXEPIN HCl)

150-mg CAPSULE*

CONVENIENT ONCE-A-DAY *h.s.* DOSAGE

which may improve patient compliance. The total daily dosage, up to 150 mg per day, may be given on a once-a-day schedule without loss of effectiveness. Sinequan may also be given on a divided dosage schedule, up to 300 mg per day.

PROMINENT SEDATIVE EFFECT

which may help to relieve the difficulty in falling and staying asleep, and the early-morning awakening often associated with depression.

ESTABLISHED ANTIANXIETY ACTIVITY

to help alleviate the anxiety which often accompanies clinical depression.

USUALLY WELL TOLERATED

At doses up to 150 mg per day, Sinequan does not generally affect the antihypertensive activity of guanethidine and related compounds. Tachycardia and hypotension have been reported occasionally. Drowsiness is the most commonly observed side effect. Dry mouth, blurred vision, constipation and urinary retention have been reported.

EXTENDED RANGE OF DOSAGE STRENGTHS

for flexibility in individualizing therapy.

*The 150-mg capsule strength is intended for maintenance therapy only and is not recommended for initiation of treatment.

BRIEF SUMMARY
SINEQUAN® (doxepin HCl) Capsules/Oral Concentrate

Contraindications. Contraindicated in individuals who have shown hypersensitivity to the drug, and in patients with glaucoma or a tendency to urinary retention. These disorders should be ruled out, particularly in older patients. Possibility of cross sensitivity with other dibenzoxepines should be kept in mind.

Warnings. The once-a-day dosage regimen of SINEQUAN (doxepin HCl) in patients with intercurrent illness or patients taking other medications should be carefully adjusted. This is especially important in patients receiving other medications with anticholinergic effects.

Usage in Geriatrics: The use of SINEQUAN on a once-a-day dosage regimen in geriatric patients should be adjusted carefully based on the patient's condition.

Usage in Pregnancy: Reproduction studies performed in animals have shown no evidence of harm to the animal fetus. Since there is no experience in pregnant women receiving this drug, safety in pregnancy has not been established. There are no data with respect to the secretion of the drug in human milk and its effect on the nursing infant.

Usage in Children: Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

MAO Inhibitors: Serious side effects and even death have been reported following the concomitant use of certain drugs with MAO inhibitors. Therefore, MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with this drug. The exact length of time may vary and is dependent upon the particular MAO inhibitor being used, the length of time it has been administered and the dosage involved.

Usage with Alcohol: It should be borne in mind that alcohol ingestion may increase the danger inherent in any intentional or unintentional SINEQUAN overdosage. This is especially important in patients who may use alcohol excessively.

Precautions. Since drowsiness may occur with the use of this drug, patients should be warned of that possibility and cautioned against driving a car or operating dangerous machinery while taking this drug.

Patients should also be cautioned that their response to alcohol may be potentiated.

Since suicide is an inherent risk in any depressed patient, and may remain so until significant improvement has occurred, patients should be closely supervised during the early course of therapy. Prescriptions should be written for the smallest feasible amount.

Should increased symptoms of psychosis or shift to manic symptomatology occur, it may be necessary to reduce dosage or add a major tranquilizer to the dosage regimen.

Adverse Reactions. NOTE: Some of the adverse reactions noted below have not been specifically reported with SINEQUAN use. However, due to the close pharmacological similarities among the tricyclics, the reactions should be considered when prescribing SINEQUAN.

Anticholinergic Effects: Dry mouth, blurred vision, constipation, and urinary retention have been reported. If they do not subside with continued therapy, or become severe, it may be necessary to reduce the dosage.

Central Nervous System Effects: Drowsiness is the most commonly noticed side effect. This tends to disappear as therapy is continued. Other infrequently reported CNS side effects are confusion, disorientation, hallucinations, numbness, paresthesias, ataxia, and extrapyramidal symptoms and seizures.

Cardiovascular: Cardiovascular effects including hypotension and tachycardia have been reported occasionally.

Allergic: Skin rash, edema, photosensitization, and pruritus have occasionally occurred.

Hematologic: Eosinophilia has been reported in a few patients. There have been occasional reports of bone marrow depression manifesting as agranulocytosis, leukopenia, thrombocytopenia, and purpura.

Gastrointestinal: Nausea, vomiting, indigestion, taste disturbances, diarrhea, anorexia, and aphthous stomatitis have been reported. (See anticholinergic effects.)

Endocrine: Raised or lowered libido, testicular swelling, gynecomastia in males, enlargement of breasts and galactorrhea in the female, raising or lowering of blood sugar levels have been reported with tricyclic administration.

Other: Dizziness, tinnitus, weight gain, sweating, chills, fatigue, weakness, flushing, jaundice, alopecia, and headache have been occasionally observed as adverse effects.

Dosage and Administration. For most patients with illness of mild to moderate severity, a starting daily dose of 75 mg is recommended. Dosage may subsequently be increased or decreased at appropriate intervals and according to individual response. The usual optimum dose range is 75 mg/day to 150 mg/day.

In more severely ill patients higher doses may be required with subsequent gradual increase t 300 mg/day if necessary. Additional therapeutic effect is rarely to be obtained by exceeding dose of 300 mg/day.

In patients with very mild symptomatology or emotional symptoms accompanying organi disease, lower doses may suffice. Some of these patients have been controlled on doses as lo as 25-50 mg/day.

The total daily dosage of SINEQUAN (doxepin HCl) may be given on a divided or once-a-da dosage schedule. If the once-a-day schedule is employed the maximum recommended dose 150 mg/day. This dose may be given at bedtime. **The 150 mg capsule strength is intended fo maintenance therapy only and is not recommended for initiation of treatment.**

Antianxiety effect is apparent before the antidepressant effect. Optimal antidepressant effec may not be evident for two to three weeks.

Overdosage.

A. Signs and Symptoms

1. Mild: Drowsiness, stupor, blurred vision, excessive dryness of mouth.
2. Severe: Respiratory depression, hypotension, coma, convulsions, cardiac arrhythmias an tachycardias.

Also: urinary retention (bladder atony), decreased gastrointestinal motility (paralytic ileus hyperthermia (or hypothermia), hypertension, dilated pupils, hyperactive reflexes.

B. Management and Treatment

1. Mild: Observation and supportive therapy is all that is usually necessary.
2. Severe: Medical management of severe SINEQUAN overdosage consists of aggressi supportive therapy. If the patient is conscious, gastric lavage, with appropriate precautions prevent pulmonary aspiration, should be performed even though SINEQUAN is rapidly absorbe The use of activated charcoal has been recommended, as has been continuous gastric lavag with saline for 24 hours or more. An adequate airway should be established in comatose patien and assisted ventilation used if necessary. EKG monitoring may be required for several day since relapse after apparent recovery has been reported. Arrhythmias should be treated with th appropriate antiarrhythmic agent. It has been reported that many of the cardiovascular and CN symptoms of tricyclic antidepressant poisoning in adults may be reversed by the slow intr venous administration of 1 mg to 3 mg of physostigmine salicylate. Because physostigmine rapidly metabolized, the dosage should be repeated as required. Convulsions may respond standard anticonvulsant therapy; however, barbiturates may potentiate any respiratory depre sion. Dialysis and forced diuresis generally are not of value in the management of overdosag due to high tissue and protein binding of SINEQUAN.

Supply. SINEQUAN is available as capsules containing doxepin HCl equivalent to: 10 mg, 75 m and 100 mg doxepin: bottles of 100, 1000, and unit-dose packages of 100 (10 x 10's). 25 mg a 50 mg doxepin: bottles of 100, 1000, 5000, and unit-dose packages of 100 (10 x 10's). 150 m doxepin: bottles of 50, 500, and unit-dose packages of 100 (10 x 10's). SINEQUAN O Concentrate (10 mg/ml) is available in 120 ml bottles with an accompanying dropper calibrat at 5 mg, 10 mg, 15 mg, 20 mg, and 25 mg. Each ml contains doxepin HCl equivalent to 10 m doxepin. Just prior to administration, SINEQUAN Oral Concentrate should be diluted w approximately 120 ml of water, whole or skimmed milk, or orange, grapefruit, tomato, prune pineapple juice. SINEQUAN Oral Concentrate is not physically compatible with a number carbonated beverages. For those patients requiring antidepressant therapy who are methadone maintenance, SINEQUAN Oral Concentrate and methadone syrup can be mix together with Gatorade®, lemonade, orange juice, sugar water, Tang®, or water; but not w grape juice. Preparation and storage of bulk dilutions is not recommended.

More detailed professional information available on request.

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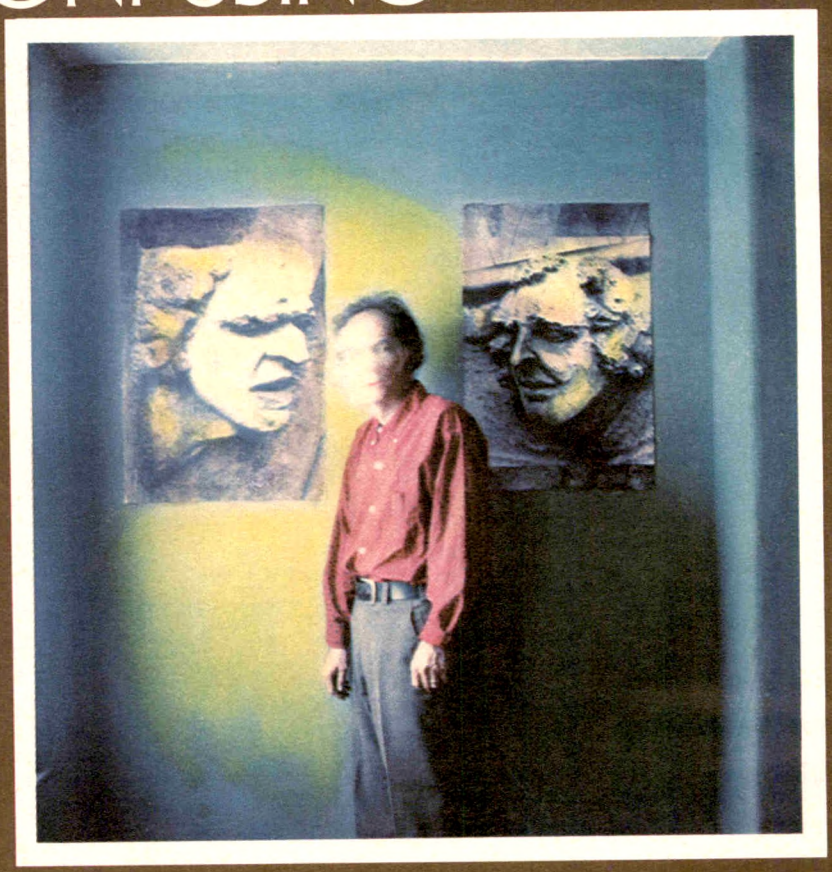
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Before prescribing, see complete prescribing information in SK&F literature or PDR. The following is a brief summary.

Indications

Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

Effective: For the management of the manifestations of psychotic disorders.

Possibly effective: To control excessive anxiety, tension and agitation as seen in neuroses or associated with somatic conditions.

'Stelazine' has not been shown effective in the management of behavioral complications in patients with mental retardation.

Final classification of the less-than-effective indications requires further investigation.

Contraindications: Comatose or greatly depressed states due to C.N.S. depressants; blood dyscrasias; bone marrow depression; liver damage.

Warnings: Generally avoid using in patients hypersensitive (e.g., blood dyscrasias, jaundice) to any phenothiazine. Caution patients about activities requiring alertness (e.g., operating vehicles or machinery), especially during the first few days' therapy.

Use in pregnancy only when necessary for patient's welfare.

Precautions: Use cautiously in angina. Avoid high doses and parenteral administration when cardiovascular system is impaired. Antiemetic effect may mask signs of toxic drug overdosage or physical disorders. Additive effect is possible with other C.N.S. depressants. Prolonged administration of high doses may result in cumulative effects with severe C.N.S. or vasomotor symptoms. If retinal changes occur, discontinue drug. Agranulocytosis, thrombocytopenia, pancytopenia, anemia, cholestatic jaundice, liver damage have been reported.

Patients on long-term therapy, especially high doses, should be evaluated periodically for possible adjustment or discontinuance of drug therapy.

Adverse Reactions: Drowsiness, dizziness, skin reactions, rash, dry mouth, insomnia, amenorrhea, fatigue, muscular weakness, anorexia, lactation, blurred vision. Neuromuscular (extrapyramidal) reactions: motor restlessness, dystonias, pseudo-parkinsonism, persistent tardive dyskinesia.

Other adverse reactions reported with Stelazine (trifluoperazine HCl, SK&F) or other phenothiazines: Some adverse effects are more frequent or intense in specific disorders (e.g., mitral insufficiency or pheochromocytoma).

Grand mal convulsions; altered cerebrospinal fluid proteins; cerebral edema; prolongation and intensification of the action of C.N.S. depressants, atropine, heat, and organophosphorus insecticides; nasal congestion, headache, nausea, constipation, obstipation, adynamic ileus, inhibition of ejaculation; reactivation of psychotic processes, catatonic-like states; hypotension (sometimes fatal); cardiac arrest; leukopenia, eosinophilia, pancytopenia, agranulocytosis, thrombocytopenic purpura; jaundice, biliary stasis; menstrual irregularities, galactorrhea, gynecomastia, false positive pregnancy tests; photosensitivity, itching, erythema, urticaria, eczema up to exfoliative dermatitis; asthma, laryngeal edema, angioneurotic edema, anaphylactoid reactions; peripheral edema; reversed epinephrine effect; hyperpyrexia; a systemic lupus erythematosus-like syndrome; pigmentary retinopathy; with prolonged administration of substantial doses, skin pigmentation, epithelial keratopathy, and lenticular and corneal deposits. EKG changes have been reported, but relationship to myocardial damage is not confirmed. Discontinue long-term, high-dose therapy gradually. NOTE: Sudden death in patients taking phenothiazines (apparently due to cardiac arrest or asphyxia due to failure of cough reflex) has been reported, but no causal relationship has been established.

Supplied: Tablets, 1 mg., 2 mg., 5 mg. and 10 mg., in bottles of 100; in Single Unit Packages of 100 (intended for institutional use only); Injection, 2 mg./ml.; and Concentrate (intended for institutional use only), 10 mg./ml.

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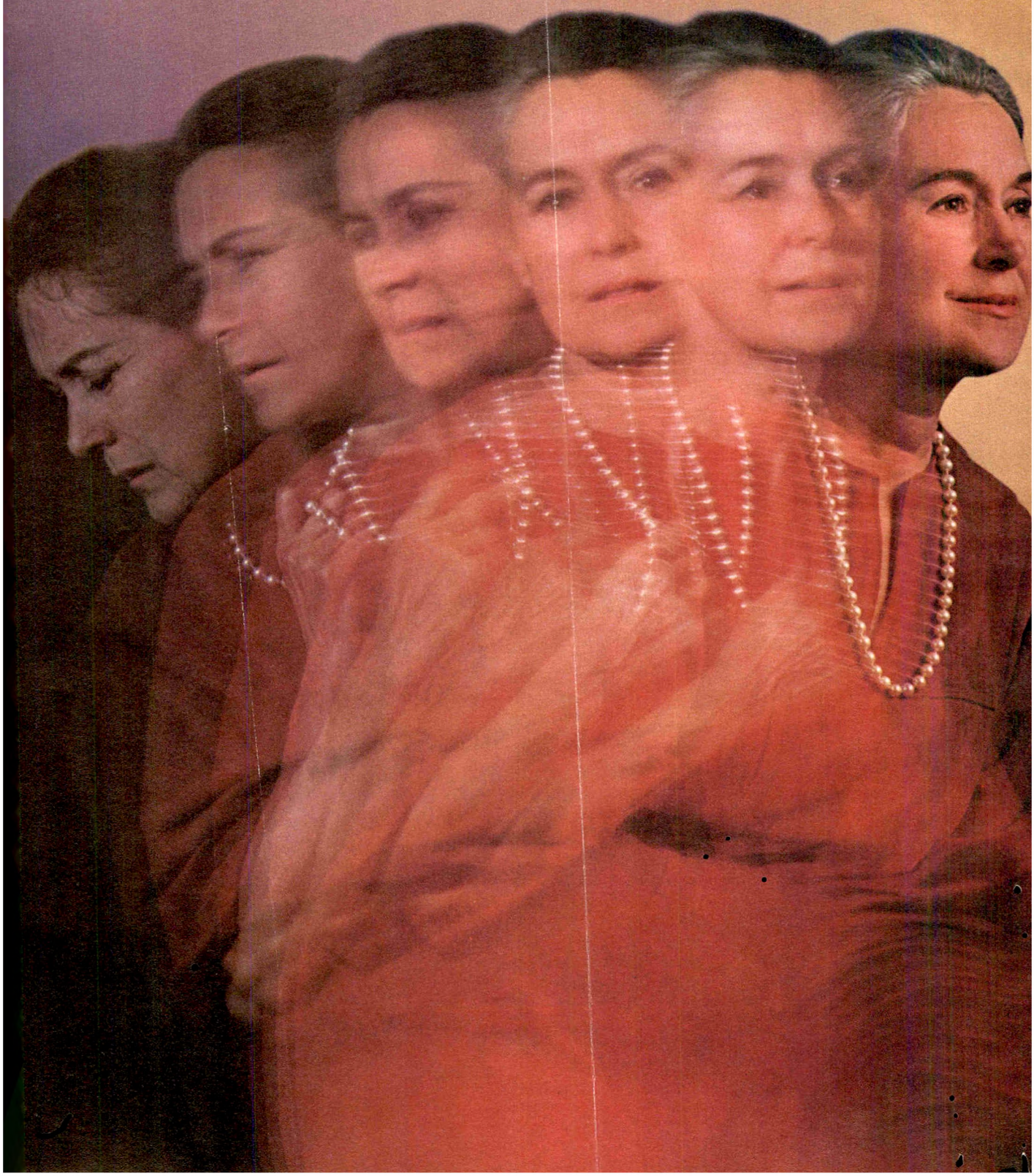
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References: 1. Hollister, L.E.: Treatment of depression with drugs, Ann. Intern. Med. 89:78, 1978. 2. Blackwell, B. et al.: Anticholinergic activity of two tricyclic antidepressants, Am. J. Psychiatry 135:722, 1978. 3. Barnes, R. J.: Clinical depression: Double-blind study of desipramine and amitriptyline in depressive neurosis, Scientific Exhibit, Southern Medical Convention, Miami, Fla., Nov. 16-19, 1975.

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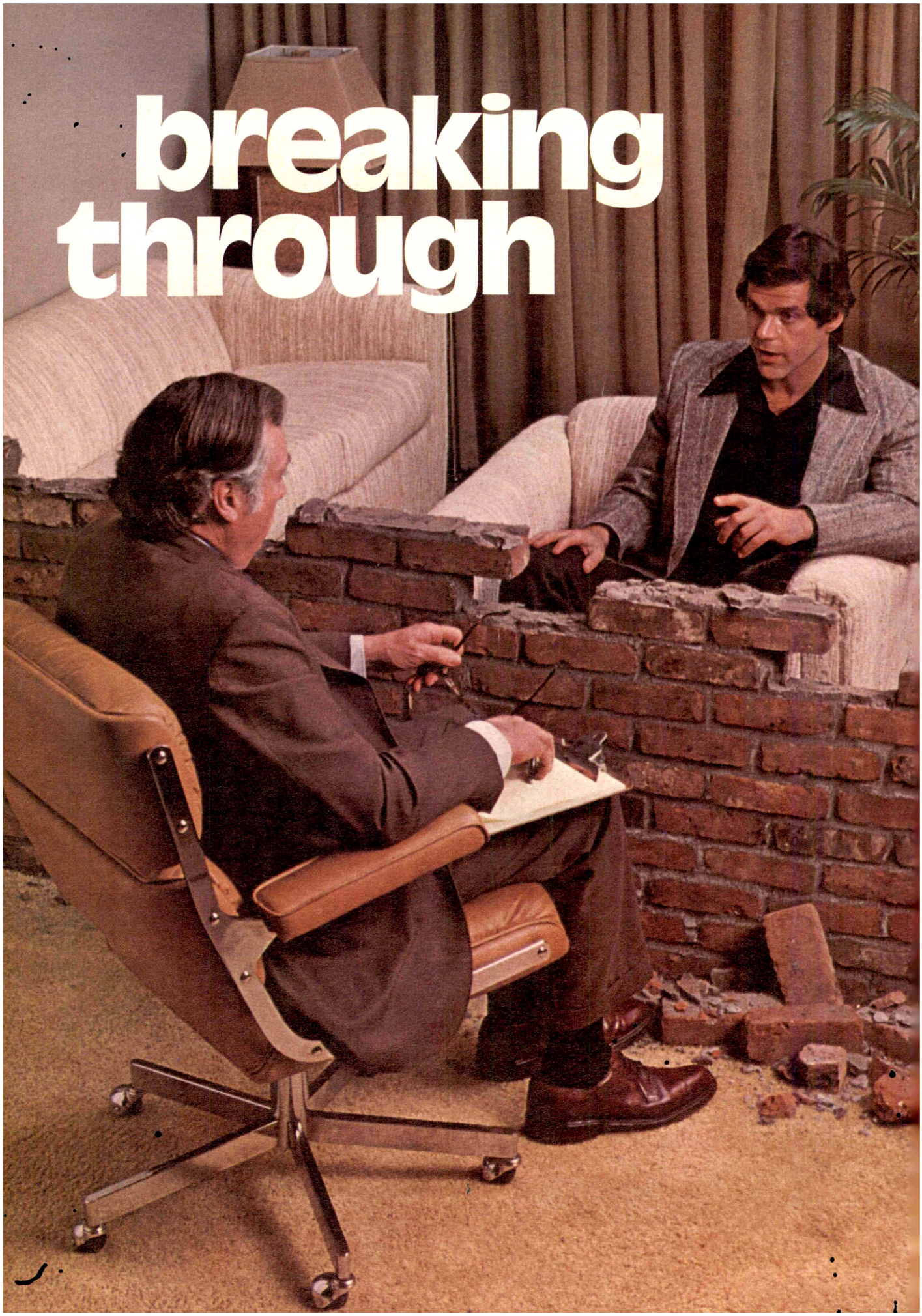
when the first tricyclic fails

Indication: For relief of mental depression. **Contraindications:** Do not use MAO inhibitors concomitantly or within 2 weeks of the use of this drug. Hyperpyretic crises or severe convulsive seizures may occur with such combinations; potentiation of adverse reactions can be serious or even fatal. When substituting Pertofrane (desipramine HCl) in patients receiving an MAO inhibitor, allow an interval of at least 14 days. Initial dosage in such patients should be low and increases should be gradual and cautiously prescribed. The drug is contraindicated following recent myocardial infarction and in patients with a known hypersensitivity to tricyclic antidepressants. **Warnings:** Activation of psychosis may occasionally be observed in schizophrenic patients. Due to atropine-like effects and sympathomimetic potentiation, use only with the greatest care in patients with narrow-angle glaucoma or urethral or ureteral spasm. Do not use in patients with the following conditions unless the need outweighs the risk: severe coronary heart disease with EKG abnormalities, progressive heart failure, angina pectoris, paroxysmal tachycardia and active seizure disorder (may lower seizure threshold). This drug may block the action of the antihypertensive, guanethidine, and related adrenergic neuron-blocking agents. Hypertensive episodes have been observed during surgery. The concurrent use of other central nervous system drugs or alcohol may potentiate adverse effects. Since many such drugs may be used during surgery, desipramine should be discontinued prior to elective procedures. The potentiation resulting from excessive use of alcohol may increase the danger inherent in a suicide attempt or overdosage. Caution patients on the possibility of impaired ability to operate a motor vehicle or dangerous machinery. Do not use in women who are or may become pregnant, or in children under 12 years of age, unless the clinical situation warrants the potential risk. Because of increased sensitivity to the drug, use lower than normal dosage in adolescent and geriatric patients. **Precautions:** Potentially suicidal patients require careful supervision and protective measures during therapy. Prescriptions should be limited to small quantities. Discontinuation of the drug may be necessary in the presence of increased agitation and anxiety shifting to hypomanic or manic excitement. Atropine-like effects may be more pronounced (e.g. paralytic ileus) in susceptible patients and in those receiving anticholinergic drugs (including antiparkinsonism agents). Prescribe cautiously in hyperthyroid patients and in those receiving thyroid medications; transient cardiac arrhythmias have occurred in rare instances. Periodic blood and liver studies should supplement careful clinical observations in all patients undergoing extended courses of therapy. **Adverse Reactions:** The following have been reported: *Nervous System:* dizziness, drowsiness, insomnia, headache, disturbed visual accommodation, tremor, unsteadiness, tinnitus, paresthesias, changes in EEG patterns, epileptiform seizures, mild extrapyramidal activity, falling and neuromuscular incoordination. A confusional state (with such symptoms as hallucinations and disorientation), particularly in older patients and at higher dosage, may require discontinuation of the drug. *Gastrointestinal Tract:* anorexia, dryness of the mouth, nausea, epigastric distress, constipation and diarrhea. *Skin:* skin rashes (including photosensitization), perspiration and flushing sensations. *Liver:* rare cases of transient jaundice (apparently of an obstructive nature) and liver damage. If jaundice or abnormalities in liver function tests occur, discontinue the drug and investigate. *Blood Elements:* bone-marrow depression, agranulocytosis, thrombocytopenia and purpura. If these occur, discontinue the drug. Transient eosinophilia has been observed. *Cardiovascular System:* orthostatic hypotension and tachycardia. Carefully supervise patients requiring concomitant vasodilating therapy, particularly during initial phases. *Genitourinary System:* urinary frequency or retention and impotence. *Endocrine System:* occasional hormonal effects, including gynecomastia, galactorrhea and breast enlargement, and decreased libido and estrogenic effect. *Sensitivity:* urticaria and rare instances of drug fever and cross-sensitivity with imipramine. **Dosage:** All patients except geriatric and adolescent: 75 to 150 mg./day in divided doses or as single daily dose. Dosage may be increased up to 200 mg. daily. Geriatric and adolescent patients should usually be started with lower dosage (25 to 50 mg. daily) and may not tolerate higher doses. Dosage may be increased up to 100 mg. daily. Lower maintenance dosages should be continued for at least 2 months after obtaining a satisfactory response. Therapy may be given in divided doses or as a single daily dose. **How Supplied:** 25 mg. capsules (pink), bottles of 100 and 1000. Also, 50 mg. capsules (maroon and pink), bottles of 100 and 1000.

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dual-action
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4 mg perphenazine and 25 mg amitriptyline HCl.
TRIAVIL® 4-10: Each tablet contains
4 mg perphenazine and 10 mg amitriptyline HCl.

CONTRAINDICATIONS: Central nervous system depression from drugs (barbiturates, alcohol, narcotics, analgesics, antihistamines); evidence of bone marrow depression; known hypersensitivity to phenothiazines or amitriptyline. Should not be given concomitantly with a monoamine oxidase inhibitor since hyperpyretic crises, severe convulsions, and deaths have occurred from such combinations. When used to replace a monoamine oxidase inhibitor, allow a minimum of 14 days to elapse before initiating therapy with TRIAVIL. Therapy should then be initiated cautiously with gradual increase in dosage until optimum response is achieved. Not recommended for use during acute recovery phase following myocardial infarction.

WARNINGS: TRIAVIL should not be given concomitantly with guanethidine or similarly acting compounds since TRIAVIL may block the antihypertensive action of such compounds. Use cautiously in patients with history of urinary retention, angle-closure glaucoma, increased intraocular pressure, or convulsive disorders. Dosage of anticonvulsive agents may have to be increased. In patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely. Tricyclic antidepressants, including amitriptyline HCl, have been reported to produce arrhythmias, sinus tachycardia, and prolongation of conduction time, particularly in high doses. Myocardial infarction and stroke have been reported with tricyclic antidepressant drugs. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. May impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle. In patients who use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage. Not recommended in children or during pregnancy.

PRECAUTIONS: Suicide is a possibility in depressed patients and may remain until significant remission occurs. Such patients should not have access to large quantities of this drug.

Perphenazine: Should not be used indiscriminately. Use with caution in patients who have previously exhibited severe adverse reactions to other phenothiazines. Likelihood of some untoward actions is greater with high doses. Closely supervise with any dosage. The antiemetic effect of perphenazine may obscure signs of toxicity due to overdosage of other drugs or make more difficult the diagnosis of disorders such as brain tumor or intestinal obstruction. A significant, not otherwise explained, rise in body temperature may suggest individual intolerance to perphenazine, in which case discontinue.

If hypotension develops, epinephrine should not be employed, as its action is blocked and partially reversed by perphenazine. Phenothiazines may potentiate the action of central nervous system depressants (opiates, analgesics, antihistamines, barbiturates, alcohol) and atropine. In concurrent therapy with any of these, TRIAVIL should be given in reduced dosage. May also potentiate the action of heat and phosphorous insecticides. There is sufficient experimental evidence to conclude that chronic administration of antipsychotic drugs which increase prolactin secretion has the potential to induce mammary neoplasms in rodents under the appropriate conditions. There are recognized differences in the physiological role of prolactin between rodents and humans. Since there are, at present, no adequate epidemiological studies, the relevance to human mammary cancer risk from prolonged exposure to perphenazine and other antipsychotic drugs is not known.

Amitriptyline: In manic-depressive psychosis, depressed patients may experience a shift toward the manic phase if they are treated with an antidepressant. Patients with paranoid symptomatology may have an exaggeration of such symptoms. The tranquilizing effect of TRIAVIL seems to reduce the likelihood of this effect. When amitriptyline HCl is given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required. Paralytic ileus may occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs.

Caution is advised if patients receive large doses of ethchlorvynol concurrently. Transient delirium has been reported in patients who were treated with 1 g of ethchlorvynol and 75-150 mg of amitriptyline HCl.

Amitriptyline HCl may enhance the response to alcohol and the effects of barbiturates and other CNS depressants.

Concurrent administration of amitriptyline HCl and electroshock therapy may increase the hazards associated with such therapy. Such treatment should be limited to patients for whom it is essential. Discontinue several days before elective surgery if possible. Elevation and lowering of blood sugar levels have both been reported. Use with caution in patients with impaired liver function.

ADVERSE REACTIONS: Similar to those reported with either constituent alone.
Perphenazine: Extrapyramidal symptoms (opisthotonus, oculogyric crisis, hyperreflexia, dystonia, akathisia, acute dyskinesia, ataxia, parkinsonism) have been reported and can usually be controlled by the concomitant use of effective antiparkinsonian drugs and/or by reduction in dosage, but sometimes persist after discontinuation of the phenothiazine.

Tardive dyskinesia may appear in some patients on long-term therapy or may occur after drug therapy with phenothiazines and related agents has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. Symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw. Involuntary movements of the extremities sometimes occur. There is no known treatment for tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms. It is advised that all antipsychotic agents be discontinued if the above symptoms appear. If treatment is reinstituted, or dosage of the particular drug increased, or another drug substituted, the syndrome may be masked. Fine vermicular movements of the tongue may be an early sign of the syndrome. The full-blown syndrome may not develop if medication is stopped when lingual vermiculation appears.

Other side effects are skin disorders (photosensitivity, itching, erythema, urticaria, eczema, up to exfoliative dermatitis); other allergic reactions (asthma, laryngeal edema, angioneurotic edema, anaphylactoid reactions); peripheral edema; reversed epinephrine effect; hyperglycemia; endocrine disturbances (lactation, galactorrhea, gynecomastia, disturbances of menstrual cycle); altered cerebrospinal fluid proteins; paradoxical excitement; hypertension, hypotension, tachycardia, and ECG abnormalities (quinidine-like effect); reactivation of psychotic processes; catatonic-like states; autonomic reactions, such as dry mouth or salivation, headache, anorexia, nausea, vomiting, constipation, obstipation, urinary frequency or incontinence, blurred vision, nasal congestion, and a change in pulse rate; other adverse reactions reported with various phenothiazine compounds, but not with perphenazine, include grand mal convulsions, cerebral edema, polyphagia, pigmentary retinopathy, photophobia, skin pigmentation, and failure of ejaculation.

The phenothiazine compounds have produced blood dyscrasias (pancytopenia, thrombocytopenic purpura, leukopenia, agranulocytosis, eosinophilia); and liver damage (jaundice, biliary stasis).

Pigmentation of the cornea and lens has been reported to occur after long-term administration of some phenothiazines. Although it has not been reported in patients receiving TRIAVIL, the possibility that it might occur should be considered.

Hypnotic effects, lassitude, muscle weakness, and mild insomnia have also been reported.

Amitriptyline: Note: Listing includes a few reactions not reported for this drug, but which have occurred with other pharmacologically similar tricyclic antidepressant drugs and must be considered when amitriptyline is administered. *Cardiovascular:* Hypotension; hypertension; tachycardia; palpitation; myocardial infarction arrhythmias; heart block; stroke. *CNS and Neuromuscular:* Confusional states disturbed concentration; disorientation; delusions; hallucinations; excitement anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth blurred vision; disturbance of accommodation; increased intraocular pressure constipation; paralytic ileus; urinary retention; dilatation of urinary tract. *Allergic* Skin rash; urticaria; photosensitization; edema of face and tongue. *Hematologic* Bone marrow depression including agranulocytosis; leukopenia; eosinophilia purpura; thrombocytopenia. *Gastrointestinal:* Nausea; epigastric distress; vomiting; anorexia; stomatitis; peculiar taste; diarrhea; parotid swelling; black tongue Rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido; elevated or lowered blood sugar levels. *Other:* Dizziness, weakness; fatigue; headache; weight gain or loss increased perspiration; urinary frequency; mydriasis; drowsiness; alopecia. *Withdrawal Symptoms:* Abrupt cessation after prolonged administration may produce nausea, headache, and malaise. These are not indicative of addiction.

OVERDOSAGE: All patients suspected of having taken an overdosage should be admitted to a hospital as soon as possible. Treatment is symptomatic and supportive. However, the intravenous administration of 1–3 mg of physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage of physostigmine should be repeated as required particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine. On this basis, in severe overdosage with perphenazine-amitriptyline combinations, symptomatic treatment of central anticholinergic effects with physostigmine salicylate should be considered.

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Brief Summary

ACTIONS — Pharmacologically, clorazepate dipotassium has the characteristics of the benzodiazepines. It has depressant effects on the central nervous system. The primary metabolite, nordiazepam, quickly appears in the blood stream. The serum half-life is about 2 days. The drug is metabolized in the liver and excreted primarily in the urine. (See CLINICAL PHARMACOLOGY section.)

INDICATIONS — TRANXENE is indicated for the symptomatic relief of anxiety associated with anxiety neurosis, in other psychoneuroses in which anxiety symptoms are prominent features, and as an adjunct in disease states in which anxiety is manifested.

TRANXENE is indicated for the symptomatic relief of acute alcohol withdrawal.

The effectiveness of TRANXENE in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should reassess periodically the usefulness of the drug for the individual patient.

CONTRAINDICATIONS — TRANXENE is contraindicated in patients with a known hypersensitivity to the drug, and in those with acute narrow angle glaucoma.

WARNINGS — TRANXENE is not recommended for use in depressive neuroses or in psychotic reactions.

Patients on TRANXENE should be cautioned against engaging in hazardous occupations requiring mental alertness, such as operating dangerous machinery including motor vehicles.

Since TRANXENE has a central nervous system depressant effect, patients should be advised against the simultaneous use of other CNS-depressant drugs, and cautioned that the effects of alcohol may be increased.

Because of the lack of sufficient clinical experience, TRANXENE is not recommended for use in patients less than 18 years of age.

Physical and Psychological Dependence:

Withdrawal symptoms (similar in character to those noted with barbiturates and alcohol) have occurred following abrupt discontinuance of clorazepate. Symptoms of nervousness, insomnia, irritability, diarrhea, muscle aches and memory impairment have followed abrupt withdrawal after long-term use of high dosage.

Caution should be observed in patients who are considered to have a psychological potential for drug dependence.

Evidence of drug dependence has been observed in dogs and rabbits which was characterized by convulsive seizures when the drug was abruptly withdrawn or the dose was reduced; the syndrome in dogs could be abolished by administration of clorazepate.

Usage in Pregnancy:

An increased risk of congenital malformations associated with the use of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) during the first trimester of pregnancy has been suggested in several studies. TRANXENE, a benzodiazepine derivative, has not been studied adequately to determine whether it, too, may be associated with an increased risk of fetal abnormality. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physician about the desirability of discontinuing the drug.

Usage during Lactation:

TRANXENE should not be given to nursing mothers since it has been reported that nordiazepam is excreted in human breast milk.

PRECAUTIONS — In those patients in which a degree of depression accompanies the anxiety, suicidal tendencies may be present and protective measures may be required. The least amount of drug that is feasible should be available to the patient.

Patients on TRANXENE for prolonged periods should have blood counts and liver function tests periodically. The usual precautions in treating patients with impaired renal or hepatic function should also be observed.

In elderly or debilitated patients, the initial dose should be small, and increments should be made gradually, in accordance with the response of the patient, to preclude ataxia or excessive sedation.

ADVERSE REACTIONS — The side effect most frequently reported was drowsiness. Less commonly reported (in descending order of occurrence) were: dizziness, various gastrointestinal complaints, nervousness, blurred vision, dry mouth, headache, and mental confusion. Other side effects included insomnia, transient skin rashes, fatigue, ataxia, genitourinary complaints, irritability, diplopia, depression and slurred speech.

There have been reports of abnormal liver and kidney function tests and of decrease in hematocrit.

Decrease in systolic blood pressure has been observed.

DOSAGE AND ADMINISTRATION

For the symptomatic relief of anxiety:

TRANXENE is administered orally. The capsules may be given in divided doses. The usual daily dose is 30 mg. The dose should be adjusted gradually within the range of 15 to 60 mg. daily in accordance with the response of the patient. In elderly or debilitated patients it is advisable to initiate treatment at a daily dose of 7.5 to 15 mg.

TRANXENE capsules may also be administered as a single dose daily at bedtime; the recommended initial dose is 15 mg. After the initial dose, the response of the patient may require adjustment of subsequent dosage. Lower doses may be indicated in the elderly patient. Drowsiness may occur at the initiation of treatment and with dosage increment.

TRANXENE-SD tablets (22.5 mg.) may be administered as a single dose every 24 hours. This tablet is intended as an alternate dosage form for the convenience of patients stabilized on a dose of 7.5 mg. capsules three times a day. TRANXENE-SD tablets should not be used to initiate therapy.

TRANXENE-SD HALF STRENGTH tablets (11.25 mg.) may be administered as a single dose every 24 hours.

For the symptomatic relief of acute alcohol withdrawal:

Recommended schedule: 1st 24 hours, 30 mg. TRANXENE initially, followed by 30 to 60 mg. in divided doses; 2nd 24 hours, 45 to 90 mg. in divided doses; 3rd 24 hours, 22.5 to 45 mg. in divided doses; 4th day, 15 to 30 mg. in divided doses. Thereafter gradually reduce to 7.5 to 15 mg. daily, and discontinue as soon as condition is stable. Maximum daily dose is 90 mg. Avoid excessive reductions in total drug on successive days.

DRUG INTERACTIONS — If TRANXENE is to be combined with other drugs acting on the central nervous system, careful consideration should be given to the pharmacology of the agents to be employed. Animal experience indicates that TRANXENE prolongs the sleeping time after hexobarbital or after ethyl alcohol, increases the inhibitory effects of chlorpromazine, but does not exhibit monoamine oxidase inhibition. Clinical studies have shown increased sedation with concurrent hypnotic medications. The actions of the benzodiazepines may be potentiated by barbiturates, narcotics, phenothiazines, monoamine oxidase inhibitors or other antidepressants.

If TRANXENE is used to treat anxiety associated with somatic disease states, careful attention must be paid to possible drug interaction with concomitant medication.

MANAGEMENT OF OVERDOSAGE — Overdosage is usually manifested by varying degrees of CNS depression ranging from slight sedation to coma. As in the management of overdosage with any drug, it should be borne in mind that multiple agents may have been taken.

There are no specific antidotes for the benzodiazepines. The treatment of overdosage should consist of the general measures employed in the management of overdosage of any CNS depressant. Gastric evacuation either by the induction of emesis, lavage, or both, should be performed immediately. General supportive care, including frequent monitoring of the vital signs and close observation of the patient, is indicated. Hypotension, though rarely reported, may occur with large overdoses. In such cases the use of agents such as Levophed® (levarterenol) or Aramine® (metaraminol) should be considered.

While reports indicate that individuals have survived overdoses of TRANXENE (clorazepate dipotassium) as high as 450 to 675 mg., these doses are not necessarily an accurate indication of the amount of drug absorbed since the time interval between ingestion and the institution of treatment was not always known. Sedation in varying degrees was the most common physiological manifestation of TRANXENE overdosage. Deep coma when it occurred was usually associated with the ingestion of other drugs in addition to TRANXENE.

CLINICAL PHARMACOLOGY — Studies in healthy men have shown that TRANXENE has depressant effects on the central nervous system. Prolonged administration of single daily doses as high as 120 mg. was without toxic effects. Abrupt cessation of high doses was followed in some patients by nervousness, insomnia, irritability, diarrhea, muscle aches, or memory impairment.

Absorption — Excretion:

After oral administration of TRANXENE, there is essentially no circulating parent drug. Nordiazepam, its primary metabolite, quickly appears in the blood stream. In 2 volunteers given 15 mg. (50 μC) of ^{14}C-TRANXENE, about 80% was recovered in the urine and feces within 10 days. Excretion was primarily in the urine with about 1% excreted per day on day 10.

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Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

Effective: For the management of the manifestations of psychotic disorders.

Possibly effective: To control excessive anxiety, tension and agitation as seen in neuroses or associated with somatic conditions.

'Stelazine' has not been shown effective in the management of behavioral complications in patients with mental retardation.

Final classification of the less-than-effective indications requires further investigation.

Contraindications: Comatose or greatly depressed states due to C.N.S. depressants; blood dyscrasias; bone marrow depression; liver damage.

Warnings: Generally avoid using in patients hypersensitive (e.g., blood dyscrasias, jaundice) to any phenothiazine. Caution patients about activities requiring alertness (e.g., operating vehicles or machinery), especially during the first few days' therapy.

Use in pregnancy only when necessary for patient's welfare.

Precautions: Use cautiously in angina. Avoid high doses and parenteral administration when cardiovascular system is impaired. Antiemetic effect may mask signs of toxic drug overdosage or physical disorders. Additive effect is possible with other C.N.S. depressants. Prolonged administration of high doses may result in cumulative effects with severe C.N.S. or vasomotor symptoms. If retinal changes occur, discontinue drug. Agranulocytosis, thrombocytopenia, pancytopenia, anemia, cholestatic jaundice, liver damage have been reported.

Patients on long-term therapy, especially high doses, should be evaluated periodically for possible adjustment or discontinuance of drug therapy.

Adverse Reactions: Drowsiness, dizziness, skin reactions, rash, dry mouth, insomnia, amenorrhea, fatigue, muscular weakness, anorexia, lactation, blurred vision. Neuromuscular (extrapyramidal) reactions: motor restlessness, dystonias, pseudo-parkinsonism, persistent tardive dyskinesia.

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Prepared by the APA Committee on History, Library, and Museum; Daniel Blain, M.D. and Michael Barton, Ph.D.

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Relating Environment to Mental Health and Illness: The Ecopsychiatric Base, Task Force Report 16

Prepared by the APA Task Force on the Ecopsychiatric Data Base; Jay T. Shurley, M.D., Chairperson

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Ethical as well as legal considerations require careful attention to the protection of a patient's anonymity in case reports and elsewhere. Identifying information such as names, initials, hospital numbers, and dates must be avoided. In addition, authors should disguise identifying information about a patient's characteristics and personal history.

Informed Consent

Manuscripts that report the results of experimental investigation with human subjects must include a statement that informed consent was obtained after the procedure(s) had been fully explained.

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Papers are reviewed by at least two experts to determine the originality, validity, and significance of content and conclusions. Authors will usually be advised within 2–3 months of the decision on their paper, although delays are sometimes unavoidable. Reviewers' comments will be returned with rejected manuscripts if they are judged to be useful to the authors. All reviewers remain anonymous.

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Manuscripts should be submitted in triplicate to John C. Nemiah, M.D., Editor, *American Journal of Psychiatry*, 1700 Eighteenth St., N.W., Washington, D.C. 20009. All correspondence will be sent to the first-named author unless otherwise specified. Papers that are not accompanied by the appropriate cover letter, including the copyright transfer statement, will not be reviewed until such a statement is received.

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The *Journal* has first refusal rights for numbered papers accepted for presentation at the Association's annual meetings. Authors of annual meeting papers should follow instructions they will receive in a letter from the Editor before the meeting. Annual meeting papers may be submitted for review before the meeting only if they are in *final form*, i.e., are ready for review. Authors must *not* submit their papers before the meeting if they expect any substantive revisions due to discussion at the meeting, further research, etc. These papers are subject to the same peer review criteria as other submissions, and not all papers can be published in the *Journal*. Authors who wish to submit their papers elsewhere must secure permission from the Editor. The *Journal* will assign its own number to each paper received for review; this number must appear in any further communications about the paper. The copyright transfer statement described above must accompany annual meeting papers submitted for review.

Length

The length of submitted material should not exceed the following specifications unless a special arrangement has been made with the Editor. Regular articles—3,800 words or the equivalent, including references, tables, and figures (about 15 manuscript pages). Brief Communications—2,500 words or equivalent (about 10 manuscript pages). Clinical and Research Reports—1,000 words, 10 references, 1 table (no figures can be used in this section). Letters to the Editor—500 words, 5 references. The number of words, tables, and figures should be noted on the title page.

TYPES OF ARTICLES

Overviews

Overview articles attempt to bring together important relevant information on a topic of general interest to psychiatry. They are usually written at the invitation of the Editor; authors who have ideas for overview articles are advised to check with the Editor to ensure that a similar work is not in preparation. Overviews should not exceed 25 double-spaced pages (about 7,500 words) and should have no more than 100 references. All overviews, including those written by invitation, are given the same peer review received by other papers.

Regular Articles and Brief Communications

The primary difference between these two types of papers is length. The "brief communication" designation does not imply less sophisticated or complete work: it merely means that the content can be expressed within the upper limit of 2,500 words. There is no difference in the review procedure or scheduling of these articles. Authors who submit unnecessarily long articles with redundancies and loose writing style will be asked to shorten them, which can delay publication. Authors should choose their words carefully, avoiding lengthy introductions containing material that is common knowledge, summaries that merely repeat the results, unnecessary tables, figures or references, etc. Single case reports will not be accepted as regular articles or Brief Communications; these papers should be submitted as Clinical and Research Reports.

Clinical and Research Reports

This section contains very brief articles (maximum of 1,000 words, 10 references, 1 table, and *no figures*) reporting new research findings, including preliminary data from pilot

studies, and case reports that 1) describe new syndromes, 2) cast a new light on established ones, 3) indicate a new therapeutic procedure of potential value, or 4) describe adverse effects or previously unreported complications of drugs or therapeutic interventions. Because of the stringent criteria for this section, authors can expect more rapid publication than is possible in other sections. Submitted papers that substantially exceed the stated maximum length or contain figures will be returned to the author unreviewed.

Prompt Publication Policy

Articles submitted for prompt publication can be of either regular or Brief Communication length (a "prompt publication policy" is automatically in effect for Clinical and Research Reports). These papers are given priority in scheduling; however, authors should be aware that the minimum publication time is four months. Prompt publication papers must meet stringent criteria of originality and be of major, immediate importance to the field. Authors must state their reasons for wanting rapid review in a cover letter to the Editor. It is important to think carefully about the nature of the paper before requesting prompt publication; a paper may be delayed if it is submitted inappropriately.

Other Sections

Letters to the Editor. Brief letters (maximum of 500 words and 5 references) will be considered if they include the notation "for publication" in the upper right corner. Letters critical of an article published in the *Journal* will automatically be sent to the authors for reply. The *Journal* is unable to notify authors of receipt of letters or to return those not published. All letters are subject to editing. Letters must be typed *double-spaced* throughout on letter-size bond paper; two copies are required. Letters that are not typed appropriately or do not conform to *Journal* reference style will be returned to the author for revision.

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All manuscripts must be typed in upper- and lowercase on one side only of 22 × 28 cm (8½ × 11 inches) *nonerasable* bond paper. All parts of the manuscript, including case reports, quotations, references, and tables, must be double-spaced throughout. All 4 margins must be 3¾ cm (1½ inches). The manuscript should be arranged in the following order, with each numbered item beginning a new page: 1) title page, 2) précis, 3) text, 4) references, 5) tables, 6) footnotes to text, and 7) figure captions. All pages must be numbered, with the title page as number 1. Please consult the following section on *Journal* style specifications for criteria for each part of the paper.

STYLE SPECIFICATIONS

Title Page

Title. The title should be informative but as brief as possible. Headline style (declarative sentences) should be avoided. Abbreviations and acronyms are rarely used.

By-line. Authors named in the by-line should be limited to principal researchers and/or writers; collaborators can be acknowledged in a footnote. Please include degrees (other than honorary or undergraduate) after the authors' names.

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Other requirements. The number of words, tables, and figures should appear in the upper right corner and a phone number for the corresponding author in the lower right.

Précis

The précis should be up to 100 words for regular articles and Brief Communications (no précis is used in the Clinical and Research Reports section). The précis should be a single paragraph using complete, connected sentences, active verbs, and the third person. In most cases, the précis replaces a summary.

Text

Headings and subheadings should be inserted at reasonable intervals in all types of papers. Footnotes to text material should be typed on a separate page at the end of the manuscript. Summaries are rarely desirable, although a final "Comment" or "Conclusions" section may be used. The body of the paper should be written in the active voice and first person where appropriate.

Statistics. Authors should be sure that statistics used are both necessary and appropriate. Statistical tests should be described and a reference citation given if the tests are not generally known. It is not sufficient to say "differences between the groups were significant." In cases of lengthy complicated analyses, the authors may summarize results and indicate that details are available from them on request.

Abbreviations. All abbreviations (other than those for metric units) should be explained the first time they are used. Idiosyncratic abbreviations should be avoided: overuse of abbreviations may hinder rather than facilitate readability.

Drugs. Generic rather than trade names of drugs should be used; trade names may be given parenthetically if necessary.

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References should be restricted to closely pertinent material; a complete review of the literature is rarely desirable. *Accuracy of citation is the author's responsibility.* References should conform exactly to the original spelling, accents, punctuation, etc. Authors should be sure that all references listed have been cited in text; no bibliographies can be used.

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Type references in the style shown below and on the next page, double-spaced *throughout* (not just a line between

references). List up to three authors; designate one or more authors past the third "et al." Abbreviations of journal names should conform to the style used in *Index Medicus*; journals not indexed there should not be abbreviated.

1. Berne E: Principles of Group Treatment. New York, Oxford University Press, 1966, p 26
2. Blackwell B, Marley E, Price J, et al: Hypertensive interactions between monoamine oxidase inhibitors and foodstuffs. Br J Psychiatry 113:349-365, 1967
3. Gold PW, Goodwin FK: Urinary free cortisol in depression and mania. Presented at the 130th annual meeting of the American Psychiatric Association, Toronto, Ont, Canada, May 2-6, 1977
4. Brosin H: Communication systems of the consultation process, in The Psychiatric Consultation. Edited by Mendel W, Solomon P. New York, Grune & Stratton, 1968

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Tables should be self-explanatory and should supplement rather than duplicate text. Authors may be asked to delete tables that present data which could be given succinctly in text or repeat information in the Results section. Tables are generally reserved for data presentation and not used as lists or charts. They should be no wider than 120 typewriter characters, including spaces. Consult recent issues of the *Journal* for table style. Please note that no vertical rules or internal horizontal rules are used. It is usually desirable to give both numbers and percentages where applicable. All units of measurement must be specified within the table. Metric measurements are used in tables and throughout the text. Each table should be identified numerically and include a concise title.

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Figures should be used only for data that cannot be adequately presented in text or tabular form. Figures should be submitted as glossy prints with one photocopy attached to each manuscript copy. The author's name and the title of the paper should appear on a gummed label affixed to the back of each glossy. All figures should be able to withstand reduction to about 8 cm (3¼ inches). Authors are urged to consider carefully the necessity of figures; they may be asked to delete figures that repeat information available in text. It is often advisable to obtain professional assistance in the preparation of figures.

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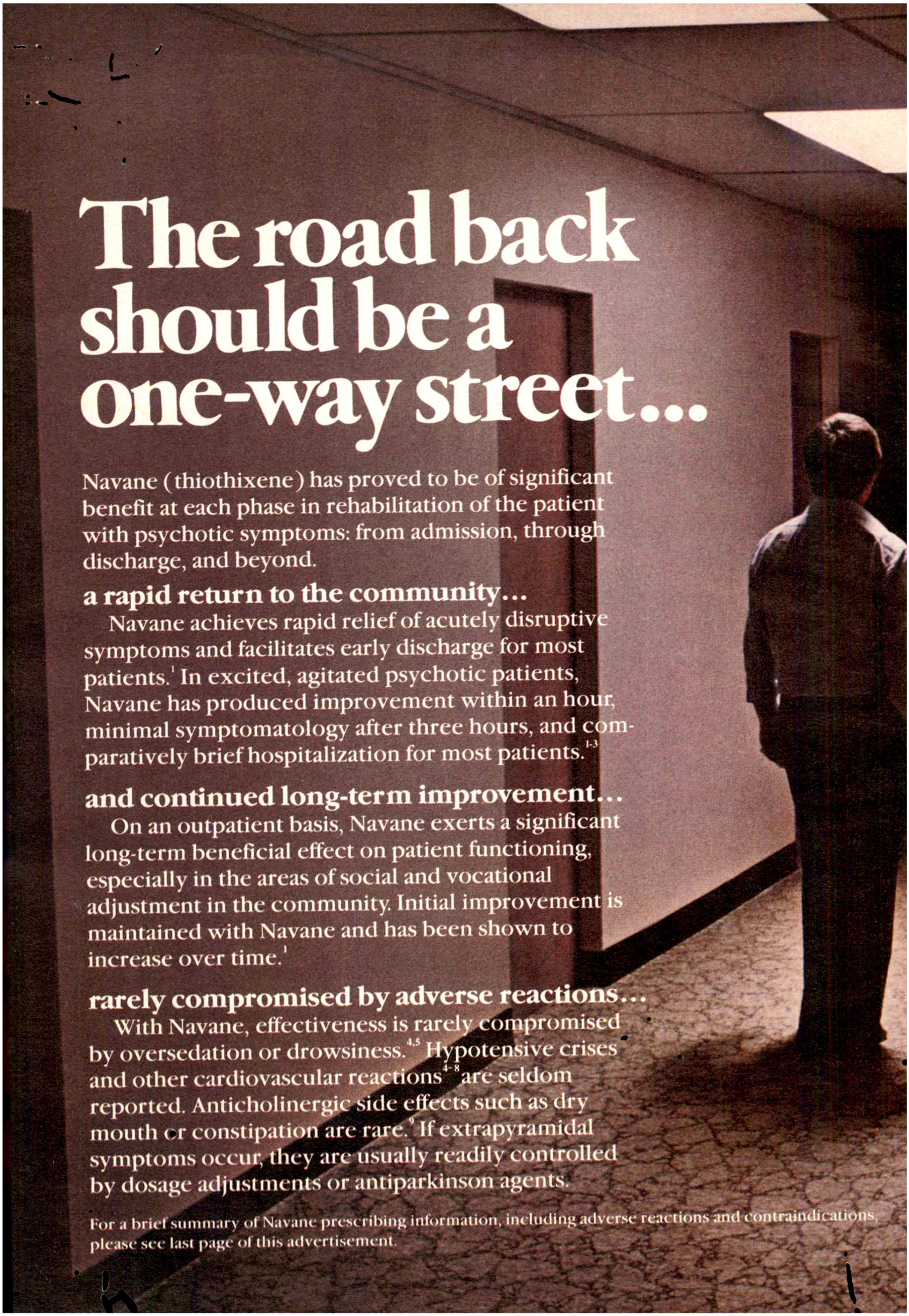
The July 1979 issue of

The American Journal of Psychiatry

will feature

- **Time, Age, and the Life Cycle**

  By Bernice L. Neugarten



The road back should be a one-way street...

Navane (thiothixene) has proved to be of significant benefit at each phase in rehabilitation of the patient with psychotic symptoms: from admission, through discharge, and beyond.

a rapid return to the community...

Navane achieves rapid relief of acutely disruptive symptoms and facilitates early discharge for most patients.[1] In excited, agitated psychotic patients, Navane has produced improvement within an hour, minimal symptomatology after three hours, and comparatively brief hospitalization for most patients.[1-3]

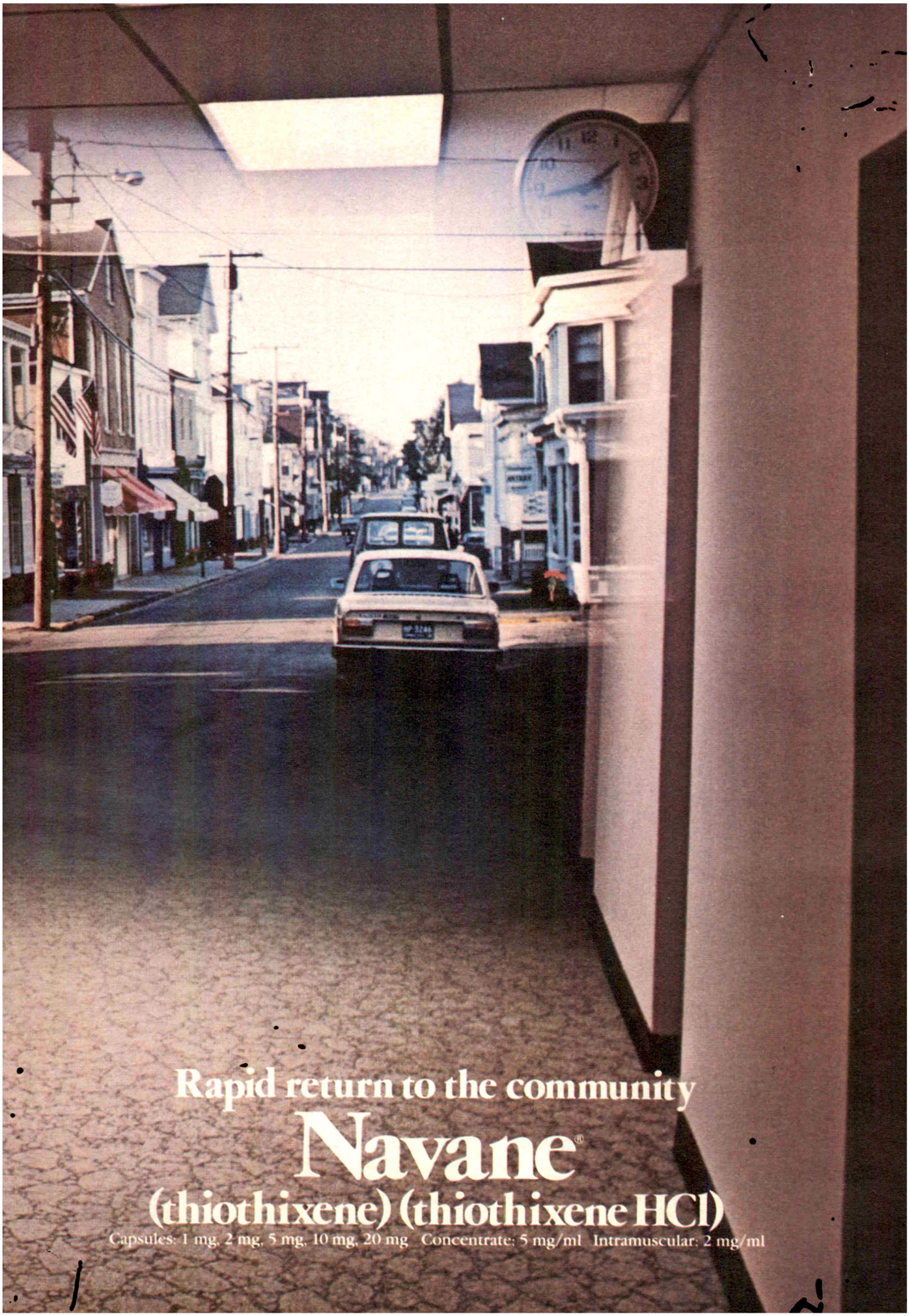
and continued long-term improvement...

On an outpatient basis, Navane exerts a significant long-term beneficial effect on patient functioning, especially in the areas of social and vocational adjustment in the community. Initial improvement is maintained with Navane and has been shown to increase over time.[1]

rarely compromised by adverse reactions...

With Navane, effectiveness is rarely compromised by oversedation or drowsiness.[4,5] Hypotensive crises and other cardiovascular reactions[4-8] are seldom reported. Anticholinergic side effects such as dry mouth or constipation are rare.[9] If extrapyramidal symptoms occur, they are usually readily controlled by dosage adjustments or antiparkinson agents.

For a brief summary of Navane prescribing information, including adverse reactions and contraindications, please see last page of this advertisement.


Rapid return to the community
Navane®
(thiothixene) (thiothixene HCl)
Capsules: 1 mg, 2 mg, 5 mg, 10 mg, 20 mg Concentrate: 5 mg/ml Intramuscular: 2 mg/ml

Rapid return to the community

Navane® (thiothixene) (thiothixene hydrochloride)

Capsules: 1 mg, 2 mg, 5 mg, 10 mg, 20 mg Concentrate: 5 mg/ml Intramuscular: 2 mg/ml

BRIEF SUMMARY OF PRESCRIBING INFORMATION
Navane® (thiothixene)
Capsules: 1 mg, 2 mg, 5 mg, 10 mg, 20 mg (thiothixene hydrochloride) Concentrate: 5 mg/ml, Intramuscular: 2 mg/ml

Contraindications. Navane (thiothixene) is contraindicated in patients with circulatory collapse, comatose states, central nervous system depression due to any cause, and blood dyscrasias. Navane is contraindicated in individuals who have shown hypersensitivity to the drug. It is not known whether there is a cross-sensitivity between the thioxanthenes and the phenothiazine derivatives, but this possibility should be considered.

Warnings. *Usage in Pregnancy*– Safe use of Navane during pregnancy has not been established. Therefore, this drug should be given to pregnant patients only when, in the judgment of the physician, the expected benefits from the treatment exceed the possible risks to mother and fetus. Animal reproduction studies and clinical experience to date have not demonstrated any teratogenic effects.

In the animal reproduction studies with Navane, there was some decrease in conception rate and litter size, and an increase in resorption rate in rats and rabbits, changes which have been similarly reported with other psychotropic agents. After repeated oral administration of Navane to rats (5 to 15 mg/kg/day), rabbits (3 to 50 mg/kg/day), and monkeys (1 to 3 mg/kg/day) before and during gestation, no teratogenic effects were seen. (See Precautions.)

Usage in Children– The use of Navane in children under 12 years of age is not recommended because safety and efficacy in the pediatric age group have not been established.

As is true with many CNS drugs, Navane may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery, especially during the first few days of therapy. Therefore, the patient should be cautioned accordingly.

As in the case of other CNS-acting drugs, patients receiving Navane should be cautioned about the possible additive effects (which may include hypotension) with CNS depressants and with alcohol.

Precautions. An antiemetic effect was observed in animal studies with Navane; since this effect may also occur in man, it is possible that Navane may mask signs of overdosage of toxic drugs and may obscure conditions such as intestinal obstruction and brain tumor.

In consideration of the known capability of Navane and certain other psychotropic drugs to precipitate convulsions, extreme caution should be used in patients with a history of convulsive disorders or those in a state of alcohol withdrawal since it may lower the convulsive threshold. Although Navane potentiates the actions of the barbiturates, the dosage of the anticonvulsant therapy should not be reduced when Navane is administered concurrently.

Caution as well as careful adjustment of the dosage is indicated when Navane is used in conjunction with other CNS depressants other than anticonvulsant drugs.

Though exhibiting rather weak anticholinergic properties, Navane should be used with caution in patients who are known or suspected to have glaucoma, or who might be exposed to extreme heat, or who are receiving atropine or related drugs.

Use with caution in patients with cardiovascular disease.

Also, careful observation should be made for pigmentary retinopathy, and lenticular pigmentation (fine lenticular pigmentation has been noted in a small number of patients treated with Navane for prolonged periods). Blood dyscrasias (agranulocytosis, pancytopenia, thrombocytopenic purpura), and liver damage (jaundice, biliary stasis) have been reported with related drugs.

Undue exposure to sunlight should be avoided. Photosensitive reactions have been reported in patients on Navane.

Intramuscular Administration– As with all intramuscular preparations, Navane Intramuscular should be injected well within the body of a relatively large muscle. The preferred sites are the upper outer quadrant of the buttock (i.e., gluteus maximus) and the mid-lateral thigh.

The deltoid area should be used only if well developed, such as in certain adults and older children, and then only with caution to avoid radial nerve injury. Intramuscular injections should not be made into the lower and mid-thirds of the upper arm. As with all intramuscular injections, aspiration is necessary to help avoid inadvertent injection into a blood vessel.

Adverse Reactions. *Note:* Not all of the following adverse reactions have been reported with Navane (thiothixene). However, since Navane has certain chemical and pharmacologic similarities to the phenothiazines, all of the known side effects and toxicity associated with phenothiazine therapy should be borne in mind when Navane is used.

Cardiovascular effects: Tachycardia, hypotension, lightheadedness, and syncope. In the event hypotension occurs, epinephrine should not be used as a pressor agent since a paradoxical further lowering of blood pressure may result. Nonspecific EKG changes have been observed in some patients receiving Navane. These changes are usually reversible and frequently disappear on continued Navane therapy. The incidence of these changes is lower than that observed with some phenothiazines. The clinical significance of these changes is not known.

CNS effects: Drowsiness, usually mild, may occur although it usually subsides with continuation of Navane therapy. The incidence of sedation appears similar to that of the piperazine group of phenothiazines, but less than that of certain aliphatic phenothiazines. Restlessness, agitation and insomnia have been noted with Navane (thiothixene). Seizures and paradoxical exacerbation of psychotic symptoms have occurred with Navane infrequently.

Hyperreflexia has been reported in infants delivered from mothers having received structurally related drugs.

In addition, phenothiazine derivatives have been associated with cerebral edema and cerebrospinal fluid abnormalities.

Extrapyramidal symptoms, such as pseudo-parkinsonism, akathisia, and dystonia have been reported. Management of these extrapyramidal symptoms depends upon the type and severity. Rapid relief of acute symptoms may require the use of an injectable antiparkinson agent. More slowly emerging symptoms may be managed by reducing the dosage of Navane and/or administering an oral antiparkinson agent.

Persistent Tardive Dyskinesia: As with all antipsychotic agents tardive dyskinesia may appear in some patients on long term therapy or may occur after drug therapy has been discontinued. The risk seems to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities.

There is no known effective treatment for tardive dyskinesia: antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear.

Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked.

It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time, the syndrome may not develop.

Hepatic effects: Elevations of serum transaminase and alkaline phosphatase, usually transient, have been infrequently observed in some patients. No clinically confirmed cases of jaundice attributable to Navane have been reported.

Hematologic effects: As is true with certain other psychotropic drugs, leukopenia and leukocytosis, which are usually transient, can occur occasionally with Navane. Other antipsychotic drugs have been associated with agranulocytosis, eosinophilia, hemolytic anemia, thrombocytopenia and pancytopenia.

Allergic reactions: Rash, pruritus, urticaria, photosensitivity and rare cases of anaphylaxis have been reported with Navane. Undue exposure to sunlight should be avoided. Although not experienced with Navane, exfoliative dermatitis and contact dermatitis (in nursing personnel) have been reported with certain phenothiazines.

Endocrine disorders: Lactation, moderate breast enlargement and amenorrhea have occurred in a small percentage of females receiving Navane. If persistent, this may necessitate a reduction in dosage or the discontinuation of therapy. Phenothiazines have been associated with false positive pregnancy tests, gynecomastia, hypoglycemia, hyperglycemia, and glycosuria.

Autonomic effects: Dry mouth, blurred vision, nasal congestion, constipation, increased sweating, increased salivation, and impotence have occurred infrequently with Navane therapy. Phenothiazines have been associated with miosis, mydriasis, and adynamic ileus.

Other adverse reactions: Hyperpyrexia, anorexia, nausea, vomiting, diarrhea, increase in appetite and weight, weakness or fatigue, polydipsia and peripheral edema.

Although not reported with Navane, evidence indicates there is a relationship between phenothiazine therapy and the occurrence of a systemic lupus erythematosus-like syndrome.

NOTE: Sudden deaths have occasionally been reported in patients who have received certain phenothiazine derivatives. In some cases the cause of death was apparently cardiac arrest or asphyxia due to failure of the cough reflex. In others, the cause could not be determined nor could it be established that death was due to phenothiazine administration.

Dosage and Administration. Dosage of Navane should be individually adjusted depending on the chronicity and severity of the condition. In general, small doses should be used initially and gradually increased to the optimal effective level, based on patient response.

Some patients have been successfully maintained on once-a-day Navane therapy.

Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

Navane Intramuscular Solution–For Intramuscular Use Only. Where more rapid control and treatment of acute behavior is desirable, the intramuscular form of Navane may be indicated. It is also of benefit where the very nature of the patient's symptomatology, whether acute or chronic, renders oral administration impractical or even impossible.

For treatment of acute symptomatology or in patients unable or unwilling to take oral medication, the usual dose is 4 mg of Navane Intramuscular administered 2 to 4 times daily. Dosage may be increased or decreased depending on response. Most patients are controlled on a total daily dosage of 16 to 20 mg. The maximum recommended dosage is 30 mg/day. An oral form should supplant the injectable form as soon as possible. It may be necessary to adjust the dosage when changing from the intramuscular to oral dosage forms. Dosage recommendations for Navane (thiothixene) Capsules and Concentrate appear in the following paragraphs.

Navane Capsules; Navane Concentrate– In milder conditions, an initial dose of 2 mg three times daily. If indicated, a subsequent increase to 15 mg/day total daily dose is often effective.

In more severe conditions, an initial dose of 5 mg twice daily.

The usual optimal dose is 20 to 30 mg daily. If indicated, an increase to 60 mg/day total daily dose is often effective. Exceeding a total daily dose of 60 mg rarely increases the beneficial response.

Overdosage. Manifestations include muscular twitching, drowsiness, and dizziness. Symptoms of gross overdosage may include CNS depression, rigidity, weakness, torticollis, tremor, salivation, dysphagia, hypotension, disturbances of gait, or coma.

Treatment: Essentially symptomatic and supportive. For Navane oral, early gastric lavage is helpful. For Navane oral and Intramuscular, keep patient under careful observation and maintain an open airway, since involvement of the extrapyramidal system may produce dysphagia and respiratory difficulty in severe overdosage. If hypotension occurs, the standard measures for managing circulatory shock should be used (I.V. fluids and/or vasoconstrictors).

If a vasoconstrictor is needed, levarterenol and phenylephrine are the most suitable drugs. Other pressor agents, including epinephrine, are not recommended, since phenothiazine derivatives may reverse the usual pressor action of these agents and cause further lowering of blood pressure.

If CNS depression is present, recommended stimulants include amphetamine, dextroamphetamine, or caffeine and sodium benzoate. Stimulants that may cause convulsions (e.g. picrotoxin or pentylenetetrazol) should be avoided. Extrapyramidal symptoms may be treated with antiparkinson drugs.

There are no data on the use of peritoneal or hemodialysis, but they are known to be of little value in phenothiazine intoxication.

How Supplied. Navane (thiothixene) is available as capsules containing 1 mg, 2 mg, 5 mg, and 10 mg of thiothixene in bottles of 100, 1,000, and unit-dose pack of 100 (10 x 10's). Navane is also available as capsules containing 20 mg of thiothixene in bottles of 100, 500, and unit-dose pack of 100 (10 x 10's).

Navane (thiothixene hydrochloride) Concentrate is available in 120 ml (4 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, 5 mg, 6 mg, 8 mg, and 10 mg, and in 30 ml (1 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, and 5 mg. Each ml contains thiothixene hydrochloride equivalent to 5 mg of thiothixene. Contains alcohol, U.S.P. 7.0% v/v (small loss unavoidable).

Navane (thiothixene hydrochloride) Intramuscular solution is available in a 2 ml amber glass vial in packages of 10 vials. Each ml contains thiothixene hydrochloride equivalent to 2 mg of thiothixene, dextrose 5% w/v, benzyl alcohol 0.9% w/v, and propyl gallate 0.02% w/v.

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For additional information on Navane, please consult your Roerig representative or write to: Roerig Medical Department, 235 East 42nd Street, New York, NY 10017.

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EARLY RECOLLECTIONS: Their Use in Diagnosis and Psychotherapy *compiled and edited by* **Harry A. Olson,** *Private Practice, Reisterstown, Maryland. Foreword by* **Raymond J. Corsini.** *(34 Contributors)* This book is both an exhaustive compendium of the literature and an up-to-date manual for the use of early recollections in diagnosis and psychotherapy. Contributions include specific techniques for the use of early recollections with children, to reduce unwanted feelings of clients, and to enhance vocational counseling. Retrieval and use of early recollections under hypnosis are also examined. A presentation of the complete Manaster-Perryman scoring manual is featured. *'79, 400 pp., 4 il., 10 tables, $27.50*

LAW, PSYCHOLOGY, AND THE COURTS: Rethinking Treatment of the Young and the Disturbed *by* **Ellsworth A. Fersch Jr.,** *Harvard Univ., Cambridge, Massachusetts.* Herein, the author provides authoritative, thought-provoking discussions on such topics as punishing young criminals, awarding of custody and facing ethical issues in court settings. Chapters are also included on handling young criminals and treatment in the court clinic setting. Readers will discover new ideas and perspectives in this book that will help them with professional pursuits related to law, psychology and the courts. *'79, 184 pp., 2 tables, $14.75*

CLASSROOM MANAGEMENT: A Guide for the School Consultant *by* **Jack Tanaka,** *Institute for Juvenile Research, Chicago.* Written from a social learning perspective, this book addresses school consultation considerations helpful in classroom management of problem student behaviors. Initial chapters define the role of the consultant, outline negotiations with teachers and principals, and review relevant behavior concepts. Practical applications of behavioral principles follow using actual and potential consultation experiences, a point system, and basic problem-solving techniques. The final segment details data on current situations, personality variables, and affective components. *'79, 240 pp., $14.75*

CASE STUDIES IN CLINICAL AND SCHOOL PSYCHOLOGY *by* **Ralph F. Blanco** *and* **Joseph G. Rosenfeld,** *both of Temple Univ., Philadelphia, Pennsylvania.* The fifteen case studies presented encompass behavior and emotional problems, learning disabilities, sensory and multiple handicaps, and giftedness. The authors outline symptoms, etiology, treatments and follow-up. Workbook sections permit psychology trainees to sharpen diagnostic skills and create prescriptive interventions, and to score and interpret responses on a variety of psychological tests. The authors' treatments are listed separately for comparison. These treatments include psychotherapy, IEP strategies, desensitization, extinction, and others. *'78, 256 pp., 4 il., $11.50*

LEISURE COUNSELING: An Aspect of Leisure Education *edited by* **Arlin Epperson,** *Univ. of Ottawa, Ottawa, Ontario, Canada; and* **Gerald Hitzhusen,** *Univ. of Missouri, Columbia. (31 Contributors)* Leisure counseling, as one specific aspect of the overall leisure education process, is the focal point of this volume. While institutional or rehabilitation leisure counseling has dominated the field in the past, the authors see future counseling expanding into community settings and a wider range of client groups. The contributors identify needs, examine priorities, and draft implications for education and research. *'77, 392 pp., 16 il., 22 tables, $20.75*

THE COUNSELOR AND SUICIDAL CRISIS: Diagnosis and Intervention *by* **John L. Hipple** *and* **Peter Cimbolic,** *both of North Texas State Univ., Denton.* The discussions in this book will help the professional counselor provide quality treatment for the actively suicidal client and his or her family. Chapters offer detailed information on determining the depth of depression, estimating lethality, providing protection during crisis, assisting in identification and resolution of conflicts, and terminating treatment appropriately. Also presented are ideas on use of the telephone as an adjunct to face-to-face counseling. *'79, 128 pp., $9.75*

THE THEORY AND TECHNIQUE OF FAMILY THERAPY *by* **Charles P. Barnard,** *Univ. of Wisconsin, Menomonie, and* **Ramon Garrido Corrales,** *Counseling Center for Human Development, Kansas City, Missouri.* A well-balanced blend of both therapy and practical application, this book provides a comprehensive overview of the theoretical constructs in the field of family therapy. The reader will find a tremendous variety of sources and orientations, topics ranging from the roots of human motivation and family development to specific intervention techniques and didactic aids. Techniques for intervention in a variety of circumstances are examined, including such subjects as premarital counseling, divorce counseling, alcoholism, and death and the family. *'79, 336 pp., 13 il., 1 table, $14.75*

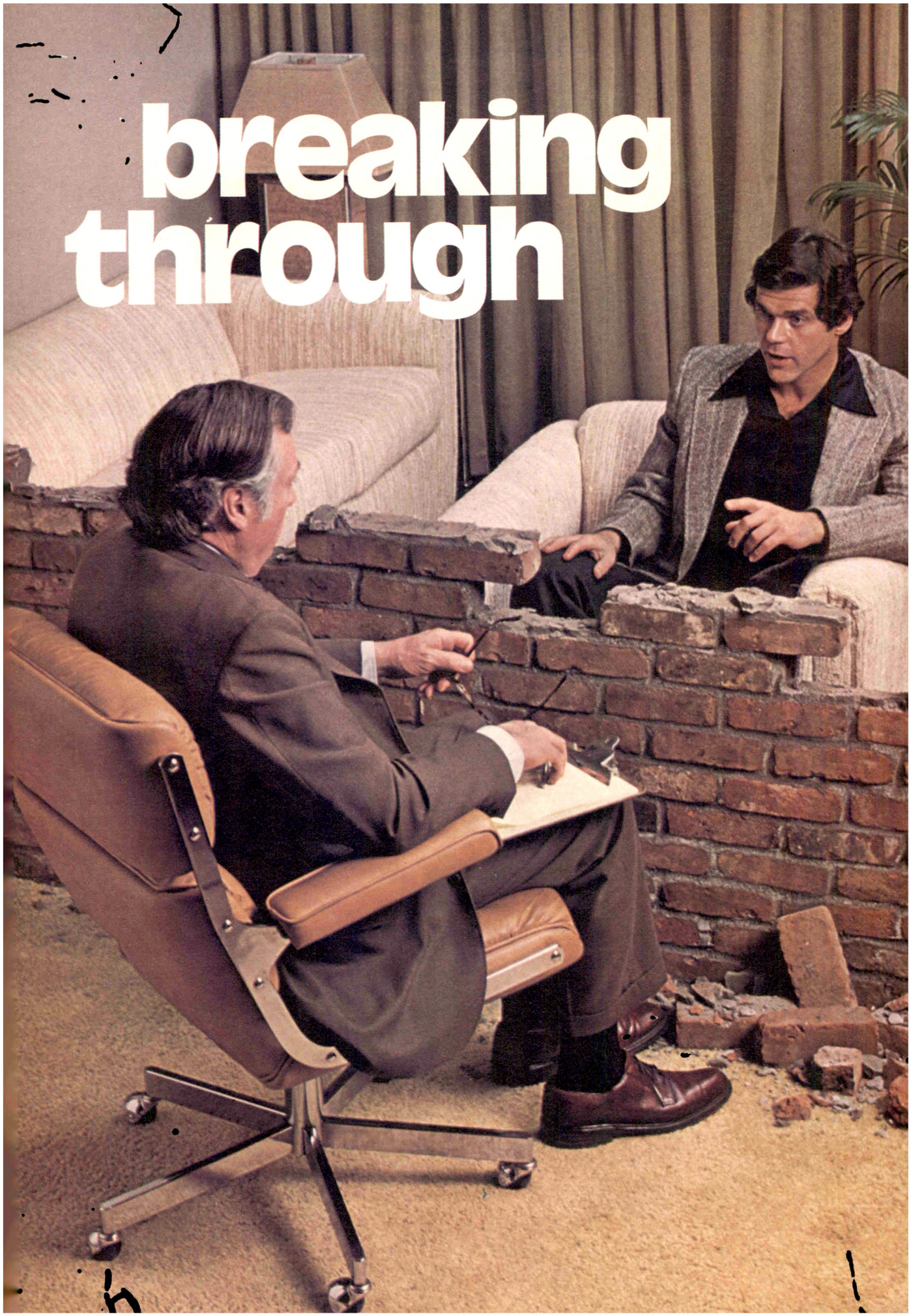
PEOPLE IN PAIN: A Guide to Pastoral Care *by* **James A. Vanderpool,** *District of Columbia Government Rehabilitation Center for Alcoholics, Occoquan, Virginia.* General counseling considerations are presented first in this concise volume followed by brief descriptions of atypical behavior. The case study method is then used to illustrate techniques for counseling individuals suffering from atypical and bizarre thinking, severe atypical feelings and emotional behavior, neuroses, depressions, atypical acting-out and antisocial behavior, alcoholism, sociopathology, drug addiction, and human sexuality problems. *'79, 208 pp., $12.75*

JUVENILE OFFENDERS (Rev. 6th Ptg.) *by* **Clyde B. Vedder,** *Univ. of Texas of the Permian Basin, Odessa. Foreword by* **Donal E. J. MacNamara.** For this Revised Sixth Printing, the author has updated and augmented the text and brought it into line with current practices. He continues to portray juvenile crime as it occurs in society, to note the various patterns of delinquency among juveniles, and to list official and informal treatment approaches. Specific chapter topics include the extent of juvenile delinquency, economic conditions and familial factors, community institutions, personality and behavior problems, apprehension and detention, and juvenile courts. *'79, 288 pp., 1 il., 5 tables, $11.95*

CAREER DEVELOPMENT AND COUNSELING OF WOMEN *edited by* **L. Sunny Hansen,** *Univ. of Minnesota, Minneapolis, and* **Rita S. Rapoza,** *Wilson Learning Corp., Eden Prairie, Minnesota. (46 Contributors)* The collection of articles in this text offers detailed discussion on the influences, both psychological and environmental, that either inhibit or facilitate the female decision-making and developmental process. Specific topics include occupational socialization, negotiating and planning for multiple roles, assessment of women's career interests, and cross-cultural perspectives of female career development. An extensive bibliography is included. *'78, 672 pp., 57 tables, $27.25*

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In marked agitation with depression symptomatic relief may make the patient more accessible and responsive.

Rapid relief of marked agitation in many patients
The tranquilizer component alleviates symptoms of agitation and anxiety within a few days, without apparent dulling of mental acuity. Hypnotic effects from the tranquilizer component appear to be minimal, particularly in patients permitted to remain active. However, TRIAVIL may impair mental and/or physical abilities required for performance of hazardous tasks.

Highly effective antidepressant action
The antidepressant component relieves symptoms of depression such as poor concentration and feelings of hopelessness as well as early morning awakening; adequate relief of symptoms may take a few weeks or even longer.

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As symptoms are relieved, many patients often communicate more effectively, become more cooperative, and are able to return to normal daily activities.

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There are now *five* tablet strengths of TRIAVIL for ease of dosage adjustment. For many patients with agitation and depression, you can now initiate therapy with one TRIAVIL™ 4-50, containing 4 mg perphenazine and 50 mg amitriptyline HCl, b.i.d. The regimen is simple, economical, and may well enhance patient compliance.

Treatment with TRIAVIL—a balanced view:
TRIAVIL is contraindicated in CNS depression from drugs, in the presence of evidence of bone marrow depression, and in patients hypersensitive to phenothiazines or amitriptyline. It should not be used during the acute recovery phase following myocardial infarction or in patients who have received an MAOI within two weeks. Patients with cardiovascular disorders should be watched closely. Not recommended in children or during pregnancy. TRIAVIL may impair mental and/or physical abilities required for performance of hazardous tasks and may enhance the response to alcohol. Antiemetic effect may obscure toxicity due to overdosage of other drugs or mask other disorders. The possibility of suicide in depressed patients remains until significant remission occurs. Such patients should not have access to large quantities of the drug. Hospitalize as soon as possible any patient suspected of having taken an overdose.

For marked agitation with depression

dual-action®

Triavil

containing perphenazine and amitriptyline HCl



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Please see following page for a brief summary of prescribing information.

More dosage strengths
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dual-action TRIAVIL®

containing perphenazine and amitriptyline HCl

Available:
TRIAVIL® 2-25: Each tablet contains
2 mg perphenazine and 25 mg amitriptyline HCl.
TRIAVIL® 2-10: Each tablet contains
2 mg perphenazine and 10 mg amitriptyline HCl.
TRIAVIL® 4-50: Each tablet contains
4 mg perphenazine and 50 mg amitriptyline HCl.
TRIAVIL® 4-25: Each tablet contains
4 mg perphenazine and 25 mg amitriptyline HCl.
TRIAVIL® 4-10: Each tablet contains
4 mg perphenazine and 10 mg amitriptyline HCl.

CONTRAINDICATIONS: Central nervous system depression from drugs (barbiturates, alcohol, narcotics, analgesics, antihistamines); evidence of bone marrow depression; known hypersensitivity to phenothiazines or amitriptyline. Should not be given concomitantly with a monoamine oxidase inhibitor since hyperpyretic crises, severe convulsions, and deaths have occurred from such combinations. When used to replace a monoamine oxidase inhibitor, allow a minimum of 14 days to elapse before initiating therapy with TRIAVIL. Therapy should then be initiated cautiously with gradual increase in dosage until optimum response is achieved. Not recommended for use during acute recovery phase following myocardial infarction.

WARNINGS: TRIAVIL should not be given concomitantly with guanethidine or similarly acting compounds since TRIAVIL may block the antihypertensive action of such compounds. Use cautiously in patients with history of urinary retention, angle-closure glaucoma, increased intraocular pressure, or convulsive disorders. Dosage of anticonvulsive agents may have to be increased. In patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely. Tricyclic antidepressants, including amitriptyline HCl, have been reported to produce arrhythmias, sinus tachycardia, and prolongation of conduction time, particularly in high doses. Myocardial infarction and stroke have been reported with tricyclic antidepressant drugs. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. May impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle. In patients who use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage. Not recommended in children or during pregnancy.

PRECAUTIONS: Suicide is a possibility in depressed patients and may remain until significant remission occurs. Such patients should not have access to large quantities of this drug.

Perphenazine: Should not be used indiscriminately. Use with caution in patients who have previously exhibited severe adverse reactions to other phenothiazines. Likelihood of some untoward actions is greater with high doses. Closely supervise with any dosage. The antiemetic effect of perphenazine may obscure signs of toxicity due to overdosage of other drugs or make more difficult the diagnosis of disorders such as brain tumor or intestinal obstruction. A significant, not otherwise explained, rise in body temperature may suggest individual intolerance to perphenazine, in which case discontinue.

If hypotension develops, epinephrine should not be employed, as its action is blocked and partially reversed by perphenazine. Phenothiazines may potentiate the action of central nervous system depressants (opiates, analgesics, antihistamines, barbiturates, alcohol) and atropine. In concurrent therapy with any of these, TRIAVIL should be given in reduced dosage. May also potentiate the action of heat and phosphorous insecticides. There is sufficient experimental evidence to conclude that chronic administration of antipsychotic drugs which increase prolactin secretion has the potential to induce mammary neoplasms in rodents under the appropriate conditions. There are recognized differences in the physiological role of prolactin between rodents and humans. Since there are, at present, no adequate epidemiological studies, the relevance to human mammary cancer risk from prolonged exposure to perphenazine and other antipsychotic drugs is not known.

Amitriptyline: In manic-depressive psychosis, depressed patients may experience a shift toward the manic phase if they are treated with an antidepressant. Patients with paranoid symptomatology may have an exaggeration of such symptoms. The tranquilizing effect of TRIAVIL seems to reduce the likelihood of this effect. When amitriptyline HCl is given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required. Paralytic ileus may occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs.

Caution is advised if patients receive large doses of ethchlorvynol concurrently. Transient delirium has been reported in patients who were treated with 1 g of ethchlorvynol and 75-150 mg of amitriptyline HCl.

Amitriptyline HCl may enhance the response to alcohol and the effects of barbiturates and other CNS depressants.

Concurrent administration of amitriptyline HCl and electroshock therapy may increase the hazards associated with such therapy. Such treatment should be limited to patients for whom it is essential. Discontinue several days before elective surgery if possible. Elevation and lowering of blood sugar levels have both been reported. Use with caution in patients with impaired liver function.

ADVERSE REACTIONS: Similar to those reported with either constituent alone.
Perphenazine: Extrapyramidal symptoms (opisthotonus, oculogyric crisis, hyperreflexia, dystonia, akathisia, acute dyskinesia, ataxia, parkinsonism) have been reported and can usually be controlled by the concomitant use of effective antiparkinsonian drugs and/or by reduction in dosage, but sometimes persist after discontinuation of the phenothiazine.

Tardive dyskinesia may appear in some patients on long-term therapy or may occur after drug therapy with phenothiazines and related agents has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. Symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw. Involuntary movements of the extremities sometimes occur. There is no known treatment for tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms. It is advised that all antipsychotic agents be discontinued if the above symptoms appear. If treatment is reinstituted, or dosage of the particular drug increased, or another drug substituted, the syndrome may be masked. Fine vermicular movements of the tongue may be an early sign of the syndrome. The full-blown syndrome may not develop if medication is stopped when lingual vermiculation appears.

Other side effects are skin disorders (photosensitivity, itching, erythema, urticaria, eczema, up to exfoliative dermatitis); other allergic reactions (asthma, laryngeal edema, angioneurotic edema, anaphylactoid reactions); peripheral edema; reversed epinephrine effect; hyperglycemia; endocrine disturbances (lactation, galactorrhea, gynecomastia, disturbances of menstrual cycle); altered cerebrospinal fluid proteins; paradoxical excitement; hypertension, hypotension, tachycardia, and ECG abnormalities (quinidine-like effect); reactivation of psychotic processes; catatonic-like states; autonomic reactions, such as dry mouth or salivation, headache, anorexia, nausea, vomiting, constipation, obstipation, urinary frequency or incontinence, blurred vision, nasal congestion, and a change in pulse rate; other adverse reactions reported with various phenothiazine compounds, but not with perphenazine, include grand mal convulsions, cerebral edema, polyphagia, pigmentary retinopathy, photophobia, skin pigmentation, and failure of ejaculation.

The phenothiazine compounds have produced blood dyscrasias (pancytopenia, thrombocytopenic purpura, leukopenia, agranulocytosis, eosinophilia) and liver damage (jaundice, biliary stasis).

Pigmentation of the cornea and lens has been reported to occur after long-term administration of some phenothiazines. Although it has not been reported in patients receiving TRIAVIL, the possibility that it might occur should be considered.

Hypnotic effects, lassitude, muscle weakness, and mild insomnia have also been reported.

Amitriptyline: Note: Listing includes a few reactions not reported for this drug, but which have occurred with other pharmacologically similar tricyclic antidepressant drugs and must be considered when amitriptyline is administered. *Cardiovascular:* Hypotension; hypertension; tachycardia; palpitation; myocardial infarction; arrhythmias; heart block; stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesia of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth; blurred vision; disturbance of accommodation; increased intraocular pressure; constipation; paralytic ileus; urinary retention; dilatation of urinary tract. *Allergic:* Skin rash; urticaria; photosensitization; edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis; leukopenia; eosinophilia; purpura; thrombocytopenia. *Gastrointestinal:* Nausea; epigastric distress; vomiting; anorexia; stomatitis; peculiar taste; diarrhea; parotid swelling; black tongue. Rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido; elevated or lowered blood sugar levels. *Other:* Dizziness, weakness; fatigue; headache; weight gain or loss; increased perspiration; urinary frequency; mydriasis; drowsiness; alopecia. *Withdrawal Symptoms:* Abrupt cessation after prolonged administration may produce nausea, headache, and malaise. These are not indicative of addiction.

OVERDOSAGE: All patients suspected of having taken an overdosage should be admitted to a hospital as soon as possible. Treatment is symptomatic and supportive. However, the intravenous administration of 1–3 mg of physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage of physostigmine should be repeated as required particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine. On this basis, in severe overdosage with perphenazine-amitriptyline combinations, symptomatic treatment of central anticholinergic effects with physostigmine salicylate should be considered.

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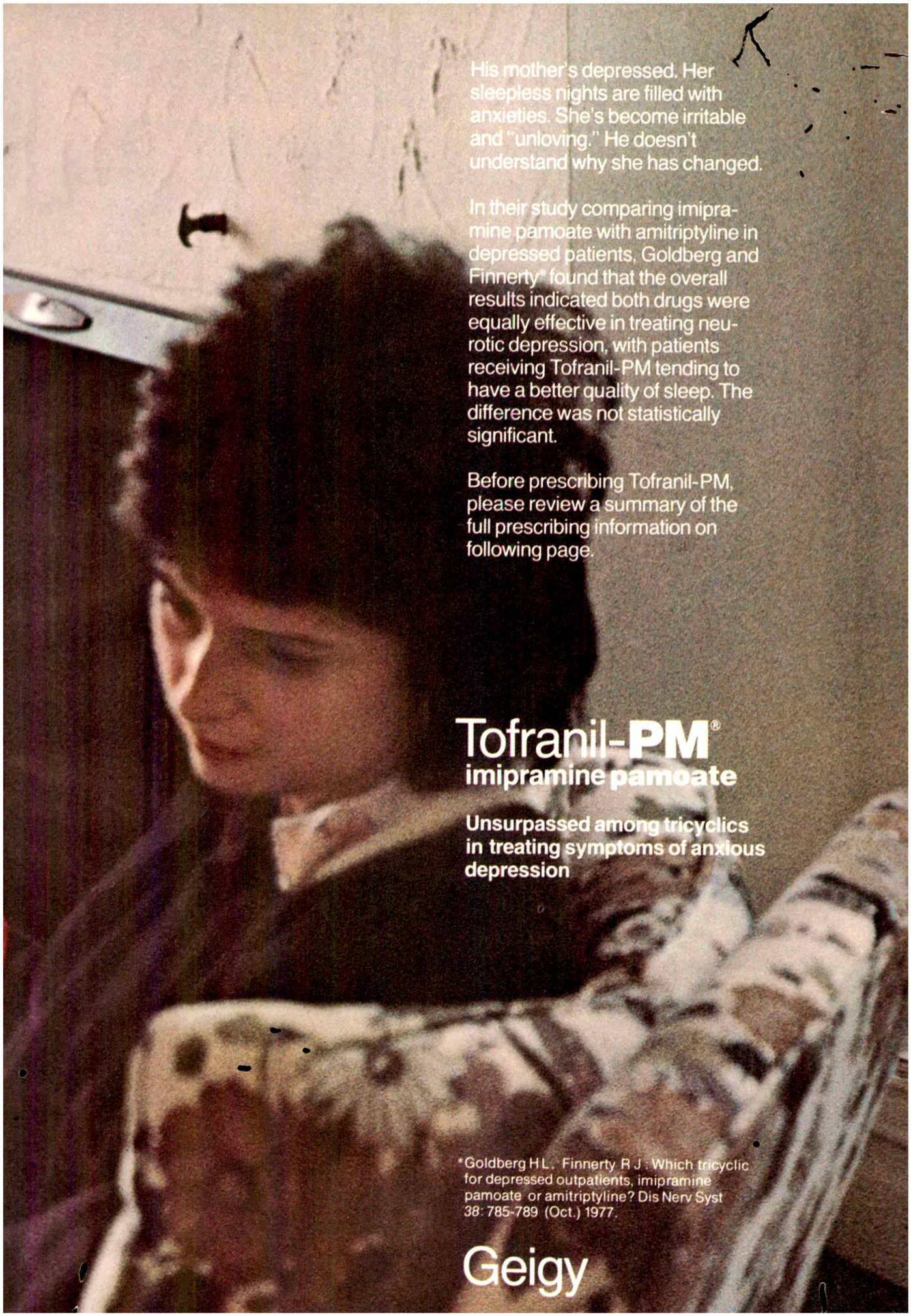
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Depression: Unhappily often a family affair...


“Please don’t cry.”



His mother's depressed. Her sleepless nights are filled with anxieties. She's become irritable and "unloving." He doesn't understand why she has changed.

In their study comparing imipramine pamoate with amitriptyline in depressed patients, Goldberg and Finnerty* found that the overall results indicated both drugs were equally effective in treating neurotic depression, with patients receiving Tofranil-PM tending to have a better quality of sleep. The difference was not statistically significant.

Before prescribing Tofranil-PM, please review a summary of the full prescribing information on following page.

Tofranil-PM®
imipramine pamoate

Unsurpassed among tricyclics in treating symptoms of anxious depression

*Goldberg H L, Finnerty R J : Which tricyclic for depressed outpatients, imipramine pamoate or amitriptyline? Dis Nerv Syst *38*: 785-789 (Oct.) 1977.

Geigy

Tofranil-PM®
imipramine **pamoate**

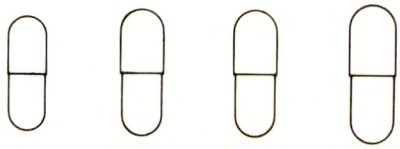
Unsurpassed among tricyclics in treating a broad range of depressions—from the more retarded to the more anxious.

Effectively relieves sleep disturbances, anxiety, and other symptoms of depression.

Notable mood-brightening effect especially helpful in the more retarded depressions.

Good results can be seen with a starting dose of 75 mg h.s.; however, higher doses may be needed.

Capsules of*



75 mg | 100 mg | 125 mg | 150 mg

As with all tricyclics, sedation may occur. For details, please read the full prescribing information, a summary of which appears on this page.

*Each capsule contains imipramine pamoate equivalent to 75, 100, 125 or 150 mg of imipramine hydrochloride.

Geigy

Tofranil-PM®
imipramine pamoate

Indications For the relief of symptoms of depression. Endogenous depression is more likely to be alleviated than other depressive states. One to three weeks of treatment may be needed before optimal therapeutic effects are evident.

Contraindications The concomitant use of monoamine oxidase inhibiting compounds is contraindicated. Hyperpyretic crises or severe convulsive seizures may occur in patients receiving such combinations. The potentiation of adverse effects can be serious, or even fatal. When it is desired to substitute Tofranil-PM in patients receiving a monoamine oxidase inhibitor, as long an interval should elapse as the clinical situation will allow, with a minimum of 14 days. Initial dosage should be low and increases should be gradual and cautiously prescribed.

The drug is contraindicated during the acute recovery period after a myocardial infarction. Patients with a known hypersensitivity to this compound should not be given the drug. The possibility of cross-sensitivity to other dibenzazepine compounds should be kept in mind.

Warnings Extreme caution should be used when this drug is given to:

patients with cardiovascular disease because of the possibility of conduction defects, arrhythmias, congestive heart failure, myocardial infarction, strokes and tachycardia. These patients require cardiac surveillance at all dosage levels of the drug;

patients with increased intraocular pressure, history of urinary retention, or history of narrow-angle glaucoma because of the drug's anticholinergic properties;

hyperthyroid patients or those on thyroid medication because of the possibility of cardiovascular toxicity;

patients with a history of seizure disorder because this drug has been shown to lower the seizure threshold;

patients receiving guanethidine, clonidine, or similar agents, since imipramine pamoate may block the pharmacologic effects of these drugs;

patients receiving methylphenidate hydrochloride. Since methylphenidate hydrochloride may inhibit the metabolism of imipramine pamoate, downward dosage adjustment of imipramine pamoate may be required when given concomitantly with methylphenidate hydrochloride.

Since imipramine pamoate may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks, such as operating an automobile or machinery, the patient should be cautioned accordingly.

Tofranil-PM may enhance the CNS depressant effects of alcohol. Therefore, it should be borne in mind that the dangers inherent in a suicide attempt or accidental overdosage with the drug may be increased for the patient who uses excessive amounts of alcohol. (See **Precautions**.)

Usage in Children: Tofranil-PM should not be used in children of any age because of the increased potential for acute overdosage due to the high unit potency (75 mg, 100 mg, 125 mg and 150 mg). Each capsule contains imipramine pamoate equivalent to 75 mg, 100 mg, 125 mg or 150 mg imipramine hydrochloride.

Precautions An ECG recording should be taken prior to the initiation of larger-than-usual doses of imipramine pamoate and at appropriate intervals thereafter until steady state is achieved. (Patients with any evidence of cardiovascular disease require cardiac surveillance at all dosage levels of the drug. See **Warnings**.) Elderly patients and patients with cardiac disease or a prior history of cardiac disease are at special risk of developing the cardiac abnormalities associated with the use of imipramine pamoate. It should be kept in mind that the possibility of suicide in seriously depressed patients is inherent in the illness and may persist until significant remission occurs. Such patients should be carefully supervised during the early phase of treatment with imipramine pamoate and may require hospitalization. Prescriptions should be written for the smallest amount feasible.

Hypomanic or manic episodes may occur, particularly in patients with cyclic disorders. Such reactions may necessitate discontinuation of the drug. If needed, imipramine pamoate may be resumed in lower dosage when these episodes are relieved. Administration of a tranquilizer may be useful in controlling such episodes.

An activation of the psychosis may occasionally be observed in schizophrenic patients and may require reduction of dosage and the addition of a phenothiazine.

Concurrent administration of imipramine pamoate with electroshock therapy may increase the hazards; such treatment should be limited to those patients for whom it is essential, since there is limited clinical experience.

Usage During Pregnancy and Lactation:

Animal reproduction studies have yielded inconclusive results. (See also **Animal Pharmacology & Toxicology**.)

There have been no well-controlled studies conducted with pregnant women to determine the effect of imipramine on the fetus. However, there have been clinical reports of congenital malformations associated with the use of the drug. Although a causal relationship between these effects and the drug could not be established, the possibility of fetal risk from the maternal ingestion of imipramine cannot be excluded. Therefore, imipramine should be used in women who are or might become pregnant only if the clinical condition clearly justifies potential risk to the fetus.

Limited data suggest that imipramine is likely to be excreted in human breast milk. As a general rule, a woman taking a drug should not nurse since the possibility exists that the drug may be excreted in breast milk and be harmful to the child.

Patients should be warned that imipramine pamoate may enhance the CNS depressant effects of alcohol. (See **Warnings**.)

Imipramine pamoate should be used with caution in p
with significantly impaired renal or hepatic function.

Patients who develop a fever and a sore throat during
therapy with imipramine pamoate should have leukoc
differential blood counts performed. Imipramine pamo
should be discontinued if there is evidence of patholo
neutrophil depression.

Prior to elective surgery, imipramine pamoate should b
continued for as long as the clinical situation will allow

In occasional susceptible patients or in those receivin
ticholinergic drugs (including anti-parkinsonism agent
addition, the atropine-like effects may become more p
nounced (e.g., paralytic ileus). Close supervision and
adjustment of dosage is required when imipramine pa
is administered concomitantly with anticholinergic dru

Avoid the use of preparations, such as decongestants
local anesthetics, which contain any sympathomimetic
amine (e.g., epinephrine, norepinephrine), since it has
reported that tricyclic antidepressants can potentiate t
fects of catecholamines.

Caution should be exercised when imipramine pamoa
used with agents that lower blood pressure.

Imipramine pamoate may potentiate the effects of CN
pressant drugs.

Patients taking imipramine pamoate should avoid exc
exposure to sunlight since there have been reports of
sensitization.

Both elevation and lowering of blood sugar levels have
reported with imipramine pamoate use.

Adverse Reactions Note: Although the listing which f
includes a few adverse reactions which have not been
ported with this specific drug, the pharmacological
similarities among the tricyclic antidepressant drugs re
that each of the reactions be considered when imipran
administered.

Cardiovascular: Orthostatic hypotension, hypertensio
tachycardia, palpitation, myocardial infarction, arrhyth
heart block, ECG changes, precipitation of congestive
failure, stroke.

Psychiatric: Confusional states (especially in the elder
with hallucinations, disorientation, delusions; anxiety, r
lessness, agitation; insomnia and nightmares; hypoma
exacerbation of psychosis.

Neurological: Numbness, tingling, paresthesias of ex-
tremities; incoordination, ataxia, tremors; peripheral
neuropathy; extrapyramidal symptoms; seizures, alter
in EEG patterns; tinnitus.

Anticholinergic: Dry mouth, and, rarely, associated sut
gual adenitis; blurred vision, disturbances of accommo
tion, mydriasis; constipation, paralytic ileus; urinary ret
tion, delayed micturition, dilation of the urinary tract.

Allergic: Skin rash, petechiae, urticaria, itching, photos
sitization; edema (general or of face and tongue); drug
cross-sensitivity with desipramine.

Hematologic: Bone marrow depression including agra
cytosis; eosinophilia; purpura; thrombocytopenia.

Gastrointestinal: Nausea and vomiting, anorexia, epig
distress, diarrhea; peculiar taste, stomatitis, abdominal
cramps, black tongue.

Endocrine: Gynecomastia in the male; breast enlarger
and galactorrhea in the female; increased or decrease
libido, impotence; testicular swelling; elevation or depr
of blood sugar levels.

Other: Jaundice (simulating obstructive); altered liver f
tion; weight gain or loss; perspiration; flushing; urinary
quency; drowsiness, dizziness, weakness and fatigue;
headache; parotid swelling; alopecia; proneness to fall

Withdrawal Symptoms: Though not indicative of addict
abrupt cessation of treatment after prolonged therapy r
produce nausea, headache and malaise.

How Supplied Tofranil-PM is available as follows:

75-mg, coral-colored capsules equivalent to 75 mg imi
ramine hydrochloride in bottles of 30, 100 and 1,000 ca
sules, and unit strip packages of 100.

100-mg, dark yellow/coral-colored capsules equivalent
mg imipramine hydrochloride in bottles of 30, 100 and 1
capsules.

125-mg, light yellow/coral-colored capsules equivalent
125 mg imipramine hydrochloride in bottles of 30, 100 a
1,000 capsules.

150-mg, coral-colored capsules equivalent to 150 mg in
ramine hydrochloride in bottles of 30, 100 and 1,000 ca
sules, and unit strip packages of 100.

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and administration, and overdosage, please see full
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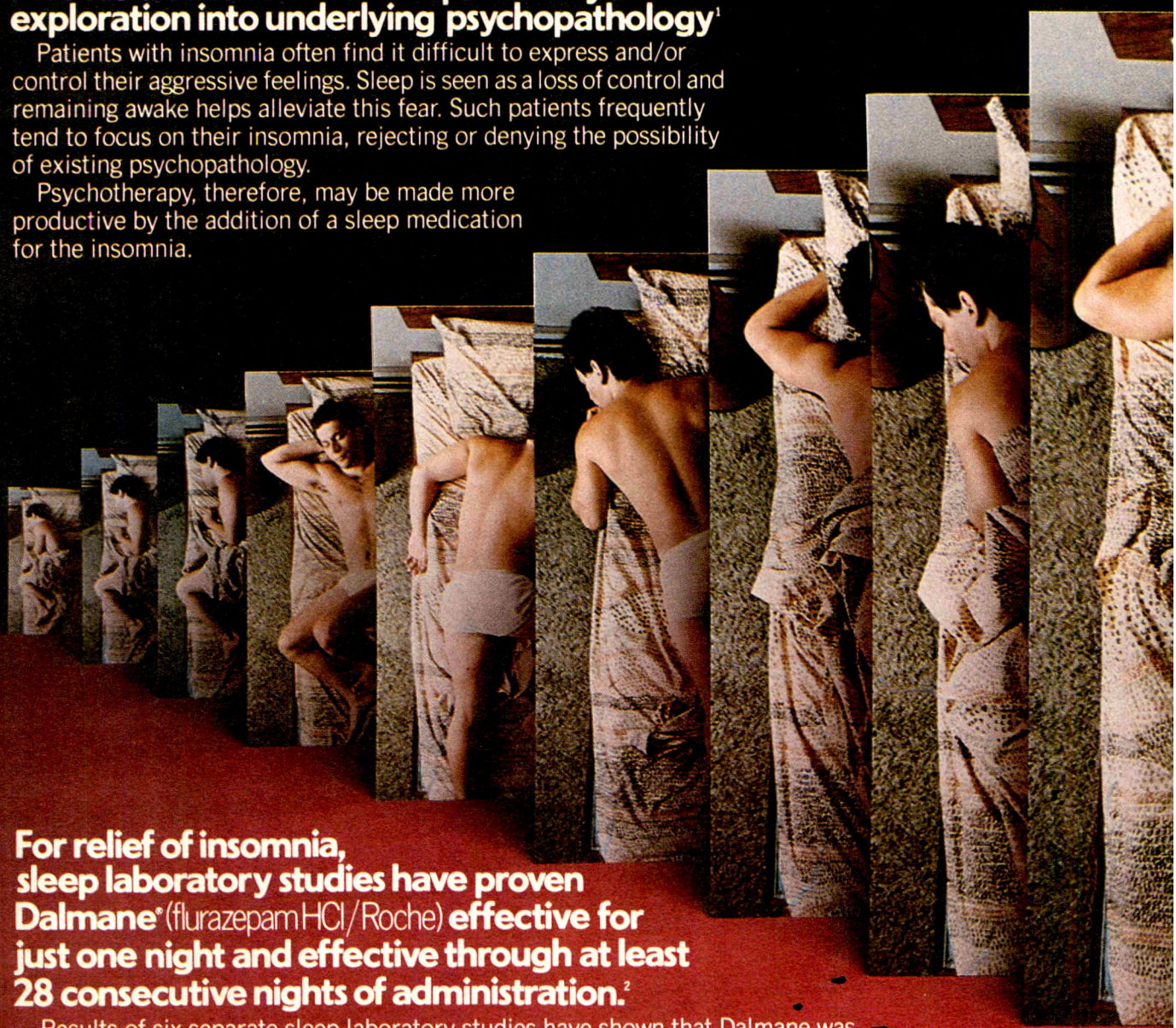
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References:
1. Kales A, Kales JD, Humphry FJ II: Sleep and dreams, chap. 2.3, in *Comprehensive Textbook of Psychiatry/II,* edited by Freedman AM, Kaplan HI, Sadock BJ, ed 2. Baltimore, The Williams & Wilkins Company, vol 1, 1976, pp. 114-128
2. Kales A, *et al: Clin Pharmacol Ther* 19:576-583, May 1976
3. Jacobson A, *et al: Psychophysiology* 7:345, Sep 1970
4. Data on file, Medical Department, Hoffmann-La Roche Inc., Nutley NJ

Before prescribing Dalmane (flurazepam HCl/Roche), please consult complete product information, a summary of which follows:

Indications: Effective in all types of insomnia characterized by difficulty in falling asleep, frequent nocturnal awakenings and/or early morning awakening; in patients with recurring insomnia or poor sleeping habits; in acute or chronic medical situations requiring restful sleep. Objective sleep laboratory data have shown effectiveness for at least 28 consecutive nights of administration. Since insomnia is often transient and intermittent, prolonged administration is generally not necessary or recommended.

Contraindications: Known hypersensitivity to flurazepam HCl.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).

***Usage in Pregnancy:* Several studies of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) suggest increased risk of congenital malformations during the first trimester of pregnancy. Dalmane (flurazepam HCl/Roche), a benzodiazepine, has not been studied adequately to determine whether it may be associated with such an increased risk. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage.

Precautions: In elderly and debilitated patients, it is recommended that the dosage be limited to 15 mg to reduce risk of oversedation, dizziness, confusion and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in patients who are severely depressed, or with latent depression or suicidal tendencies, or with impaired renal or hepatic function. Periodic blood counts and liver and kidney function tests are advised during repeated therapy.

Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity, and elevated SGOT, SGPT, total and direct bilirubins and alkaline phosphatase.

Dosage: Individualize for maximum beneficial effect.
Adults: 30 mg usual dosage; 15 mg may suffice in some patients. *Elderly or debilitated patients:* 15 mg **recommended** initially until response is determined.

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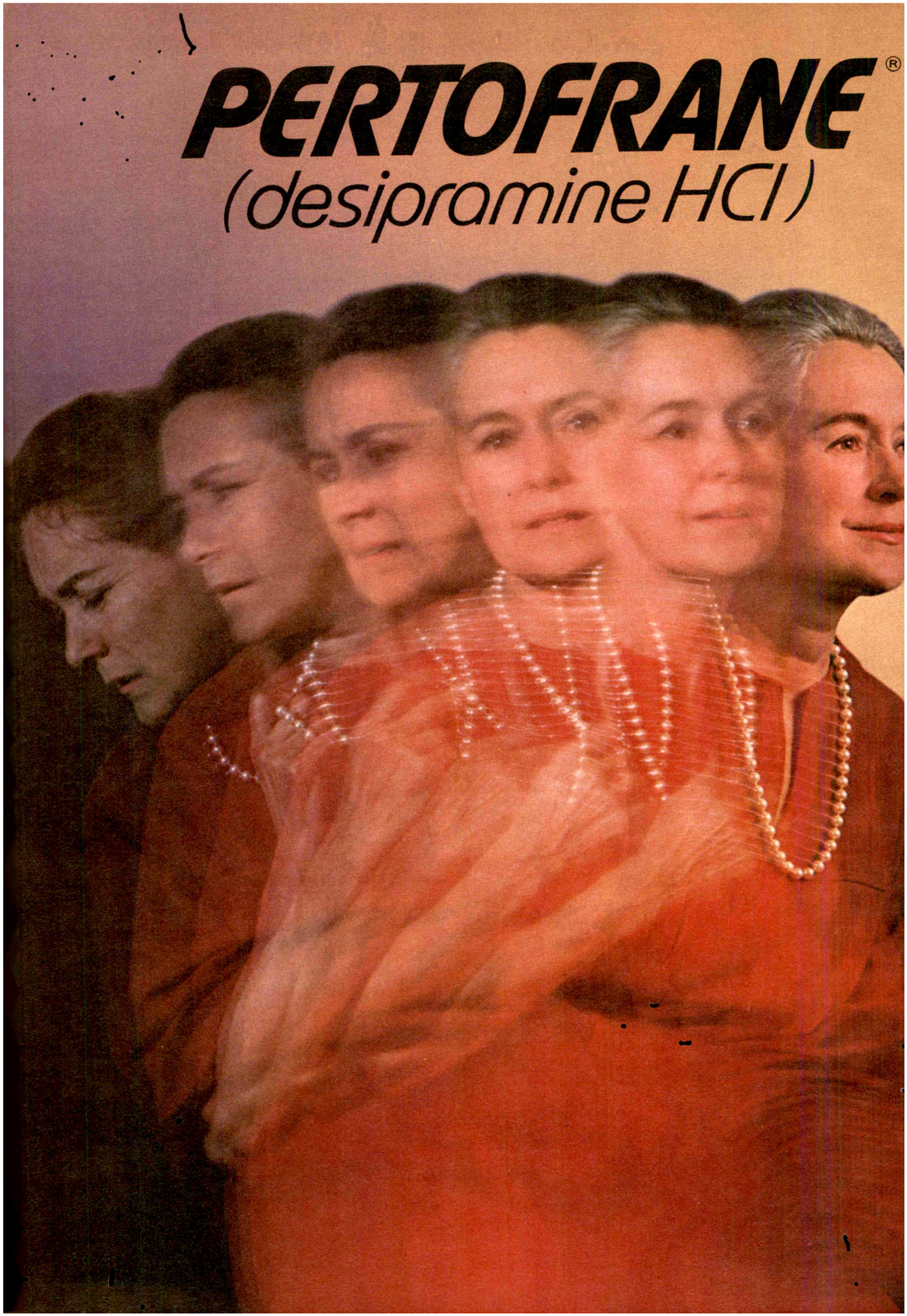
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References: 1. Hollister, L.E.: Treatment of depression with drugs, Ann. Intern. Med. 89:78, 1978. 2. Blackwell, B. et al.: Anticholinergic activity of two tricyclic antidepressants, Am. J. Psychiatry 135:722, 1978. 3. Barnes, R. J.: Clinical depression: Double-blind study of desipramine and amitriptyline in depressive neurosis, Scientific Exhibit, Southern Medical Convention, Miami, Fla., Nov. 16-19, 1975.

PERTOFRANE® (desipramine HCl)

when the first tricyclic fails

Indication: For relief of mental depression. **Contraindications:** Do not use MAO inhibitors concomitantly or within 2 weeks of the use of this drug. Hyperpyretic crises or severe convulsive seizures may occur with such combinations; potentiation of adverse reactions can be serious or even fatal. When substituting Pertofrane (desipramine HCl) in patients receiving an MAO inhibitor, allow an interval of at least 14 days. Initial dosage in such patients should be low and increases should be gradual and cautiously prescribed. The drug is contraindicated following recent myocardial infarction and in patients with a known hypersensitivity to tricyclic antidepressants. **Warnings:** Activation of psychosis may occasionally be observed in schizophrenic patients. Due to atropine-like effects and sympathomimetic potentiation, use only with the greatest care in patients with narrow-angle glaucoma or urethral or ureteral spasm. Do not use in patients with the following conditions unless the need outweighs the risk: severe coronary heart disease with EKG abnormalities, progressive heart failure, angina pectoris, paroxysmal tachycardia and active seizure disorder (may lower seizure threshold). This drug may block the action of the antihypertensive, guanethidine, and related adrenergic neuron-blocking agents. Hypertensive episodes have been observed during surgery. The concurrent use of other central nervous system drugs or alcohol may potentiate adverse effects. Since many such drugs may be used during surgery, desipramine should be discontinued prior to elective procedures. The potentiation resulting from excessive use of alcohol may increase the danger inherent in a suicide attempt or overdosage. Caution patients on the possibility of impaired ability to operate a motor vehicle or dangerous machinery. Do not use in women who are or may become pregnant, or in children under 12 years of age, unless the clinical situation warrants the potential risk. Because of increased sensitivity to the drug, use lower than normal dosage in adolescent and geriatric patients. **Precautions:** Potentially suicidal patients require careful supervision and protective measures during therapy. Prescriptions should be limited to small quantities. Discontinuation of the drug may be necessary in the presence of increased agitation and anxiety shifting to hypomanic or manic excitement. Atropine-like effects may be more pronounced (e.g. paralytic ileus) in susceptible patients and in those receiving anticholinergic drugs (including antiparkinsonism agents). Prescribe cautiously in hyperthyroid patients and in those receiving thyroid medications; transient cardiac arrhythmias have occurred in rare instances. Periodic blood and liver studies should supplement careful clinical observations in all patients undergoing extended courses of therapy. **Adverse Reactions:** The following have been reported: *Nervous System:* dizziness, drowsiness, insomnia, headache, disturbed visual accommodation, tremor, unsteadiness, tinnitus, paresthesias, changes in EEG patterns, epileptiform seizures, mild extrapyramidal activity, falling and neuromuscular incoordination. A confusional state (with such symptoms as hallucinations and disorientation), particularly in older patients and at higher dosage, may require discontinuation of the drug. *Gastrointestinal Tract:* anorexia, dryness of the mouth, nausea, epigastric distress, constipation and diarrhea. *Skin:* skin rashes (including photosensitization), perspiration and flushing sensations. *Liver:* rare cases of transient jaundice (apparently of an obstructive nature) and liver damage. If jaundice or abnormalities in liver function tests occur, discontinue the drug and investigate. *Blood Elements:* bone-marrow depression, agranulocytosis, thrombocytopenia and purpura. If these occur, discontinue the drug. Transient eosinophilia has been observed. *Cardiovascular System:* orthostatic hypotension and tachycardia. Carefully supervise patients requiring concomitant vasodilating therapy, particularly during initial phases. *Genitourinary System:* urinary frequency or retention and impotence. *Endocrine System:* occasional hormonal effects, including gynecomastia, galactorrhea and breast enlargement, and decreased libido and estrogenic effect. *Sensitivity:* urticaria and rare instances of drug fever and cross-sensitivity with imipramine. **Dosage:** All patients except geriatric and adolescent: 75 to 150 mg./day in divided doses or as single daily dose. Dosage may be increased up to 200 mg. daily. Geriatric and adolescent patients should usually be started with lower dosage (25 to 50 mg. daily) and may not tolerate higher doses. Dosage may be increased up to 100 mg. daily. Lower maintenance dosages should be continued for at least 2 months after obtaining a satisfactory response. Therapy may be given in divided doses or as a single daily dose. **How Supplied:** 25 mg. capsules (pink), bottles of 100 and 1000. Also, 50 mg. capsules (maroon and pink), bottles of 100 and 1000.

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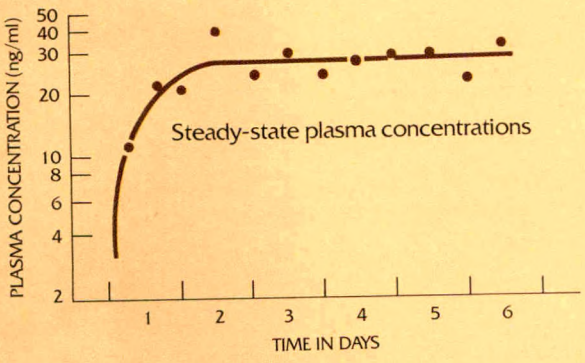
Most benzodiazepines take at least 7 to 10 days to reach steady-state serum levels. With Ativan, that equilibrium is reached in 2 to 3 days. A factor that can be important when you modify dosage schedules to meet individual needs.

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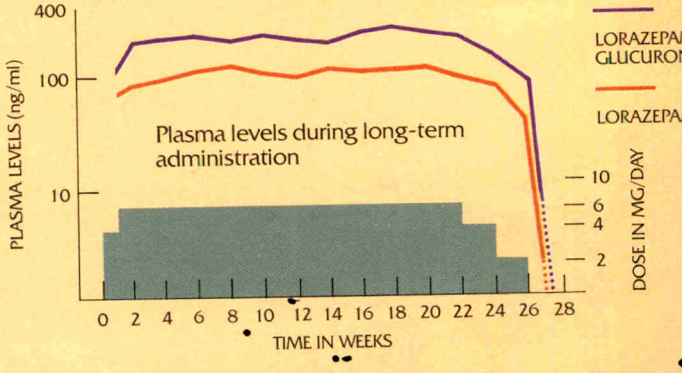
Because Ativan has no active metabolites to be eliminated at varying rates, steady-state levels are achieved predictab and smoothly. Six-month studies show no evidence of drug-accumulation above steady-state levels even at maximum daily dosages.

*The pharmacokinetic profile of a drug cannot, at present, be directly related to its therapeutic effectiveness.

Please refer to page after next for a summary of prescribing information.



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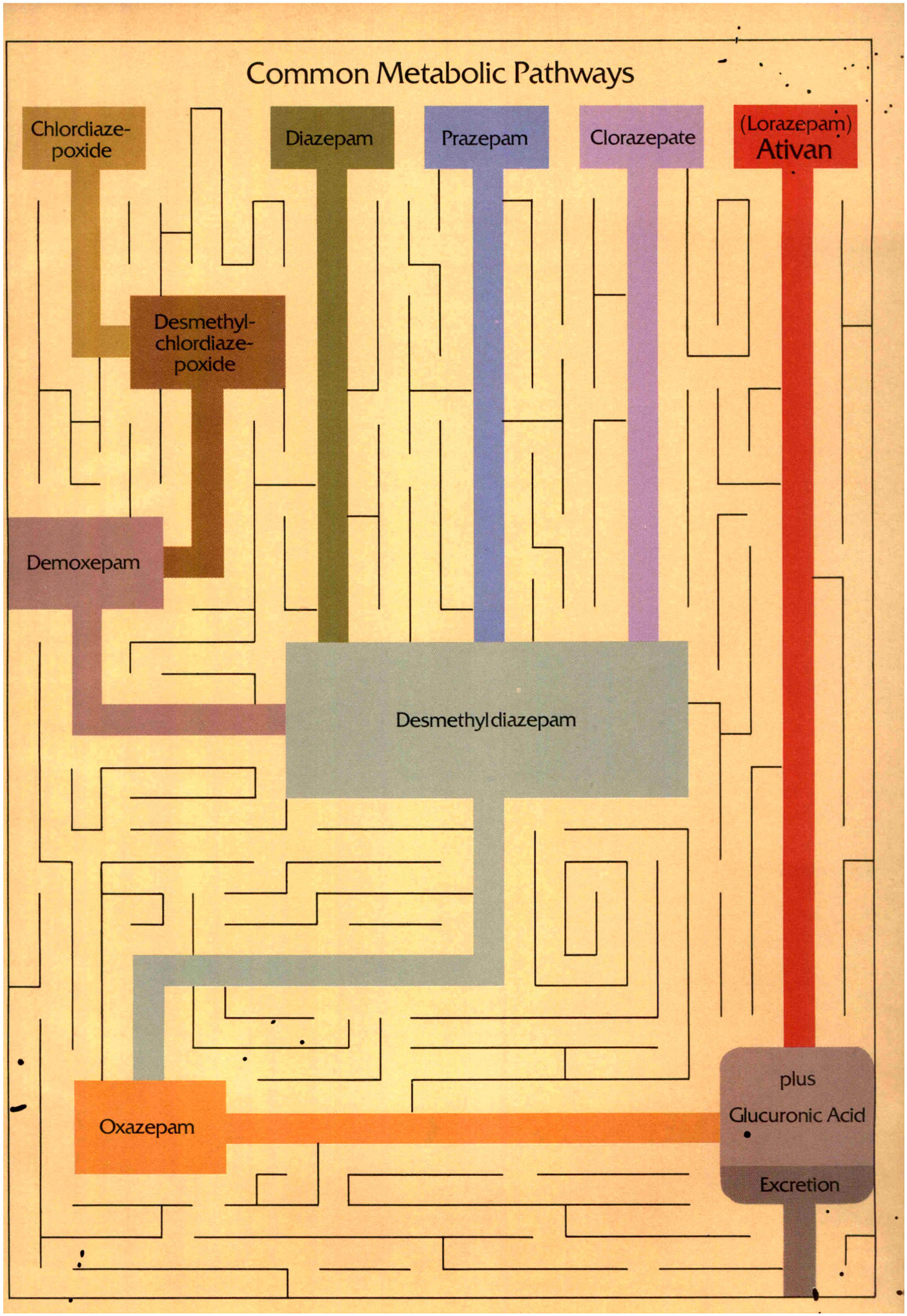


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Contraindications: Ativan is contraindicated in patients with known sensitivity to the benzodiazepines or with acute narrow-angle glaucoma.

Warnings: Ativan is not recommended for use in patients with a primary depressive disorder or psychosis. As with all patients on CNS-acting drugs, patients receiving lorazepam should be warned not to operate dangerous machinery or motor vehicles and that their tolerance for alcohol and other CNS depressants will be diminished.

Physical and Psychological Dependence: Withdrawal symptoms similar in character to those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepine drugs. These symptoms include convulsions, tremor, abdominal and muscle cramps, vomiting and sweating. Addiction-prone individuals, such as drug addicts and alcoholics, should be under careful surveillance when receiving benzodiazepines because of the predisposition of such patients to habituation and dependence.

Precautions: In patients with depression accompanying anxiety, a possibility for suicide should be borne in mind.

For elderly or debilitated patients, the initial daily dosage should not exceed 2 mg in order to avoid oversedation.

Ativan dosage should be terminated gradually since abrupt withdrawal of any antianxiety agent may result in symptoms similar to those for which patients are being treated: anxiety, agitation, irritability, tension, insomnia and occasional convulsions.

The usual precautions for treating patients with impaired renal or hepatic function should be observed.

In patients where gastrointestinal or cardiovascular disorders coexist with anxiety, it should be noted that lorazepam has not been shown to be of significant benefit in treating the gastrointestinal or cardiovascular component.

Esophageal dilation occurred in rats treated with lorazepam for more than one year at 6 mg/kg/day. The no-effect dose was 1.25 mg/kg/day (approximately 6 times the maximum human therapeutic dose of 10 mg per day). The effect was reversible only when the treatment was withdrawn within two months of first observation of the phenomenon. The clinical significance of this is unknown. However, use of lorazepam for prolonged periods and in geriatric patients requires caution, and there should be frequent monitoring for symptoms of upper G.I. disease.

Safety and effectiveness of Ativan in children of less than 12 years have not been established.

Essential Laboratory Tests: Some patients on Ativan have developed leukopenia and some have had elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function tests are recommended for patients on long-term therapy.

Clinically Significant Drug Interactions: The benzodiazepines including Ativan produce CNS depressant effects when administered with such medications as barbiturates or alcohol.

Carcinogenesis and Mutagenesis: No evidence of carcinogenic potential emerged in rats during an 18-month study with Ativan. No studies regarding mutagenesis have been performed.

Pregnancy: Reproductive studies in animals were performed in mice, rats, and two strains of rabbits. Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastroschisis, malformed skull and microphthalmia) were seen in drug-treated rabbits without relationship to dosage. Although all of these anomalies were not present in the concurrent control group, they have been reported to occur randomly in historical controls. At doses of 40 mg/kg and higher, there was evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lower doses.

The clinical significance of the above findings is not known. However, an increased risk of congenital malformations associated with the use of minor tranquilizers (chlordiazepoxide, diazepam and meprobamate) during the first trimester of pregnancy has been suggested in several studies. Because the use of these drugs is rarely a matter of urgency, the use of lorazepam during this period should almost always be avoided. The possibility that a woman of child-bearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant, they should communicate with their physician about the desirability of discontinuing the drug.

In humans, blood levels obtained from umbilical cord blood indicate placental transfer of lorazepam and lorazepam glucuronide.

Nursing Mothers: It is not known whether oral lorazepam is excreted in human milk like the other benzodiazepine tranquilizers. As a general rule, nursing should not be undertaken while a patient is on a drug since many drugs are excreted in human milk.

Adverse Reactions: Adverse reactions, if they occur, are usually observed at the beginning of therapy and generally disappear on continued medication or upon decreasing the dose. In a sample of about 3,500 anxious patients, the most frequent adverse reaction to Ativan is sedation (15.9%), followed by dizziness (6.9%), weakness (4.2%) and unsteadiness (3.4%). Less frequent adverse reactions are disorientation, depression, nausea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, eye function disturbance, together with various gastrointestinal symptoms and autonomic manifestations. The incidence of sedation and unsteadiness increased with age.

Small decreases in blood pressure have been noted but are not clinically significant, probably being related to the relief of anxiety produced by Ativan.

Overdosage: In the management of overdosage with any drug, it should be borne in mind that multiple agents may have been taken.

Manifestations of Ativan overdosage include somnolence, confusion and coma. Induced vomiting and/or gastric lavage should be undertaken followed by general supportive care, monitoring of vital signs and close observation of the patient. Hypotension, though unlikely, usually may be controlled with Levarterenol Bitartrate Injection, U.S.P. Caffeine and Sodium Benzoate Injection, U.S.P. may be used to counteract CNS depressant effects. The usefulness of dialysis has not been determined.

Dosage and Administration: Ativan is administered orally. For optimal results, dose, frequency of administration and duration of therapy should be individualized according to patient response. To facilitate this, scored 1.0 and 2.0 mg tablets are available.

The usual range is 2 to 6 mg/day given in divided doses, the largest dose being taken before bedtime, but the daily dosage may vary from 1 to 10 mg/day. For anxiety, most patients require an initial dose of 2 to 3 mg/day given b.i.d. or t.i.d.

For insomnia due to anxiety or transient situational stress, a single daily dose of 2 to 4 mg may be given, usually at bedtime.

For elderly or debilitated patients, an initial dosage of 1 to 2 mg/day in divided doses is recommended, to be adjusted as needed and tolerated.

The dosage of Ativan should be increased gradually when needed to help avoid adverse effects. When higher dosage is indicated, the evening dose should be increased before the daytime doses.

How Supplied: Ativan (lorazepam) is available in scored 1.0 and 2.0 mg tablets in bottles of 100.

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ANNOUNCING PUBLICATION OF

THE CHRONIC MENTAL PATIENT

Report of a Conference held in January 1978
Edited by
John A. Talbott, M.D.

with contributions by James T. Barter, M.D., Paul R. Friedman, J.D., Raymond Glasscote, M.A., Trevor D. Glenn, M.D., Norman V. Lourie, M.S.W., W. Walter Menninger, M.D., Arthur T. Meyerson, M.D., Kenneth Minkoff, M.D., Samuel Muszynski, M.S.W., Lucy D. Ozarin, M.D., Gordon L. Paul, Ph.D., Ronald Peterson, Steven S. Sharfstein, M.D., Judith Clark Turner, Jane Bloom Yohalem, J.D.

This 277-page Report of the Conference, sponsored by APA and President Carter's Commission on Mental Health, identifies the chronic mental patient population, spells out where they are and what their needs and rights are. It specifies what programs work and what programs do not work in meeting the needs of these patients. It elaborates on the obstacles to implementing effective programs and the economic issues involved. It delineates the pros and cons of case management and specifies responsibility for coordinating, implementing, and monitoring services to chronic mental patients.

Finally, it proposes a *Call to Action* which opens with this statement: "There is no more urgent concern than the needs of the chronic mentally ill who suffer from severe, persistent, or recurrent mental illnesses with residual social and vocational disabilities. As a result of the deinstitutionalization programs of the past decade and the continuing growth of high risk populations that generate chronically ill, the problems associated with the care of these patients constitute a national crisis."

Since the Conference, the APA Assembly and the Board of Trustees have both approved the "Call to Action" which calls upon the APA to take the lead in undertaking programs to elevate the prestige and value of work with chronic mentally ill patients.

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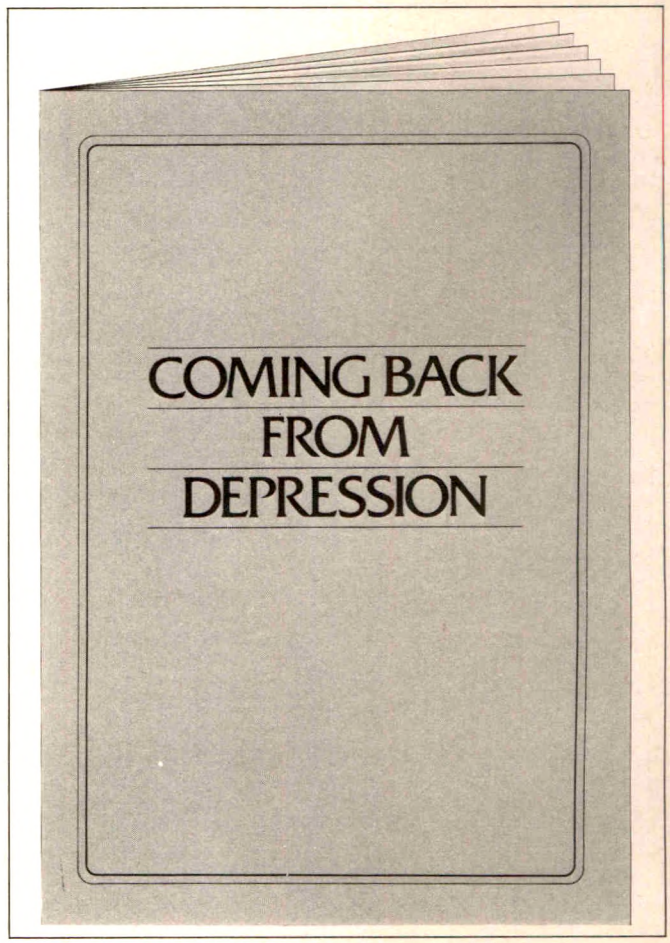
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CONTRAINDICATIONS: Contraindicated in patients with a history of hypersensitivity to amitriptyline. Do not administer concomitantly with a monamine oxidase (MAO) inhibitor since hyperpyretic crises, severe convulsions, and deaths occurred when tricyclic antidepressants and MAO inhibitors were administered simultaneously. When an MAO inhibitor is to be replaced by amitriptyline, allow 14 days to elapse after discontinuation of the former and then initiate amitriptyline cautiously and gradually increase dosage until optimum response is achieved. Amitriptyline is not recommended for use during the acute recovery phase following myocardial infarction.

WARNINGS: Amitriptyline may block antihypertensive action of guanethidine or similarly acting drugs. Use with caution in patients with history of seizures and (because of atropine-like action of amitriptyline) in patients with history of narrow-angle glaucoma (even average doses may precipitate an attack), increased intraocular pressure, or urinary retention. Closely watch patients with cardiovascular disorders since, in addition to having caused myocardial infarction and stroke, tricyclic antidepressants (including amitriptyline) particularly with high dosage have been reported to produce arrhythmias, sinus tachycardia, and prolongation of conduction time. Close supervision is required when amitriptyline is given to hyperthyroid patients or those on thyroid medication. Amitriptyline may impair mental and/or physical abilities required for performance of hazardous tasks such as operating machinery or driving a motor vehicle. Bear in mind that in patients who may use alcohol excessively the potentiation may increase the danger inherent in any suicide attempt or overdosage.

Usage in Pregnancy: Safe use during pregnancy and lactation has not been established; therefore, in administering the drug to pregnant patients, nursing mothers, or women who may become pregnant, weigh the possible benefits against the possible hazards to the mother and child. Animal reproduction studies have been inconclusive, and clinical experience has been limited.

Usage in Children: At the present time, not recommended for patients under 12 years of age because of lack of experience with use in children.

PRECAUTIONS: Schizophrenic patients may develop increased symptoms of psychosis; patients with paranoid symptomatology may have an exaggeration of such symptoms; manic depressive patients may experience a shift to the manic phase. In these circumstances the dose of amitriptyline may be reduced or a major tranquilizer may be administered concurrently.

Closely supervise and carefully adjust dosage in concomitant use with anticholinergic or sympathomimetic drugs, including combination of epinephrine and local anesthetics. Paralytic ileus may occur with concomitant use of tricyclic antidepressants and anticholinergic-type drugs. Caution is advised if used concurrently with large doses of ethchlorvynol since transient delirium has been reported when one gram of that drug and 75 to 150 mg of amitriptyline HCl were administered. Amitriptyline may enhance response to alcohol and the effects of barbiturates and other CNS depressants. The possibility of suicide in depressed patients remains during treatment and until significant remission occurs. Potentially suicidal patients should not have easy access to large quantities of the drug. Prescriptions should be written for the smallest amount feasible. Limit concurrent administration of this drug and electroshock therapy to patients for whom it is essential since the hazards associated with such therapy may be increased. Discontinue this drug, when possible, several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported. Use amitriptyline with caution in presence of impaired liver function.

ADVERSE REACTIONS: NOTE: A few of the adverse reactions listed below have not been reported with this specific drug, but each of the reactions should be considered when administering amitriptyline because of pharmacological similarities among tricyclic antidepressants.

Cardiovascular: Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, and stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness; tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; and syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, increased intraocular pressure, constipation, paralytic ileus, urinary retention, and dilatation of the urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, and edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, leukopenia, eosinophilia, purpura, and thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, and black tongue. Rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, and elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, and alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache, and malaise. These are not indicative of addiction.

For full prescribing information, consult package insert.

HOW SUPPLIED: Available for oral administration in tablets providing 10, 25, 50, 75, and 100 mg amitriptyline hydrochloride. The 10, 25, and 50 mg tablets are available in bottles of 100 and 1000. The 75 and 100 mg tablets are available in bottles of 100.

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Extrapyramidal Effects of Selected Antipsychotic Agents*[2]

DRUG	EXTRAPYRAMIDAL EFFECTS
acetophenazine	moderate
chlorpromazine	moderate
chlorprothixene	moderate
fluphenazine	high
haloperidol	high
perphenazine	high
prochlorperazine	high
thioridazine **(MELLARIL®)**	low
thiothixene	moderate
trifluoperazine	high

*Based on antipsychotic dosage ranges

References

1. Van Putten T: The rising rehospitalization rate of psychiatric patients. Scientific Exhibit, American Psychiatric Association, 130th Annual Meeting, Toronto, Canada, May 2-6, 1977.

2. Byck R: Drugs and the treatment of psychiatric disorders, in Goodman LS, Gilman A (eds): *The Pharmacological Basis of Therapeutics*, ed. 5. New York, Macmillan Publishing Co, Inc, 1975, pp 170-171.

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Contraindications: Severe central nervous system depression, comatose states from any cause, hypertensive or hypotensive heart disease of extreme degree.

Warnings: Administer cautiously to patients who have previously exhibited a hypersensitivity reaction (e.g., blood dyscrasias, jaundice) to phenothiazines. Phenothiazines are capable of potentiating central nervous system depressants (e.g., anesthetics, opiates, alcohol, etc.) as well as atropine and phosphorus insecticides; carefully consider benefit versus risk in less severe disorders. During pregnancy, administer only when the potential benefits exceed the possible risks to mother and fetus.

Precautions: There have been infrequent reports of leukopenia and/or agranulocytosis and convulsive seizures. In epileptic patients, anticonvulsant medication should also be maintained. Pigmentary retinopathy, observed primarily in patients receiving larger than recommended doses, is characterized by diminution of visual acuity, brownish coloring of vision, and impairment of night vision; the possibility of its occurrence may be reduced by remaining within recommended dosage limits. Administer cautiously to patients participating in activities requiring complete mental alertness (e.g., driving), and increase dosage gradually. Orthostatic hypotension is more common in females than in males. Do not use epinephrine in treating drug-induced hypotension since phenothiazines may induce a reversed epinephrine effect on occasion. Daily doses in excess of 300 mg should be used only in severe neuropsychiatric conditions.

Adverse Reactions: *Central Nervous System*—Drowsiness, especially with large doses, early in treatment; infrequently, pseudoparkinsonism and other extrapyramidal symptoms; rarely, nocturnal confusion, hyperactivity, lethargy, psychotic reactions, restlessness, and headache. *Autonomic Nervous System*—Dryness of mouth, blurred vision, constipation, nausea, vomiting, diarrhea, nasal stuffiness, and pallor. *Endocrine System*—Galactorrhea, breast engorgement, amenorrhea, inhibition of ejaculation, and peripheral edema. *Skin*—Dermatitis and skin eruptions of the urticarial type, photosensitivity. *Cardiovascular System*—ECG changes (see *Cardiovascular Effects* below). *Other*—Rare cases described as parotid swelling.

It should be noted that efficacy, indications and untoward effects have varied with the different phenothiazines. It has been reported that old age lowers the tolerance for phenothiazines; the most common neurologic side effects are parkinsonism and akathisia, and the risk of agranulocytosis and leukopenia increases. The following reactions have occurred with phenothiazines and should be considered whenever one of these drugs is used. *Autonomic Reactions*—Miosis, obstipation, anorexia, paralytic ileus. *Cutaneous Reactions*—Erythema, exfoliative dermatitis, contact dermatitis. *Blood Dyscrasias*—Agranulocytosis, leukopenia, eosinophilia, thrombocytopenia, anemia, aplastic anemia, pancytopenia. *Allergic Reactions*—Fever, laryngeal edema, angioneurotic edema, asthma. *Hepatotoxicity*—Jaundice, biliary stasis. *Cardiovascular Effects*—Changes in terminal portion of electrocardiogram, including prolongation of Q-T interval, lowering and inversion of T-wave, and appearance of a wave tentatively identified as a bifid T or a U wave have been observed with phenothiazines, including Mellaril (thioridazine); these appear to be reversible and due to altered repolarization, not myocardial damage. While there is no evidence of a causal relationship between these changes and significant disturbance of cardiac rhythm, several sudden and unexpected deaths apparently due to cardiac arrest have occurred in patients showing characteristic electrocardiographic changes while taking the drug. While proposed, periodic electrocardiograms are not regarded as predictive. Hypotension, rarely resulting in cardiac arrest. *Extrapyramidal Symptoms*—Akathisia, agitation, motor restlessness, dystonic reactions, trismus, torticollis, opisthotonus, oculogyric crises, tremor, muscular rigidity, and akinesia. *Persistent Tardive Dyskinesia*—Persistent and sometimes irreversible tardive dyskinesia, characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and sometimes of extremities may occur on long-term therapy or after discontinuation of therapy, the risk being greater in elderly patients on high-dose therapy, especially females; if symptoms appear, discontinue all antipsychotic agents. Syndrome may be masked if treatment is reinstituted, dosage is increased, or antipsychotic agent is switched. Fine vermicular movements of tongue may be an early sign, and syndrome may not develop if medication is stopped at that time. *Endocrine Disturbances*—Menstrual irregularities, altered libido, gynecomastia, lactation, weight gain, edema, false positive pregnancy tests. *Urinary Disturbances*—Retention, incontinence. *Others*—Hyperpyrexia; behavioral effects suggestive of a paradoxical reaction, including excitement, bizarre dreams, aggravation of psychoses, and toxic confusional states; following long-term treatment, a peculiar skin-eye syndrome marked by progressive pigmentation of skin or conjunctiva and/or accompanied by discoloration of exposed sclera and cornea; stellate or irregular opacities of anterior lens and cornea; systemic lupus erythematosus-like syndrome.

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Am J Psychiatry 136:6, June 1979

THE AMERICAN JOURNAL OF PSYCHIATRY

"Folk" Criteria for the Diagnosis of Mental Illness in Rural Laos: On Being Insane in Sane Places

BY JOSEPH WESTERMEYER, M.D., PH.D., AND RONALD WINTROB, M.D.

"Folk" criteria for identifying the mentally ill, as distinguished from folk theories about the causes of mental illness, have been comparatively neglected in cultural psychiatry. The authors describe the criteria by which villagers in Laos labeled 35 subjects as baa *(insane). Unprovoked assaultive or destructive behavior, social isolation, self-endangerment due to neglect of personal needs, nonviolent but socially disruptive or inappropriate behavior, and inability to do productive work were found to be important folk criteria. The authors emphasize that folk criteria for mental illness are determined primarily by the persistence of socially dysfunctional behavior rather than by disturbances in thought and affect.*

"FOLK" THEORIES regarding mental disorder have been studied in several societies (1-6). Relatives of psychotic patients have also been surveyed regarding their attitudes toward the mentally ill and their treatment (7-9). On the other hand, folk criteria for labeling a particular person as mentally ill have been comparatively neglected.

The study of the mentally ill in Laos provided an opportunity to determine such folk criteria *not* influenced by psychiatrists or other mental health professionals, since there were no such practitioners there. Diagnoses of mental illness were made by community members. The purpose of this study was to clarify the criteria used for arriving at this form of social labeling or "folk diagnosis."

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This study was supported in part by the Minnesota Medical Foundation and the International Programs Office at the University of Minnesota.

Drs. Jerry Kroll, Lloyd Sines, and Vicente Tuason collaborated in assigning psychiatric diagnoses, Mr. Tou Fu Vang and Ms. Beth Stone assisted in tabulating and analyzing the data, and Mr. Phimpha assisted in collecting the data.

METHOD

We surveyed 27 villages in rural Laos in 1975 to identify persons socially labelled as *baa*, or insane. Characteristics of the 35 subjects so designated are summarized in table 1. Psychiatric diagnoses were assigned independently by a panel of 4 experienced clinicians to whom summaries of the clinical data on each case were provided. This procedure has been described elsewhere (10). For each *baa* subject, a mean of 6.9 informants were interviewed, consisting of family, neighbors, village elders, and Buddhist monks. They were asked on what basis they assigned the social label of *baa* to the individual; each answer was probed for further details. There were no health workers or individuals with more than 10 years of formal education among the informants. All informants were members of the Lao ethnic group.

Data were obtained from each *baa* person's informants in a group setting. Any given response or "criterion" was counted only once for each subject. That is, even though two informants might say that a given person was eating garbage, that item was counted only one time. Items related by the informants were grouped using the informants' frame of reference insofar as possible.

The least number of responses reported for a given *baa* subject was 5 and the most was 19, with a mean of 12.4 (standard deviation of 3.9) and a median of 12 items per subject. The 3 subjects with the lowest number of items (i.e., 5, 6, and 7) had fewer than the average number of informants, and these informants did

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TABLE 1
Demographic and Clinical Characteristics of the 35 *Baa* Subjects

Characteristic	Number of Subjects
Age (years)	
Under 20	7
20–29	5
30–39	9
40–49	9
50–59	2
Over 59	3
Sex	
Male	18
Female	17
Marital status	
Single	18
Married	5
Married according to common law	2
Separated or divorced	9
Widowed	1
Education	
Illiterate	16
1 to 8 years	12
9 to 12 years	7
Ethnicity	
Lao	30
Tribal (all Laoteung)	4
Chinese	1
Psychiatric diagnosis	
Functional psychosis	24
Organic psychosis	9
Borderline condition	2
Age at onset of illness	
Under 20	9
20–29	12
30–39	9
40–49	2
50–59	0
Over 59	3
Duration of illness (years)	
Under 1	6
2–5	14
6–10	8
11–15	5
Over 15	2

not know the *baa* individuals well. These 3 subjects were vagrants whose family members could not be located. In the case of the 3 subjects with the highest ratings (2 with 18 items and 1 with 19) the high ratings were not related to the number of informants. These 3 subjects had extremely high scores on several clinical rating scales for psychopathology; they were severely impaired socially, and their care constituted a tremendous burden for their family and extended kinship.

Data on folk diagnosis and the onset of unusual or problematic behavior were obtained from family members who shared the same household with the *baa* subject. In addition, these same data were obtained from relatives not sharing the same household, as well as from neighbors, merchants, and other people in the neighborhood or village. The reports given by these different groups were then compared. (Information from family members could not be obtained for 6 people who were no longer living in their home village or close to kinship members.)

FINDINGS

Categories of Criteria

These categories are presented in the approximate order that informants tended to follow in relating the items.

Danger to others and property. Unprovoked assaultive behavior was reported as a criterion in 19 cases. Fourteen *baa* subjects attacked others with their fists or feet, 2 used weapons such as a board or knife, and 3 used both methods. Two women severely beat their own children for no apparent reason, and a man struck his small grandchildren on several occasions.

Eight subjects had threatened others verbally; 3 of them had not actually harmed anyone.

Informants for 8 subjects specified destruction of property as a criterion for the *baa* label. Damage was extensive, as the following case example illustrates:

> A 24-year-old man suddenly became argumentative with his family. He began throwing furniture and household items around the house. During the next few days, he threw furniture out of doors and windows. Having emptied the house of the furniture, he began ripping up clothes and bedding. By the time he could be restrained, he had torn down one wall of the family's wood frame house.

In each of these 8 cases there were also reports of unprovoked violence toward others and/or self. Instances of violence toward property not reported as a criterion for being *baa* (but subsequently elicited by direct questions) tended to be rather minor (e.g., pounding the side of a house with a board but not creating major damage), compared with reported cases (e.g., burning a family member's clothes, destroying furniture, burning down a kitchen).

There was harm by neglect in the case of a woman who became *baa* shortly after the birth of a daughter. Since she fed and cared for the infant irregularly, the child did not gain weight and gradually became weak, thin, and ill. Family members tried to assume care of the child, but the mother vehemently refused to relinquish the care to others. The child died of malnutrition during the second year of life.

Only 1 person, a 22-year-old man who had been *baa* for 4 years, had assaulted animals; he killed a dog and a cat by strangling them. This man had also threatened to kill himself, had assaulted several people in the village, and had threatened to knife children who teased him.

Danger to self. While some reported behaviors were apparently not intended by the *baa* person to be dangerous, they nonetheless produced a risk of injury to 26 subjects. Examples of self-risk were as follows:

> Once he drank mosquito poison saying it was a medicine for his cough. Sometimes he goes down the street as though he were a car or a truck himself, ignoring the traffic.

> Recently she climbed an electrical pole and danced and sang up there near the wires like she was a monkey. [The wires were 220 volts and known to be dangerous.]

Informants reported "sleeping in the open" as a criterion for assigning a *baa* diagnosis to 8 subjects. This behavior was cause for marked social concern in Laos, since exposure with subsequent fever and death was known as a common cause of death among *baa* individuals.

There were 5 reports that the *baa* individual's behavior had led to self injury, albeit sometimes in an indeliberate way. For example, one man who believed that he was an "important general" was severely beaten by the military police after he had stolen a jeep and driven it until it ran out of gas.

There were 3 cases of eating garbage or other unsanitary material. All 3 of these *baa* subjects had expressed the idea that people were trying to poison them. Thus they did not beg for food (as vagrant *baa* people usually did) or accept food from family or neighbors. A typical case was as follows:

> Last week when a car struck and killed a cat, he tore it open and ate it. He drinks filthy water and eats out of garbage pails. Once he made a funeral pyre for a dead goat as though it were a human [the Buddhist Lao cremate their dead], and then he ate the goat.

One man attempted to kill himself by not eating. He had been acutely psychotic for 10 days when encountered during the course of this study and was severely dehydrated, comatose, and near death.

Running away did involve some risk to *baa* persons themselves, but none of the subjects in this study sustained serious injury in this manner. Moreover, reports of serious injuries or death resulting from running away were rare. Since this type of behavior appeared to be more a social problem than a risk to self, it is included in a subsequent section.

Nonviolent socially disruptive behavior. Violence toward self, others, and property produced problems for the family and community of these *baa* subjects. Other behaviors, nonviolent in themselves, also caused a variety of problems for family, community, and society.

The most common complaint in this category was that the *baa* subject refused or was unable to work; this occurred in 27 cases. (Later direct questioning revealed that 34 of the 35 subjects were unproductive or underproductive.) Since the per capita gross national income was $70[1] in U.S. currency this behavior had major implications for the family and community in terms of survival.

Informants complained of *baa* subjects "wandering or running away" in 19 cases. Some wandered along roadways, eventually stopping in villages where people would shelter them until their relatives could be contacted and come and get them. Other individuals wandered into the forest, where they were at risk from starvation and dangerous animals. Their absence caused considerable distress to their families, in part because violent death would create both a spiritual and a social "stain" on the family. (It is widely believed in rural Laos that people who die violently may return as malevolent spirits to torment the family.) Families and neighbors often expended considerable time and effort in retrieving them.

"Yelling or screaming at night" was a complaint in 7 cases. This was a socially disturbing behavior because most Lao homes are built in such a way that screaming or yelling can easily be heard throughout an entire village.

Four subjects—all young men—created social crises that led to their being jailed for illegal activities. One 28-year-old *baa* man went to the homes of high government officials and declared that he was waiting to be paid. He was jailed for 15 days on suspicion that he was a spy. In another case, a 20-year-old man was a paratrooper when he first became *baa*. He behaved as though he were a high-ranking officer. He was placed in the military jail because he refused to obey orders and pretended that he was an officer.

Problems in speech and communication. There were 26 reports regarding incomprehensible or nonsensical speech. These involved speech that was not intelligible to the informants (16 cases), talking or singing to oneself while ignoring others (7 cases), and incessant talking (3 cases). One informant described a *baa* subject as follows:

> He talks to himself all the time and laughs to himself. If you ask why he is laughing, he says, "I wasn't laughing."

Angry yelling and cursing (*daa*) were reported in 18 cases. The form of some curses was appreciably different from and taken more seriously than in the United States in that they were curses invoking spirit possession or soul loss. Cursing in these cases was usually indiscriminate, with curses yelled at anybody within earshot.

In 7 cases informants reported that *baa* subjects did not respond to questions or to attempts to initiate conversation. While the subjects remained mute in response to questions, they did verbalize at other times. This included talking or singing to themselves, yelling curses, or screaming at night.

Three men spoke in a foreign language (English or French) that the local people could not understand. All 3 had become fluent in these languages before becoming *baa*. Speaking in English or French was viewed by peers as a means of excluding them and as a form of snobbishness, since few Lao villagers spoke European languages.

There was one complaint of "not telling the truth." This particular subject did not realize any gains from her supposed lying, and it seems likely that she fabri-

[1] 1969 data provided by the Information Service of the Population Reference Bureau, Washington, D.C.

cated answers in order to respond to people's questions (albeit in a confused fashion), rather than deliberately to provide misinformation.

Socially dysfunctional behavior. This type of behavior was viewed by informants as strange, unexpected, or embarrassing. Such behavior led to social alienation from family and community, although it did not involve violence, crisis, or special problems that demanded social intervention.

There were 34 reports of behavior that was inappropriate to the social context in which it occurred. Such behavior attracted social attention to the subjects and was often an embarrassment to observers or to the *baa* person's family. Examples were as follows:

> When she goes around the town she does a *nob vai* [formal greeting of respect] to everyone, even strangers whom she has never previously seen.

> He pretends he is a famous professor teaching people, telling them historical tales all the time.

> When she goes with the family to dig up roots from the forest, she dresses up in her very best clothes as though she were going to a temple festival. Then if she actually goes to a formal ceremony at the temple, she wears her oldest and poorest clothes.

Nakedness in public was reported for 4 women.

Survival in the villages of Laos requires cooperative effort and dependence on others, since no one person can perform all of the work necessary to ensure individual or group subsistence. Tasks are divided by age and sex. In this milieu, sociability with fellow villagers is accorded high value. In 29 cases there were reports of subjects isolating themselves from others.

> She takes her food away from the table and eats by herself in another room, or she sits down to eat only after the rest have left the room.

> She lives by herself in a lean-to shelter instead of with her family.

> He does not greet even his relatives when he sees them along the road.

Self-neglect, in terms of long-standing inattention to personal cleanliness and grooming, was mentioned as a sign of *baa* behavior for 19 subjects. Most of these subjects had not bathed or groomed for months or even years.

In 11 cases the subjects appeared to work, often with considerable effort repeated over long periods. However, their activities produced no observable economic benefit.

> He always carried 20 or 30 kilograms of scrap iron draped over his shoulders and down his back. He begs for it [the iron], as though he were a monk going around begging for rice. [In Laos Buddhist monks receive ritualized rice offerings each morning at sunrise.]

Impaired psychological function. There were 25 complaints of "wrong ideas" or "false beliefs" that did not conform to reality, descriptions closely approximating the psychiatric definition of delusions. Common examples were as follows:

> He says that someone stole his wife and children, but he has never been married and has no children.

> He thinks that people have put an electric current into him and that they are trying to poison him. He also thinks that people are sending the police after him to harm him.

> He thinks that he might be killed by others, especially at night. So he keeps a knife by his bed, and he keeps the lamp on all night long. He stays awake and vigilant until sunrise.

Informants reported that 9 individuals had "no knowledge" regarding common knowledge or current issues of local or regional concern. This included plans about farming or animal husbandry or popular social issues such as the election of a village chief.

In 5 cases there were reports of an inability to remember things, such as recent occurrences or recent conversations.

Four subjects were reported to have perceptions not observed or validated by others—an equivalent of hallucinations. A typical description was as follows:

> She said that the house smelled bad and ran away. Later she said food smelled bad and threw it away or refused water, saying there was urine in it.

Perceptual disorder was an underreported phenomenon, given our finding from direct questioning and from mental status examination that many *baa* subjects had been or were currently experiencing hallucinations.

There were eight cases in which informants commented on various other items related to intellect. Six included the subject's inability to reason, comprehend, pay attention, or anticipate. Two involved constant worry and recurrent, pessimistic thoughts. Reports included the following:

> He cannot pay attention to anything for more than a few minutes.

> She does not concern herself about anything or make plans for her future.

Affective disturbance. Ten subjects were reported to have frequent, wide swings in mood.

> She is happy, then cries, then laughs, then yells angrily—all in less than an hour.

Constant or frequent anger was reported in 9 cases, sadness or tearfulness in 8 cases, and fearfulness in 5 cases. Examples were as follows:

He is angry all the time, hitting things, throwing things around.

She is very much afraid of dying, especially when it is dark or when there is a thunderstorm.

He sometimes cries for days at a time.

Five subjects showed affect that was not appropriate to their speech or the social context.

He laughs for no reason. When he says, "I'm afraid I'm going to die," he smiles.

Somatic signs and symptoms. Insomnia, which occurred in 19 cases, was the most common complaint and included the full range of sleep disturbances: difficulty falling asleep, restless sleep, early morning wakening, and staying awake all night. In some cases insomnia was noted only early in the *baa* condition, but in most cases it persisted for months or years.

There were 8 cases of weight loss and 2 of weight gain, usually occurring only in the early months of the *baa* condition. Seven subjects were noted to refuse food.

There were 4 cases of weakness; 3 cases each of "red staring eye" or "red face" and temporary "blindness" or "deafness"; 2 cases each of back pain, constipation, convulsions, fatigue, and headache; and 1 case each of "bad feeling in the heart," dizziness, eye pain, inability to swallow, and incontinence of urine or feces.

Family-Community Differences in Folk Diagnosis

Differences in chronology. In 29 cases information on *baa* labeling was obtained from both family and nonfamily members. Twenty-three of these subjects (79%) were socially defined as *baa* by both family and nonfamily members within one year of each other.

In 4 cases (14% of the 29) family and community informants did not agree on the time of onset of the *baa* condition. These 4 cases were diagnosed by the panel of clinicians as "functional psychosis, schizophrenic type." The onset in these cases was gradual rather than acute. In all 4 cases family members reported a longer duration of the *baa* condition than did others (2 years longer in 2 cases and 3 years longer in 2 cases).

Analysis of the 4 cases indicated that behavioral disturbances within the family had been present for 2 or 3 years before unusual events were recognized by the community. These disturbances included throwing food, prolonged crying, and argumentativeness with family members. Three of these four were women who lived within large kinship systems, so that the deterioration in their household and gardening activities was not publicly evident.

Differences in folk diagnosis. Family members and community informants disagreed on the diagnosis in 2 cases (although they agreed on the manifestations and the duration of the condition). These cases involved a 15-year-old woman and a 19-year-old man. Their disorders had been of comparatively short duration (5 and 3 years, respectively). Both subjects belonged to large kinship groups in which most members were literate. Family members devoted considerable time and effort to finding a way to help the *baa* individual. Both of these subjects were diagnosed by the psychiatric raters as having a "functional psychosis, schizophrenic type." The differences in folk diagnosis were in the specificity of labeling by the family members, who considered 1 subject to be suffering from *seah cheet* ("lost heart" or emotional problem) and the other to be *khiet* (a self-centered or "spoiled" adolescent), whereas several community informants defined both subjects as *baa*. In both these instances the family folk diagnosis indicated less disturbance and a more optimistic prognosis.

DISCUSSION

Characteristics of Folk Criteria

The findings of this study demonstrate that folk diagnosis of mental disorder in Laos is made not on the strength of one criterion, but several. In the average case a dozen factors were reported. The presence of only one or a few of these factors was not sufficient evidence to assign a social label of *baa*.

No one criterion was a sine qua non for the *baa* label. However, several criteria were present among at least two-thirds of the 35 subjects. These included socially inappropriate behavior (in 34 of the 35 cases), social isolation (29 cases), inability to work (27 cases), risk of violence to self (26 cases), incomprehensible speech (26 cases), and erroneous ideas or "delusions" (25 cases). Most of the cases were also characterized by some form of affective abnormality, somatic symptoms, and violence against others. These criteria overlap to a considerable extent with folk criteria noted by the anthropologist Edgerton in four East African societies (1) and by psychiatrists Gaviria and Wintrob among Puerto Ricans in Connecticut (2). These investigators noted the following socially disruptive characteristics of mental illness: aggressive behavior, assault, or destruction of property; abusing people verbally or screaming; disorder of thought or communication, such as "talking nonsense"; bizarre behavior and disorders of locomotion, such as "running wildly," going naked, risking injury or injuring self; and disorder of orientation and memory.

Notable by its absence is the lack of reports regarding hallucinations, despite the fact that in the present study hallucinations were found to be present among most of the 35 subjects. Gaviria and Wintrob did not include hallucinations as a folk criterion among Puerto Ricans. In his interviews with 500 East Africans of four different tribes, Edgerton counted only 5 reports of hallucinations among 1,926 responses. Edgerton remarked on the rarity of hallucinations as a criterion,

stating that perhaps hallucinations are "not sufficiently visible to the observer."

A review of these folk criteria from Laos, as well as those reported from other cultural areas, indicates that most criteria are social. Of the 434 criteria in this study, 285 (66%) related to behaviors that are socially dangerous, disruptive, or disturbing. By contrast, only 88 criteria (20%) were psychological; 51 of these involved cognition and 37 affect. Finally, 61 items (14%) involved somatic symptoms. Thus the factors of greatest importance to family and community in assigning a *baa* label were social. This might imply that family and community are apt to see such disorders as primarily social rather than psychological or biomedical. This folk perspective might be a source of conflict with *baa* persons themselves (who are most apt to complain of psychological symptoms or somatic discomfort), as well as with physicians (who may focus on more cognitive and somatic factors).

Behavioral, psychological, social, and somatic criteria were not the only factors that contributed to the *baa* label. There was also variance based on who was applying the label. Generally, family members and community members agreed on the diagnosis; but if differences existed, family members favored diagnoses or labels associated with a more favorable prognosis. This was true despite the fact that the family was usually more affected by the person's condition than was the community and, in some cases, noted the onset of the condition earlier. Edgerton (1) reported a similar, almost fanatical optimism among the family members of a psychotic teenager in East Africa.

Sociodynamics of Diagnosing Insanity

Lao villagers were not overly zealous in applying the term *baa* to their neighbors and family members. On the contrary, they first required evidence of major disability in several areas of functioning. In some cases the signs of disability had to be present for years before the *baa* diagnosis was validated. This observation raises the question, Why was the *baa* social label so reluctantly applied?

From the community's perspective, it was not in the interests of neighbors and family friends to allow anybody to attain the *baa* label. Possession of this social identity allowed certain socially disturbing behaviors that would lead to the person's expulsion from the village if he or she were viewed as sane. Such behaviors included yelling at night and keeping the village awake and assaulting village members and damaging their property. Refusal to accept expulsion from the village or repetition of such behaviors by a sane person could (and sometimes did) end in assassination (11–13). If an individual were defined as *baa*, however, the socially responsible course of action was not exile or assassination. Instead, people were expected to take care of the *baa* individual, hoping for improvement in his or her condition. Consequently, designation of the *baa* label had important implications for the community, just as denial of this identity in the face of certain disturbing behaviors could result in major, potentially divisive social outcomes (such as expulsion or assassination). The social dilemma in such circumstances was not which alternative was all good or all bad, but rather which was least bad.

Family members, perhaps even more than others in the community, appeared unwilling to concede that a person was *baa*. This may be due to the fact that there was more at stake for kin than for nonkin. Since *baa* people often had severe and chronic impairment, the family had to expend time, energy, and material resources in caring for their disturbed relative. They also lost the economic assistance of the *baa* person, who was usually an adult in his or her productive years. While folk etiologies gave no explicit recognition to genetic or family influences in the development of mental illness (14, 15), *baa* kin were a negative factor when marriageability was considered. *Baa* family members also signified a moral stigma for the family, since Lao cosmology explained such misfortunes as punishment for malfeasance by family members.

Edgerton (1) has contended that insanity as a status reduces a person's rights within the social group. That is true to the extent that such people's opinions are not taken seriously and they cannot participate effectively in the political activity of the village. However, the *baa* social identity does give that person the right to be provided with food, shelter, clothing, and humane care despite the fact that he or she is unproductive or underproductive. It also allows an exceptional reprieve from stringent punishments that would be enacted for certain behaviors if the person were considered sane. Thus this identity allows neglect of certain ordinary rights while providing access to other extraordinary rights.

Implications

Studies of folk criteria for mental disorder have made use of several different methods of sampling and data collection (1–9). Investigators have employed their own schemata for classifying their data. In addition, these investigators have come from different countries, as well as from different disciplines. Despite such variation in the studies, the similarity in the findings indicates a number of common features of folk criteria for mental disorder around the world. One interpretation of this similarity is that common explanations of mental disorder—such as spirit possession, soul loss, or divine punishment—have diffused around the world. Another interpretation is that mental disorder—like tuberculosis, cancer, or homicide—has a core biological and/or psychosocial basis. Studies reported thus far do not indicate which interpretation is more likely to be accurate (or whether there are elements of both interpretations). We hope that future systematic research on this subject will provide data that will allow more conclusive cross-cultural comparisons.

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β-Endorphin and Naloxone in Psychiatric Patients: Clinical and Biological Effects

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The authors conducted single- and double-blind studies of the responses of 7 chronic male schizophrenic patients to 10 mg of naloxone. BPRS ratings were made before and 6 hours after the injection; ACTH blood levels were determined before and 1½ and 6 hours after injection. Statistically significant improvement of psychotic behavior occurred after 6 hours. The greatest improvement occurred in the patient who showed the most pronounced diurnal variation of ACTH levels, and there was no improvement in the patient who had no diurnal changes. Prolactin plasma levels following endorphin injections were apparently dose-dependent and peaked at approximately 30 minutes. The mean half-life of elimination of exogenous β-endorphin was between 12 and 35 minutes. The authors theorize that positive and negative behavioral responses to naloxone depend—as possibly do many placebo responses in general—on the relative stress produced by experimental or therapeutic interventions.

THE ENDORPHINS have been called "the body's own opiates" because they attach themselves to the opiate receptors and are specifically displaced from these receptor sites by the opiate antagonist naloxone. Gunne and associates (1) reasoned that if schizophrenia were due to an excess of endorphins, administration of naloxone should reduce schizophrenic manifestations. They found dramatic reduction of hallucinations in 4 of 6 persistently hallucinating schizophrenic patients. Volavka and associates (2), Davis and associates (3), and Kurland and associates (4) failed to confirm these findings.

On the other hand, Emrich and associates (5), in a well-conducted double-blind study, and Berger and associates (6) obtained results similar to those of the Gunne study (1). No explanation has been offered for the absence of response in 2 of the 6 schizophrenics in the Gunne study and in the 8 of 20 patients reported by Emrich and associates (5) who showed less than 5% improvement in the target symptoms of hallucinations, delusions, and ideas of reference and the lack of response of several patients in the Berger investigation (6).

Despite their overall negative reports, Davis and associates (3) noted that the "unusual thought content" item of the Brief Psychiatric Rating Scale (BPRS) was significantly reduced by naloxone. Volavka and associates (2) reported improvement in 2 of 7 patients and Kurland and associates (4) stated that "only 2 of 8" showed complete disappearance of symptoms (compared with 5 of 12 placebo patients).

Could there be a rational explanation of why some patients respond to naloxone and others do not? An equally important but neglected question is how to account for the positive placebo responders.

β-ENDORPHIN RESPONSE HYPOTHESIS

It is our contention that patients should be dealt with as specific individuals rather than being submerged statistically in a group response. Perhaps if this were done it would explain the otherwise anomalous finding that the same individual responds on one occasion but does not do so on another occasion. Placebo responsivity is assumed to be due to suggestibility, conditionability, or other psychological factors, although extensive investigations have failed to establish placebo-responsiveness as an identifiable personality trait.

Our hypothesis is that not only the response to the active substance (in this case, naloxone) but also the placebo response can be accounted for by a measurable physiological factor, i.e., increased endorphin secretion.[1]

According to Mains and Eipper (7) and Roberts and

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[1]We previously had attempted to measure the endorphin levels in patients receiving electroconvulsive therapy or acupuncture or who were under hypnosis, but we found the levels to be below the sensitivity of our radioimmunoassay method. We plan to repeat this work using the ACTH-endorphin yoke index.

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Herbert (8), there is a common precursor peptide that gives rise to both adrenocorticotropin (ACTH) and β-endorphin. Guillemin and associates (9) have shown that β-endorphin and ACTH are concomitantly secreted by the pituitary in response to acute stress. They state, "Both hormones possess common and identical regulatory mechanisms . . . ." Thus it seems highly probable that because of the "yoking" of the two peptides, measurement of one would provide an index to the other. Since there is no reported specific technique for measuring β-endorphin at the level at which it is secreted by the organism (i.e., endogenously) but it is possible to measure ACTH levels under such circumstances, we have hypothesized that ACTH levels would be indicative of β-endorphin secretion.

Our hypothesis is that the individuals who clinically improve when given naloxone will have a specific, definable endorphin-response pattern. The stress of intravenous injection may similarly alter the endorphin-response pattern in some patients. Obviously, the same "placebo" factor might affect the patients given naloxone, but, if the drug is active, the test group would have a greater response than the control group.

Incidentally, this might account for Palmour and associates' findings (10). The stress response produced by dialysis (rather than the dialysis of a toxic substance) may alter the β-endorphin-response pattern. It would also account for the response of some patients but not of others, depending in part on the degree to which they are stressed.

The altered β-endorphin response to the stress of naloxone, or dialysis, may be an example of the general altered stress responsivity in schizophrenics to many life events. We believe the research results that follow are compatible with this hypothesis.

STUDY 1—NALOXONE, SINGLE-BLIND

Subjects

The base population consisted of 7 typical chronic schizophrenic patients with florid symptoms. Informed consent was obtained and the study was approved by the institutional review committee.

Patient 1 was a 60-year-old man, diagnosed as chronic schizophrenic, hebephrenic type, who had been continuously hospitalized for the last 30 years. He was regressed and withdrawn, with poor memory and concentration, marked poverty of ideation, and inappropriate affect. He spoke in monosyllables, required supervision in personal hygiene, and periodically refused to eat. He was usually disoriented as to time, place, and person and at times became catatonic.

Patient 2 was a 30-year-old man diagnosed as having paranoid schizophrenia with prominent depressive elements. He was hospitalized at age 23 following a suicide attempt with a rifle. He had only partial insight and poor judgment and was given to neologisms. He had difficulty in producing thoughts and ideas and had paranoid delusions that radio stations were stealing his songs. He admitted to hearing himself on the radio. Occasionally he displayed aggressive behavior toward other patients. The patient was refractory to therapy.

Patient 3 was a 31-year-old catatonic schizophrenic who was first admitted to the hospital at age 16 and had remained hospitalized continuously over the last 7 years. He exhibited periodic outbursts of violent behavior, was preoccupied with a sense of guilt and sought out "punishment," and was slow-moving and had a manneristic walk. He showed some insight, but his general judgment was poor.

Patient 4 was a 53-year-old paranoid chronic schizophrenic who had been hospitalized continuously for the past 16 years. He was well oriented and cheerful, functioned well within the hospital, ate and slept well, and maintained excellent personal hygiene. He was delusional, claiming that he was king of seven countries, and hallucinated virtually all the time, although he was not too troubled by it. A "click signal from God" directed his activities. He laughed inappropriately at times.

Patient 5 was a 28-year-old college-educated man who was diagnosed as paranoid schizophrenic. He was hospitalized for the first time five years previously after attempting suicide by jumping out of a moving car. He was well oriented but exhibited thought blocking, looseness of association, thought insertion, and diffusion. He talked a great deal about his "worthlessness," for which he felt depression and guilt. He had the delusions that there was a "machine in my brain" and that the staff was attempting to poison him. He had blunted affect and frequent auditory hallucinations.

Patient 6 was a 46-year-old man who was diagnosed as paranoid schizophrenic. He had been in and out of

TABLE 1
Numbers of Patients Showing Significant Change on the BPRS 6 Hours After Naloxone or Placebo Injection (10 mg)

Group, by BPRS Item	Response		
	Improvement	No Change	Deterioration
Study 1, single-blind			
BPRS tension item[a]			
Naloxone (N=6)	3	3	0
Placebo (N=4)	0	1	3
Study 2, double-blind			
Total BPRS[b]			
Naloxone (N=5)	5	0	0
Placebo (N=5)	1	3	1
Thought disturbance factor[c]			
Naloxone (N=5)	5	0	0
Placebo (N=5)	0	4	1
Hallucinatory behavior item[d]			
Naloxone (N=5)	5	0	0
Placebo (N=5)	0	5	0

[a] $\chi^2=6.27$, df=2, $p<.05$.
[b] $\chi^2=6.67$, df=2, $p<.05$.
[c] $\chi^2=10.0$, df=2, $p<.01$.
[d] $\chi^2=10.0$, df=1, $p<.01$.

TABLE 2
ACTH Plasma Levels Before and After Naloxone or Placebo Injection

Patient Number	ACTH Level (ng/ml) With 10 mg Naloxone: Pre-injection	After ½ Hour	After 6 Hours	With Placebo: Pre-injection	After ½ Hour	After 6 Hours
1	61	59	56	58	53	64
2	40	85	60	44	54	31
3	106	41	33	74	39	28
4	38	28	40	25	5	18
6	32	28	24	—	—	—

hospitals since 1960 and continuously hospitalized during the past six years. He had no insight and poor judgment and was suspicious, anxious, withdrawn, and occasionally verbally and physically aggressive. He had delusions such as having a million dollars in the bank, required minimal supervision of personal hygiene, and had occasional tactile and olfactory ("fish smell") hallucinations and frequent auditory hallucinations, which he claimed caused pain in his legs and made his heart stop.

Patient 7 was a 42-year-old man, chronic schizophrenic of the residual type, who had first been hospitalized when he was 16. He had no spontaneity of expression, was withdrawn, and had slow monotonous speech, blunted affect, and disordered thought processes. He had no insight and poor judgment and answered questions irrelevantly with "O.K., Mother," smiling inappropriately.

Method

Six patients were given 10 mg of naloxone intravenously and evaluated at baseline (immediately before injection) and at 3, 6, and 24 hours after injection. The evaluation used was the Brief Psychiatric Rating Scale (BPRS). Four of the same 6 patients were also evaluated in the same manner after saline injections.

The 10-mg dose of naloxone was selected because some investigators have been criticized for giving the therapeutic dose of .4 mg, which has been judged to be inadequate to produce a full response. There is also evidence that the optimal response of schizophrenics does not occur until approximately 6 hours after naloxone administration, which was confirmed in our study and was therefore the time for which evaluation is reported. Further, since Frederickson and associates (11) showed that naloxone-induced hyperalgesia in mice follows a diurnal rhythm, all subjects were given naloxone at 8 a.m.

Results

Six hours after injection there was a statistically significant reduction on the tension item of the BPRS (see table 1). Three of the 6 patients given saline placebo showed a nonsignificant increase on this item.

STUDY 2—NALOXONE, DOUBLE-BLIND

Five of the 7 patients described in study 1 served as subjects in this study. Naloxone and saline placebo were again given, using the same dose, method, and time of evaluation as in study 1.

Interestingly, the findings were more significant than in the single-blind study. The total BPRS score, the thought disturbance factor, and the hallucinatory behavior item were each significantly improved (see table 1).

STUDY 3—PLASMA ACTH LEVELS

The plasma ACTH levels of 5 patients were determined by radioimmunoassay. A relationship between plasma ACTH levels and clinical response was manifest in that the patient who showed no diurnal variation in ACTH level was clinically least responsive to naloxone (patient 1). Conversely, the subject (patient 3) who showed the greatest variability of ACTH levels (diurnal drop) showed the greatest clinical improvement (see table 2).

There has been much discussion of the criteria for selection of patients for naloxone studies. Watson and associates (12) stated that naloxone response is limited to a very small subgroup of continuously and vividly hallucinating schizophrenics. They screened 1,000 patients to obtain a "suitable" sample of 11 subjects. Our own results would indicate that much less rigid criteria can be used. The 5 patients we used were selected from a group of 40, using 3 criteria: hallucinations, thought disorder, and dysphoria.

We do not know what response would occur in the other schizophrenics. Perhaps they too would show improvement; even our more liberal criteria may be too restrictive. In the future, investigators might consider using random selection of schizophrenics or perhaps of all psychiatric patients, to determine what limitations, if any, would apply.

STUDY 4—PROLACTIN RESPONSE TO β-ENDORPHIN AND PLASMA HALF-LIFE OF β-ENDORPHIN

The base population for this study has been described elsewhere (13, 14).

Selected blood samples from our β-endorphin studies (13, 14) were analyzed for prolactin content. Figure 1 shows plasma levels in 1 patient from whom blood samples were collected before infusion and at frequent intervals after an infusion of 6 mg of β-endorphin. The prolactin response apparently peaks at approximately 30 minutes, at which time values close to three times those of baseline are observed. Figure 2 shows the percent of baseline levels of prolactin after injection in the same patient, in a patient given 3 mg of β-endorphin initially and another 3 mg of β-endorphin 165 minutes

FIGURE 1
Prolactin and β-Endorphin Plasma Levels In 1 Patient After Injection of 6 mg of β-Endorphin

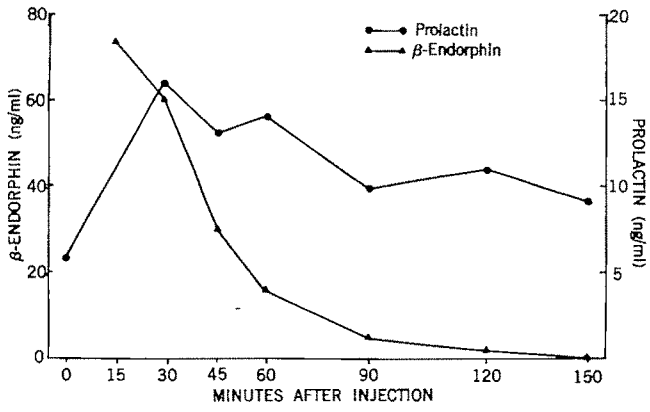
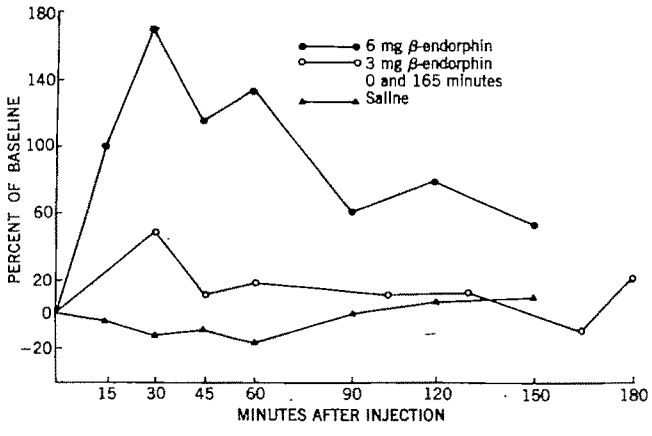


FIGURE 2
Prolactin Plasma Level Response to β-Endorphin and Saline Injection



later, and after saline infusion (control data and 3-mg β-endorphin data were obtained from the same patient).

Blood samples were collected by means of a butterfly in a vein in the antecubital fossae. The samples were collected preinjection and 15, 30, 45, 60, 90, and 120 minutes postinjection. These samples were immediately placed in iced water and kept in the cold until centrifuged (refrigerated centrifuge) and the plasma frozen.

We did 6 subject experiments in which single bolus injections of 3–9 mg of β-endorphin were given. These data were readily fitted with a single exponent. The mean half-life of elimination calculated from these data was 19.6 minutes (range, 12–35 minutes).

Preliminary attempts to measure endogenous levels of β-endorphin in these patients have not proved successful, which indicates that endogenous levels in humans, if present, are in the picogram per milliliter range.

DISCUSSION

We have three-fold evidence suggestive of a role of endorphins in depression: 1) Kline and associates (13, 14), 2) Angst and associates (15), and 3) Terenius and associates (16). All three of these studies point to a deficiency of endorphins in the depressed patient.

The role of endorphins in schizophrenia is much less clear. Indirect evidence that the endorphins are noxious is attested to by the fact that naloxone—in some patients but not all—produces improvement under certain conditions. The three leading hypotheses on the role of endorphins in schizophrenia are 1) that there is an excess, 2) that there is a deficiency, or 3) that there is an aberrant form, possibly a false transmitter. These hypotheses are not mutually exclusive.

Our own findings with intravenous injections of β-endorphin to schizophrenic patients indicate that the therapeutic phase, when it occurred, was delayed one to three days in onset. This time lag is not unlike that of ECT and the phenothiazines and related antipsychotic agents. Compatible with this is the demonstration that β-endorphin is increased in phenothiazine-treated animals only after prolonged administration (17).

The most unique feature of the clinical response of some schizophrenic patients to β-endorphin appeared to be the restructuring of a healthy personality that continued to develop even after endorphin was no longer administered. This went beyond the mere suppression of symptoms and recalled the ancient concept of *vis medicatrix naturae*, set in motion by the endorphin (reminiscent of the German "Stosstherapie").

We believe that it is premature to construct an all-encompassing theory, but at the same time we do feel that theoretical bridges between apparently disparate findings should be encouraged. They may in turn give rise to crucial experiments that will provide hard data to further expand our understanding.

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Am J Psychiatry 136:6, June 1979

Gonadotropin, Estradiol, and Testosterone Profiles in Homosexual Men

BY STEPHEN R. NEWMARK, M.D., LESLIE I. ROSE, M.D., ROBERTA TODD, M.S., LEE BIRK, M.D., AND FREDERICK NAFTOLIN, M.D., D.PHIL.

The authors evaluated the gonadotropin-testosterone-estradiol profiles of four homosexual men and four heterosexual men by a multiple-sampling technique. Although there was extensive overlap between the two groups, the homosexual subjects had higher estradiol levels than the heterosexuals (70.3 ±19.8 versus 56.8 ±10.7 pg/ml) and lower FSH values (5.1 ±1.0 versus 13.2 ±3.4 mIU/ml). Testosterone and luteinizing hormone levels were comparable in the two groups. These observations suggest that there may be subtle differences in gonadotropin and estradiol secretion in homosexual subjects that can be detected only by repeated sampling.

RECENTLY there has been renewed interest in the relationship of the endocrine system to homosexuality, and results have been conflicting in some cases (1–5). Because pituitary gonadotropins (6), testosterone (7), and estradiol (8) are secreted in a pulsatile manner, a single blood sample may not accurately reflect hypothalamic-pituitary-testicular activity—the levels may vary several-fold over any sampling period. In the present study, a multiple-sampling technique over several hours was used to determine whether the gonadotropin-testosterone-estradiol profile of homosexual subjects differed from that of heterosexual male control subjects.

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METHOD

The four male homosexual subjects were psychotherapy patients ranging in age from 22 to 30 years. Each patient was assigned a Kinsey number (9) based on his own assessment of his homosexual/heterosexual feelings plus a therapist's evaluation after several hours of interviewing. Subjects' and therapists' ratings were identical (all Kinsey number 6, or exclusively homosexual) in all four cases.

The four control subjects, aged 21–29, were considered to be heterosexual on the basis of a history taken by two interviewers. All subjects received a physical examination and detailed histories were elicited. Laboratory examination included X-rays of the chest and sella turcica, ECG, CBC, urinalysis, electrolytes, liver function tests, thyroxine, fasting blood glucose, and creatinine. All subjects denied a recent use of medications or drugs, including marijuana.

Blood samples were taken from an antecubital vein via an indwelling catheter every 15 minutes for 4–8 hours. Serum was frozen at −20°C until hormone assay. Subjects reclined during the study.

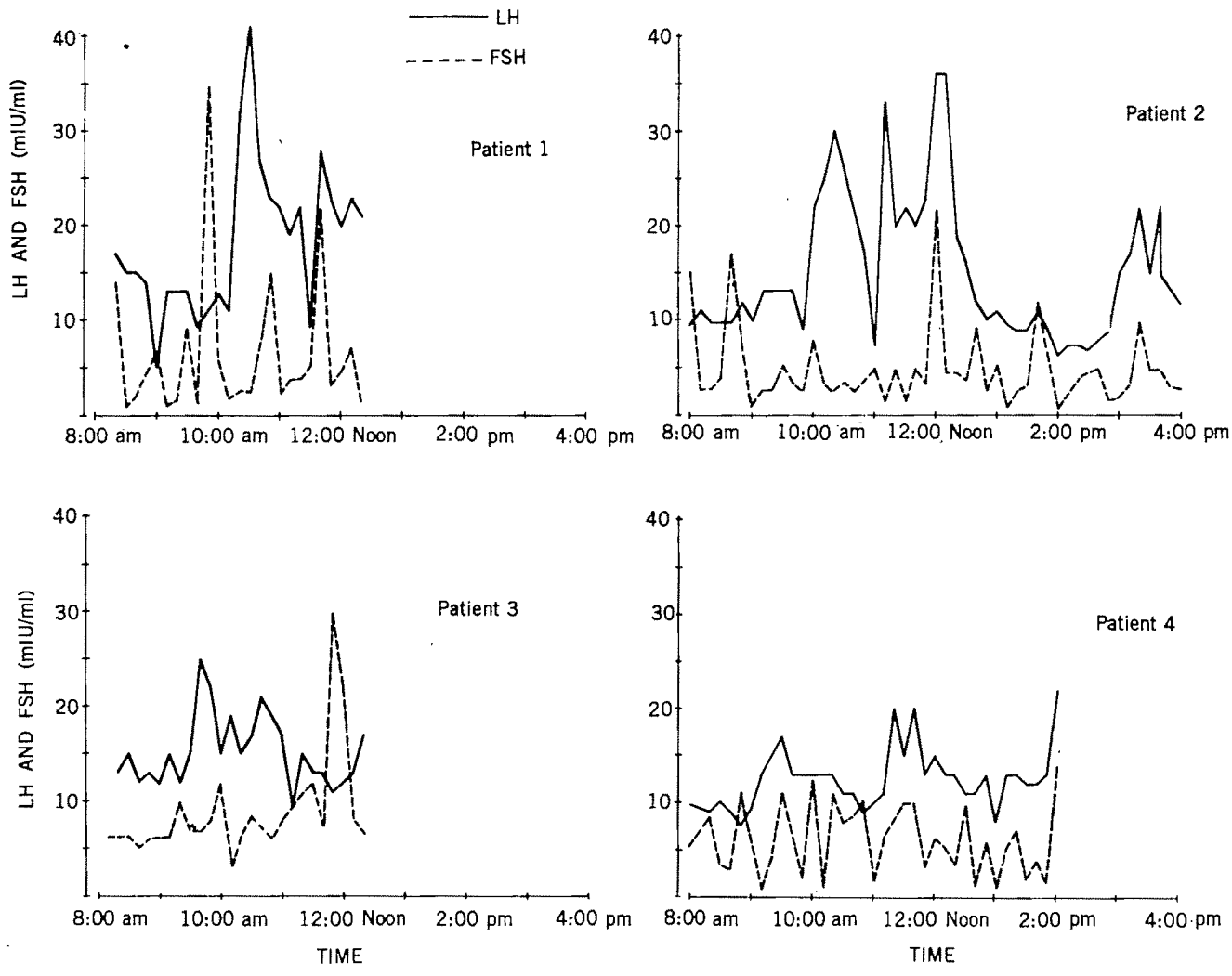
Serum gonadotropins (10, 11) were estimated by specific radioimmunoassay using the Second International Reference Preparation as a standard. Testosterone was measured by the method of Niswender and associates (12). Estradiol was determined by the method of Korenman and associates (13). Progesterone, androstenedione, estrone, and dihydrotestosterone were determined by the method of Challis and associates (14).

The standard errors of the assays (intraassay variations) were as follows: follicle-stimulating hormone (FSH), 2%; luteinizing hormone (LH), 2%; testosterone, 8%; estradiol, 5%; progesterone, 7%; androstenedione, 10%; estrone, 10%; and dihydrotestosterone, 10%. To eliminate interassay variation, all samples were assayed simultaneously in duplicate for each hormone being measured.

Semen analysis was determined by standard methodology (15). Free estradiol was determined by the method of Tulchinsky and Chopra (16). Two serum samples were tested from each patient. Data were analyzed by Student's t test (two-tailed).

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FIGURE 1
Serum FSH and LH Levels Obtained Every 10 Minutes in Homosexual Subjects



RESULTS

The FSH and LH values were examined by determining means for all samples. To minimize the effect of the variation in FSH and LH levels that results from pulsatile secretion, a log transformation was performed on each group and the means of the log transformation of FSH and LH values per 4 hours were also analyzed (17).

Gonadotropin pulses in serum were determined for each individual and were considered to reflect secretion (18). A pulse of FSH or LH was defined as an increase of 5 mIU or greater that was present for at least two consecutive determinations. The termination pulse was defined as a decrease of 5 mIU in at least two consecutive determinations. Both groups had multiple pulsatile secretion of gonadotropins (figures 1 and 2). Both groups had 0.8 FSH pulses/hour, and the heterosexual group had 0.8 LH pulses/hour as opposed to 0.6 pulses/hour for the homosexual group, a non-significant difference ($.05<p<.10$). The LH values for the heterosexual group and the homosexual group were not statistically different (see table 1). However, FSH values were significantly lower in the homosexual group.

Serum testosterone and estradiol levels were obtained hourly (figures 3 and 4). Although serum testosterone levels were similar in both groups, serum estradiol levels were significantly higher in the homosexual group (see table 2). The values for testosterone and estradiol are higher than the usual reported levels for normal subjects, but they have been consistent in our laboratory.

Single random samples of estrone, progesterone, dihydrotestosterone, and androstenedione levels were similar in both groups, although the mean progesterone level of the homosexuals was almost twice that of the heterosexuals (table 2). The percentage of total estradiol as free estradiol was significantly lower in the homosexual group ($p<.05$), although the calcu-

FIGURE 2
Serum FSH and LH Levels Obtained Every 10 Minutes in Heterosexual Subjects

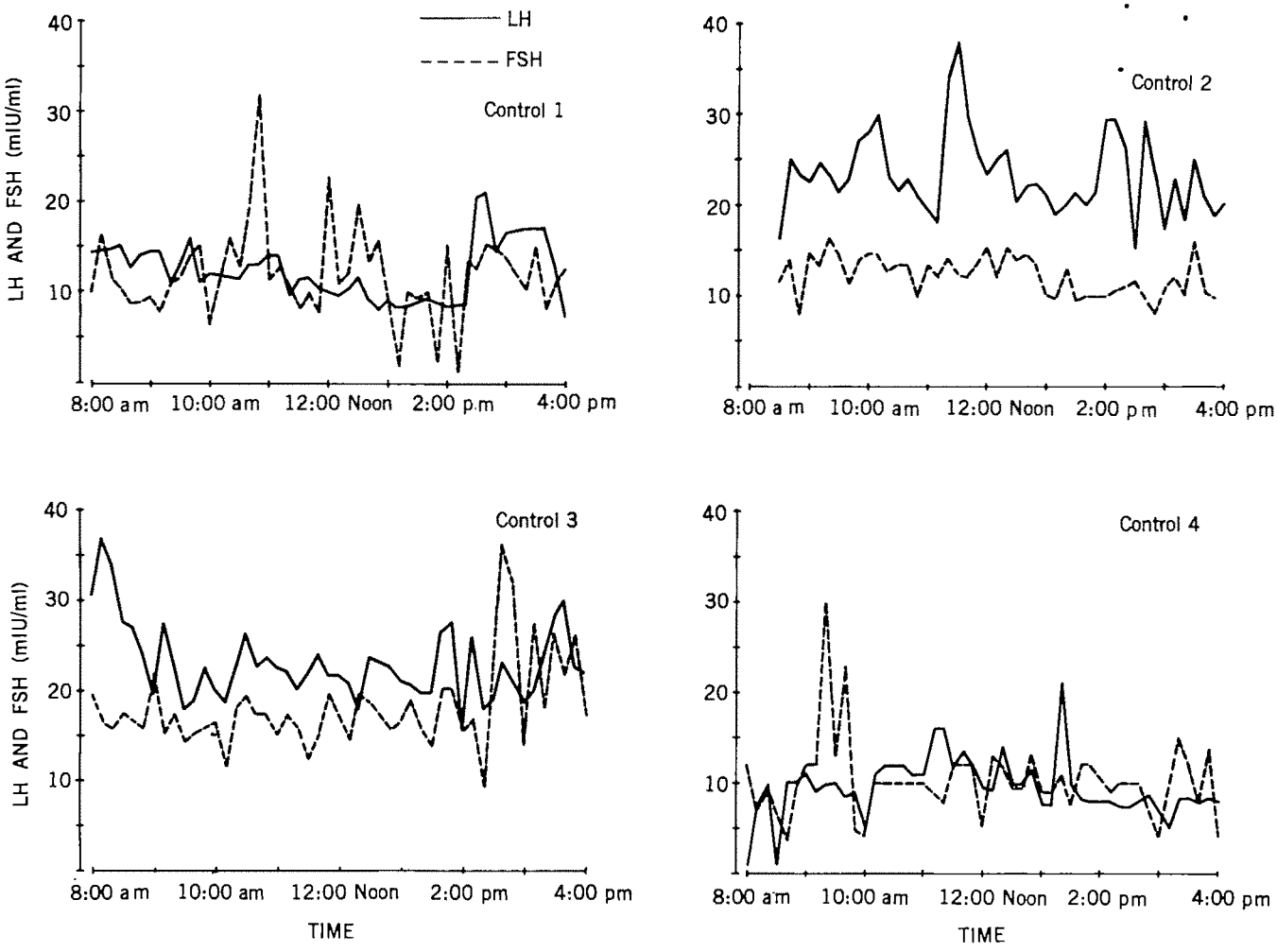


FIGURE 3
Serum Testosterone Levels in Both Homosexual and Heterosexual Subjects

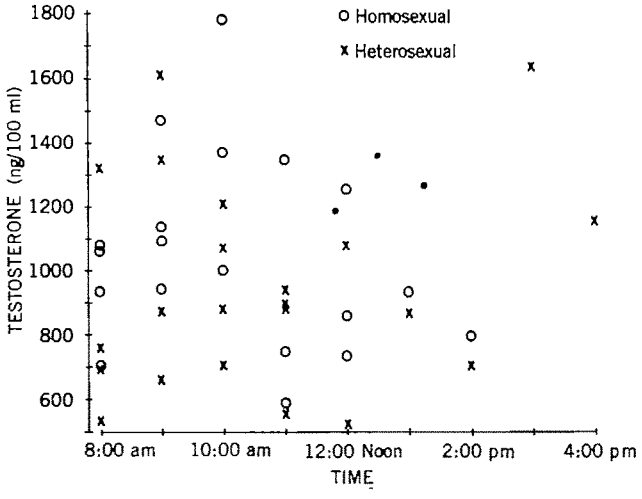


FIGURE 4
Serum Estradiol Levels in Both Homosexual and Heterosexual Subjects

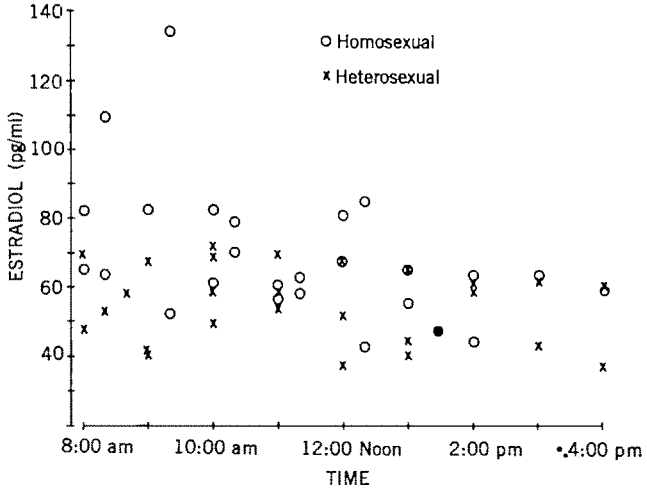


TABLE 1
LH and FSH Levels in 4 Homosexual and 4 Heterosexual Men

Item	Levels (mIU/ml) Mean	SD	p
Luteinizing hormone			
Mean of all samples			
Homosexual	15.7	5.1	n.s.
Heterosexual	17.1	4.2	
Log transformed mean			
Homosexual	1.1	0.1	n.s.
Heterosexual	1.2	0.2	
Follicle-stimulating hormone			
Mean of all samples			
Homosexual	5.1	1.0	<.005
Heterosexual	13.2	3.4	
Log transformed mean			
Homosexual	0.7	0.1	<.005
Heterosexual	1.1	0.1	

TABLE 2
Steroid Hormone Levels in 4 Homosexual and 4 Heterosexual Men

Assay[a]	Homosexual Mean	SD	Heterosexual Mean	SD	p
Testosterone (ng/100 ml)	986.2	273	882	102.6	n.s.
Estradiol (pg/ml)	70.3	19.8	56.8	10.7	<.025
Unbound estradiol (pg/ml)	2.4	0.1	2.1	0.1	n.s
Unbound estradiol (%)	3.3	0.3	3.8	0.4	<.05
Androstenedione (ng/100 ml)	118.9	48.5	100.6	31.2	n.s.
Progesterone (ng/100 ml)	93.0	39.2	58.9	18.6	n.s.
Estrone (pg/ml)	130.4	19.3	125.4	38.2	n.s.
Dihydrotestosterone (ng/100 ml)	46.4	12.9	44.1	6.2	n.s.

[a]Testosterone and estradiol values are means for all samples; androstenedione, progesterone, estrone, and dihydrotestosterone are group means, with 1 sample per subject.

lated mean free estradiol was the same for both groups.

Sperm count and morphology were similar in both groups and were all above $40 \times 10^6/mm^3$, with normal morphology and motility at 2 hours.

DISCUSSION

In recent years there have been extensive reports on the relationship of the endocrine system to male homosexuality. Kolodny and associates (1) reported lower plasma testosterone values, impaired spermatogenesis, and higher FSH and LH levels in a group of 30 homosexual patients compared to a matched group of heterosexual individuals. The elevated LH values were correlated with decreased plasma testosterone values, and the elevated FSH values were seen in men with oligo or azospermia. These abnormalities were most pronounced in those homosexuals who had higher homosexual scale values on the Kinsey rating system. Others (2–5) have demonstrated normal values for plasma testosterone and suggested that the differing endocrine profiles probably are secondary to several factors, among which are sample size, differing etiologies of homosexuality, and effects of therapy. Doerr and associates (4) reported increased plasma estradiol levels in homosexual patients without concurrently decreased plasma testosterone levels or sperm counts. All of the previous studies were based on a single sample from the subject population.

Since values for serum gonadotropins, estradiol, and testosterone vary dramatically, as shown by frequent sampling, the FSH-LH-sex steroid profile is more accurately determined by a multiple-sampling technique.

It can be seen readily (figures 1 and 2) that both subject groups had multiple secretory episodes of FSH and LH, as determined by multiple sampling. Although the LH means were identical in both subject groups, the FSH means were substantially lower in the homosexual group. This would imply that although FSH is being secreted in pulsatile form, the total amount secreted is less than in the heterosexual group; nevertheless, there is considerable overlap of the individual values in both groups. It is not apparent why FSH secretion is low in the homosexual group. One possibility is that the elevated serum estradiol in the homosexual group selectively impairs the pituitary secretion of FSH in response to endogenous hypothalamic releasing hormones; selective estrogen-induced inhibition of FSH secretion has been demonstrated elsewhere (15). Other possibilities include altered metabolic clearance of FSH, altered metabolism, or increased "inhibin" secretion (15).

Serum testosterone values were similar in individuals from both study groups, as were single samples assayed for dihydrotestosterone, androstenedione, progesterone, and estrone. Serum testosterone values displayed variations each hour (figure 3) that were presumably secondary to intermittent secretion by the testes or to altered metabolic clearance rates. The lack of correlation with FSH or LH levels or pulses could be due to the difference in the timing of the sample measurements.

The total mean serum estradiol value was increased in the homosexual group, although individual values in both subject groups overlapped (figure 4). This could reflect increased testicular secretion, increased conversion of androgen precursors, increased sex hormone binding globulins, or decreased metabolic clearance. None of these has been studied in homosexual males.

The percentage of free or unbound estradiol was significantly lower in the homosexual group. This presumably reflects higher endogenous binding proteins and may explain why the serum estradiol values are increased in the homosexual group. However, it is also possible that the increase in binding proteins is secondary to increased estradiol secretion. There is cur-

rently no way to distinguish between these two alternatives. Total unbound estrogen levels are comparable in the two groups. As unbound estradiol is presumably the active component of the steroid, it is possible that the elevated total estradiol level in the homosexual subjects does not have a biologic effect on them.

It is important to note that all four of the homosexual subjects were receiving psychotherapy, whereas the four control subjects were not in psychotherapy. The relationship between psychotherapy, mental illness, and endocrine status remains to be clearly defined. It is therefore possible that some of the endocrine changes demonstrated might be related to psychotherapy or affective state rather than homosexuality. Further studies are necessary to clarify these relationships.

At present, one cannot conclude that there is a basic or causative alteration in endocrine control systems in the male homosexual. Nevertheless, evidence is beginning to accumulate that, at least in some homosexual individuals, there are significant differences in the levels of gonadotropins and either estradiol or testosterone; however, because of individual variation or pulsatile secretion, a single blood specimen may not reflect an endocrine abnormality.

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Choline and Lecithin in the Treatment of Tardive Dyskinesia: Preliminary Results from a Pilot Study

BY ALAN J. GELENBERG, M.D., JOANNE C. DOLLER-WOJCIK, R.N., M.S., AND JOHN H. GROWDON, M.D.

Tardive dyskinesia is thought to reflect increased dopaminergic activity of the central nervous system. To compensate for this by increasing CNS cholinergic tone, the authors administered oral choline and its natural dietary source, lecithin, to 5 men with mild to severe tardive dyskinesia in a nonblind trial. Both choline and lecithin increased serum choline levels and improved abnormal movements in all patients. Lecithin had fewer adverse effects.

TARDIVE DYSKINESIA is a disorder of abnormal involuntary movements associated with the ingestion of neuroleptic drugs (1). A popular hypothesis is that tardive dyskinesia involves an imbalance in the postulated reciprocal relationship between dopaminergic and cholinergic neurons in the basal ganglia (2–4). This imbalance appears to favor dopamine transmission at the expense of acetylcholine transmission—the biochemical obverse of Parkinson's syndrome. The relative dopamine excess may result from increased dopamine turnover (5) or from a denervation type of supersensitivity (6).

Consistent with this hypothesis of the neurophysiology of tardive dyskinesia are observations that drugs which block catecholamine synthesis (alpha-methylparatyrosine) (3, 7), deplete the brain of monoamines (reserpine, tetrabenazine) (8), or antagonize dopamine's action on synaptic receptors (phenothiazines, haloperidol) (9) often suppress tardive dyskinesia, whereas drugs that indirectly stimulate dopamine receptors (amphetamine, L-dopa) (10, 11) can increase the abnormal movements. In attempts to increase central nervous system (CNS) cholinergic tone, deanol, a putative choline precursor, has been administered to patients with tardive dyskinesia, but the results have been variable (11–15). In a similar vein, physostigmine, a centrally active anticholinesterase drug, has been administered intravenously to patients with tardive dyskinesia, with resultant decreases in choreic movements (16, 17). A third strategy for increasing CNS cholinergic tone is the administration of choline chloride and the natural dietary source of choline, lecithin. In this paper we will report the preliminary results of a nonblind pilot project in which choline and lecithin were administered to outpatients with mild to severe tardive dyskinesia.

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METHOD

Patients

Ten patients have entered the study to date. In this paper we will present data on 5 of them. Three other patients are still in early phases of treatment, and data are not yet available. The remaining 2 patients were dropped from the study, 1 because of poor compliance and the other because further neurological testing revealed basal ganglia disease believed to represent a process other than tardive dyskinesia.

The 5 patients to be discussed here were treated with both choline and lecithin. All 5 were men, ranged in age from 27 to 32, and were outpatients. Two were given a diagnosis of schizophrenia; 1, severe obsessive-compulsive neurosis; 1, chronic anxiety; and 1, chronic depression.

Stable signs of tardive dyskinesia had been present for 1–5 years, onset was well documented in all cases, and global severity of movements was rated from mild to severe and agreed to by both the project psychiatrist (A.J.G.) and the project neurologist (J.H.G.). The diagnosis of tardive dyskinesia was based on the following criteria.

1. Abnormal involuntary movements of a choreoathetotic, dyskinetic variety were present in the mouth, face, and/or extremities.
2. The movements occurred in patients who had been treated with an antipsychotic drug for at least 6 months.
3. Abnormal movements appeared either while the patient was receiving an antipsychotic drug or within 4 weeks after discontinuation of such medication.

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Attempts were made to rule out naturally occurring forms of extrapyramidal disorders, other forms of drug-induced extrapyramidal disorders, and schizophrenic mannerisms and stereotypies.

Procedure

After giving written informed consent, patients were started on choline chloride, 150 mg/kg per day, in three divided doses. Dosages were then adjusted based on clinical response and adverse reactions, to a maximum of 200 mg/kg per day. Once a stable dose was reached, an attempt was made to maintain patients on choline for 6–8 weeks. A 2-week washout period was then planned, but for practical reasons it varied from 18 days to 5 weeks. After washout, patients were begun on lecithin, with an initial daily dose for all patients of 21 g in three divided doses. The dose of lecithin was increased at weekly intervals to as high as 105 g/day. Choline was supplied as a powder, which patients mixed in sweet beverages such as fruit drink or soda pop; lecithin was supplied in the form of granules that contained 20% phosphatidylcholine and was mixed with or sprinkled on apple sauce, yogurt, or ice cream.

Dyskinetic movements were rated weekly on the NIMH Abnormal Involuntary Movement Scale (AIMS) (18), frequency of movements was counted over 30 seconds, and patients were videotaped. Parkinsonian signs were also rated at weekly intervals using the Targeting Abnormal Kinetic Effects (TAKE) Scale (a scale we have devised as a comparison piece to the AIMS) and Simpson-Angus Scale (19), psychiatric state was evaluated with the Brief Psychiatric Rating Scale (20) and Clinical Global Index (21), and side effects and patients' subjective global assessments were also recorded. Laboratory testing included electrocardiograms, complete blood count, urinalysis, and serum enzyme and lipid profiles. Serum choline levels were assayed before treatment, during choline therapy, and again during lecithin therapy using a radioenzymatic method (22).

Patients were allowed to continue to take other medication (except anticholinergic agents), and these were maintained at relatively constant doses.

RESULTS

Table 1 summarizes patient responses. The AIMS scores and percentage changes are based on average weekly ratings of body parts that showed at least mild abnormal movements at baseline assessment. The AIMS assessments, performed by a trained nurse-rater (J.D.W.), were reliably correlated with neurologist-rated (J.H.G.) movement-counts; this will be discussed further in a future communication.

As the table indicates, all of the patients improved on both treatments, with a tendency toward greater improvement on lecithin. During the washout period the patients' movements began to worsen within several days, and most had returned to baseline levels between 1 and 2 weeks.

Daily doses of choline chloride ranged from 12 to 17 g, and maintenance doses from 12 to 14 g. Two subjects (patients 4 and 5) discontinued lecithin prematurely for reasons listed below. Patient 5 received lecithin for only several days, so his lecithin response is not indicated in table 1. Patient 4 received lecithin for 8 weeks, but both his daily lecithin dose and serum choline increase were considered suboptimal (see table 2). The remaining 3 patients have continued to receive 105 g of lecithin daily for up to 6 months without evidence of tolerance to the therapeutic benefit.

Four of the 5 patients continued to receive phenothiazine therapy throughout the study. Patient 1 had ingested no antipsychotic medication for 4 months before the study. Patient 2 had received fluphenazine hydrochloride, 10 mg per day, for 6 months before the study. During the last month of the study his fluphenazine was decreased to 5 mg daily and his overall AIMS scores worsened somewhat, with an increase in the average lecithin AIMS score and a decrease in the average percentage improvement on lecithin therapy. Patient 3 had received fluphenazine hydrochloride, 40 mg daily, for 3 months before the study, and this schedule was maintained throughout the study period. Patient 4 was taking fluphenazine, 10 mg daily, for 1 month before the study, and this was discontinued 1 week before the end of the lecithin trial; the discontinuation did not appear to affect his AIMS scores, however. Patient 5 had been receiving a stable dose of trifluoperazine, 40 mg daily, for $2\frac{1}{2}$ months before and during the study. During his last 3 weeks of choline therapy trifluoperazine was reduced to 30 mg a day; the change in dosage was accompanied by transient increases in abnormal movements.

The patients' serum choline levels are presented in table 2. The table illustrates that both choline and lecithin are capable of producing considerable increases in serum choline levels.

All 5 patients found the bitter taste of choline unpleasant. Furthermore, all developed a distinctive and unpleasant "fishy" body odor after several days on the higher doses of choline. Each patient also developed symptoms of gastrointestinal irritation on choline, and this was often a dose-limiting factor. There was a tendency toward the appearance of or an increase in parkinsonian signs (such as bradykinesia, tremor, and rigidity) during both treatments, but this side effect was mild and transient. The only adverse reaction specifically attributed to lecithin was a 7% increase in body weight in 1 patient. Lecithin did not produce a body odor, and the patients found its addition made food more palatable, rather than less so. No changes in laboratory test values occurred in any patient during the study.

There were no consistent changes in patients' mental status. Two subjects (patients 4 and 5) discontinued lecithin because of increasing paranoia, but the paranoia was not improved in either patient several weeks

TABLE 1
Abnormal Involuntary Movement Scale (AIMS) Scores of 5 Men with Tardive Dyskinesia

Item	AIMS Area						
	Lips and Perioral	Facial Expression	Jaw	Tongue	Upper Extremity	Lower Extremity	Trunk
Patient 1							
Prescore	—	2.00	2.00	2.00	4.00	4.00	4.00
Choline	—	1.45	0.18	1.18	2.27	2.00	3.09
Percent better		28	91	41	43	50	23
Washout	—	2.00	0.00	1.50	2.50	3.00	4.00
Lecithin	—	1.75	0.00	2.25	3.00	2.00	3.50
Percent better		13	0			33	13
Percent worse				50	20		
Patient 2							
Prescore	—	—	3.00	2.00	2.00	2.00	—
Choline	—	—	2.30	2.23	1.30	1.76	—
Percent better			23		35	12	
Percent worse				12			
Washout	—	—	1.50	2.00	1.00	1.50	—
Lecithin	—	—	0.90	0.81	0.45	0.27	—
Percent better			40	60	55	82	
Patient 3							
Prescore	—	3.00	—	—	2.00	—	—
Choline	—	2.57	—	—	2.71	—	—
Percent better		14					
Percent worse					36		
Washout	—	3.00	—	—	3.00	—	—
Lecithin	—	2.43	—	—	1.75	—	—
Percent better		19			42		
Patient 4							
Prescore	2.00	—	—	—	2.00	3.00	—
Choline	1.25	—	—	—	1.75	2.33	—
Percent better	38				13	22	
Washout	2.00	—	—	—	2.00	2.00	—
Lecithin	1.17	—	—	—	1.50	1.00	—
Percent better	42				25	50	
Patient 5							
Prescore	—	3.00	—	—	—	—	—
Choline	—	2.60	—	—	—	—	—
Percent better		13					
Washout	—	3.00	—	—	—	—	—

TABLE 2
Serum Choline Levels of 5 Men with Tardive Dyskinesia

Item	Serum Choline Level (nmol/ml)
Patient 1	
Pretreatment	15.2
Choline (17 g/day)	48.4
Lecithin (105 g/day)	43.2
Patient 2	
Pretreatment	12.2
Choline (19 g/day)	22.0
Lecithin (77 g/day)	24.0
Patient 3	
Pretreatment	7.1
Choline (19 g/day)	36.3
Lecithin (105 g/day)	21.7
Patient 4	
Pretreatment	10.5
Choline (8 g/day)	17.1
Lecithin (21 g/day)	13.3
Patient 5	
Pretreatment	12.0
Choline (8 g/day)	27.3

after discontinuation of the drugs. Two patients were noted to be slightly more depressed during the study, but 2 other patients showed a marked improvement in preexisting depression.

DISCUSSION

Choline is the physiological precursor of acetylcholine (23), which is synthesized by combining molecules of choline and acetylcoenzyme A in a reaction catalyzed by the enzyme choline acetyltransferase. Choline-induced increases in acetylcholine levels in the brain appear to reflect increased synthesis (24) and also cause increases in acetylcholine release onto postsynaptic sites (25). The brain is unable to make choline de novo and extracts it instead from the systemic circulation by a low-affinity uptake process that is normally unsaturated (25). The enzyme choline acetyltransferase, which converts choline to acetylcholine,

is also unsaturated with the circulating precursor in vivo, and increasing the amount of substance increases the net catalytic activity of the enzyme. Thus Cohen and Wurtman showed that choline, by injection (26) or dietary supplementation (24), increased blood choline, brain choline, and brain acetylcholine levels in the rat, and these findings were confirmed independently by Haubrich and associates (27). Choline administration also caused parallel increases in acetylcholine release, as judged by biochemical changes in cells that are postsynaptic to the cholinergic neuron. Thus choline administration increased tyrosine hydroxylase (the rate-limiting enzyme in cathecholamine synthesis) in both the adrenal medulla (28) and the caudate nucleus (29); prior atropine administration or denervation of the splanchnic nerve to the adrenal gland blocked this effect.

In clinical studies Davis and associates (23) described a man in whom the administration of choline (16 g per day) decreased the choreiform movements of tardive dyskinesia. This group and others (30, 31) have since reported additional evidence that choline can diminish the movements of tardive dyskinesia. Growdon and associates (32) reported that in a double-blind crossover study involving 20 patients with tardive dyskinesia, blood choline levels in all patients increased during choline ingestion, and 9 patients were significantly improved.

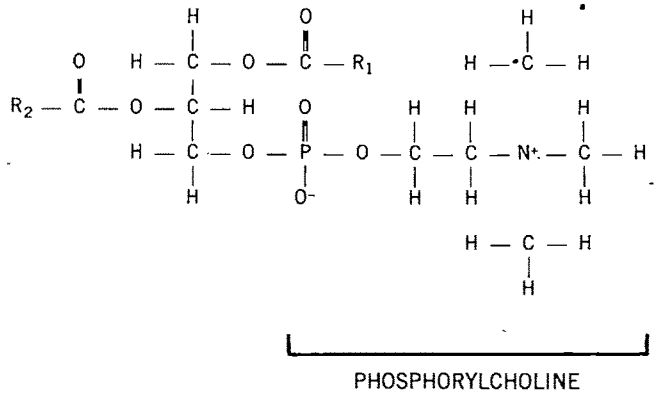
Choline in blood derives normally from two sources: dietary consumption (primarily as lecithin in egg yolk, soy beans, fish, meat, and many other foods) (33) and from synthesis in liver (34). Lecithin, the usual dietary source of choline, consists of choline covalently bound to glycerol and fatty acids (see figure 1).

The therapeutic use of choline presumably derives from the ability of orally administered choline to markedly elevate plasma levels in humans (35). Studies in normal humans indicate that the administration of large oral quantities of lecithin causes an even greater and longer-lasting increase in serum choline levels (36). In rats, oral lecithin has also been shown to increase brain choline and acetylcholine levels (37).

The data from this nonblind, uncontrolled study support earlier findings that choline chloride can alleviate some of the signs of tardine dyskinesia. This study extends these findings to a less severe, younger, and less chronic population. Furthermore, lecithin therapy appears to be at least as effective as choline, with fewer adverse reactions and greater patient acceptance; these findings were recently confirmed by Barbeau (38). Moreover, some of our patients have received lecithin at doses greater than 100 g daily for over 4 months with no evidence of ill effects.

Thus far our data can neither confirm nor refute reports that choline (and presumably lecithin, also) can cause clinical depression (31). If the cholinergic-adrenergic hypothesis of mood regulation is accurate (39), an increase in CNS cholinergic tone might certainly be expected to produce or worsen depressive symptoms. Yet if data from reserpine therapy can serve as an analogy, perhaps choline can be expected to produce depression in, say, 10% of the population with a depressive diathesis (40). This question remains open at the present time.

FIGURE 1
Structure of Lecithin



The worsening of parkinsonian signs that we observed is certainly consistent with the well-accepted dopamine-acetylcholine balance theory (41).

Since lecithin is able to achieve increases in blood choline levels, and presumably brain acetylcholine levels as well, and because our data suggest that lecithin administration can produce clinical improvement with fewer adverse reactions, we plan to discontinue further clinical use of choline chloride. We also plan to use a lecithin preparation with a higher concentration of phosphatidyl choline, thereby lowering the amount of extraneous lipids ingested. In the near future we expect to withdraw our patients from lecithin therapy and treat the responders in a double-blind, placebo-controlled, crossover design to further assess the positive responses. Future research will involve prospective, double-blind trials of large numbers of patients with tardive dyskinesia.

If the results of this pilot study are confirmed, several benefits will be realized. Headway will have been made toward the realization of therapy for patients with tardive dyskinesia, a troublesome and common long-term, irreversible reaction to antipsychotic drugs. In addition, we will have achieved further understanding of the mechanism of tardive dyskinesia in particular and basal ganglia disease in general. Finally, a dietary constituent will have been shown to affect the levels of a CNS neurotransmitter, opening possibilities for a greater understanding of normal neurophysiology and for future therapies.

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Hysterical Psychosis and Hypnotizability

BY DAVID SPIEGEL, M.D., AND ROBERT FINK, M.D.

The very existence of hysterical psychosis as a diagnostic entity has been questioned as part of the general difficulty in defining both hysteria and psychosis. However, several recent investigations have documented a syndrome that usually involves brief and intense periods of psychotic behavior, generally with graphic decompensation, severe environmental stress, and rapid recompensation, in individuals with other hysterical features. The authors assert that such a syndrome does exist as a clinical entity and that the differential diagnosis can be facilitated by using a standardized measure of hypnotic trance capacity. They hypothesize that patients with hysterical psychosis are highly hypnotizable, while those who are schizophrenic and psychotic have low hypnotizability. The authors review the literature and present two case examples.

FOR A FLORIDLY psychotic patient with delusions, loose associations, ideas of reference, and inappropriate affect, the standard differential diagnosis must include schizophrenia, an affective disorder of psychotic proportions, and various toxic and organic disease states. Another clinical entity that deserves consideration in this differential diagnosis is hysterical psychosis. Such individuals often experience rapid and florid psychotic decompensation, including delusions, ideas of reference, loose associations, and affect that can range from bland indifference which approaches flatness to intense agitation. They may or may not have a family history of thought disorder and often mobilize tremendous attention and anxiety in their social network. They frequently have a history of good premorbid adjustment and may not respond to neuroleptic medication (i.e., they are sedated but do not improve). They can be distinguished from schizophrenic patients who are experiencing a psychotic episode by psychological testing and careful evaluation of history and affect. The intact capacity for repression implied by *la belle indifference* in hysteria contrasts with the flatness traditionally associated with schizophrenia. Further, a standardized assessment of hypnotizability, the capacity to enter a hypnotic trance state, is a useful device for confirming the differential diagnosis of schizophrenia versus hysterical psychosis. According to this criterion, individuals with hysterical psychosis are generally highly hypnotizable, except perhaps during periods of intense agitation, while those with schizophrenia show little or no hypnotizability.

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LITERATURE REVIEW

Charcot is famous for (among many other things) linking hypnosis and hysteria. He recognized that hysteria had variable manifestations and that hypnotic phenomena could occur in seemingly normal individuals, but he still viewed hypnosis as a kind of artificially induced pathology (his terms included "sommeil nerveux," or nervous sleep; and "un état nerveux artificiel ou expérimental," an artificial or experimental nervous state) which could be used to demonstrate symptoms of hysteria (1). Janet (2) went even further in identifying the two as one phenomenon: "The hypnotic state has never any character which cannot be found in natural hysteric somnambulisms." He added that patients who recovered from hysteria lost their hypnotic capacity, which is unlikely according to current research, but he noted that "subjects troubled with other diseases than hysteria—epileptics, for instance, psychasthenics tormented by the mania of doubt, lunatics affected with systematic delirium—are not at all hypnotizable. . . ," which is an observation quite consistent with our hypothesis.

Breuer and Freud began their classical explorations of the unconscious via hysteria, which they defined in terms of ego disintegration and eruption of the unconscious. In their early explorations they employed hypnosis, largely for purposes of abreaction. Freud later decided to abandon the hypnosis because he thought it distracted from analysis of the transference, which left us with many unanswered questions. Breuer wrote an interesting footnote in his theoretic discussion of hysteria:

> The miraculous workings of hypnosis are, so far as I can see, only observable in hysterical patients. What we should be doing would be first to assign the phenomena of hysteria to hypnosis, and then to assert that hypnosis is the cause of those phenomena." (3, p. 248)

We would agree with this observation and argue that many hysterical signs and symptoms may be related to these patients' profound capacity to be hypnotized, although we would not agree that hypnosis is confined to hysteria or that high hypnotizability implies psychopathology.

Subsequent analytic writers have pointed out more serious character pathology underlying what Freud described as hysterical neurosis (4, 5). In reviewing the psychoanalytic literature, Chodoff (6) noted the importance of situational factors, particularly environmental stress, in eliciting hysterical features. He commented that what commonly passes for hysteria looks more like a male caricature of female traits and defined hysteria as a language of action, emphasizing the interpersonal communicative aspects of hysterical symptoms.

From a statistical diagnostic perspective, some have defined the syndrome in terms of multiple somatic complaints (7), while others have pointed to the relative instability of the diagnosis of hysteria (8). Slater (9) has gone so far as to attack the concept of hysteria as "a delusion and a snare."

The phenomenon of hysterical psychosis per se has been given some attention, largely in the work of Hollender and Hirsch (10, 11). They described a psychosis that may include hallucinations, delusions, depersonalization, and usually volatile affect. They noted that such a psychosis may occur in individuals with characteristics consistent with the diagnosis of hysterical character. These authors emphasized, in particular, extreme sensitivity to cues from other people, emotional volatility, the need to behave in accordance with the expectations of others, a tendency to live for the moment, and an orientation toward "affective truth" (10). In a later paper (11) they clarified this concept by subdividing the syndrome of hysterical psychosis into three different types: 1) "culturally sanctioned behavior," which involves data from anthropologists regarding the acts of madness that perform certain social functions in non-Western societies (12, 13); 2) "the appropriation of psychotic behavior," in which a suggestible individual acts out his or her identification with an important person in his or her life who is psychotic; and 3) "true psychosis," with eruption of unconscious material and modification of ego function relating to the evaluation of reality.

Among others who have written on hysterical psychosis, Siomopoulos (14) described it as a pattern of regressive behavior related to childlike thought activities and the merging of fantasy with reality, but with the patient always conveying some awareness that the delusional material is not entirely believed or taken seriously. In a particularly interesting paper, Mallet and Gold (15) discussed a "pseudo-schizophrenic hysterical syndrome" in women with a history of superficial object relations, multiple somatic complaints, and sexual dysfunction along with a depression characterized by complaints of loneliness and emptiness, dramatic affect, and visual hallucinations. All of these women were eventually diagnosed as hysterical but also received a schizophrenic diagnosis at one time or another during their treatment. Of particular interest is the authors' observation that these patients had great difficulty expressing their plight in words, which is perhaps a factor in their preference for dramatizing their situation in action. This use of action as a language is quite consonant with other descriptions of hysteria (6, 16).

The usefulness of assessing hypnotizability in differential diagnosis was suggested by Spiegel (16) in a clinical report on a number of highly hypnotizable patients who demonstrated in common an intense capacity for affiliation, a proneness to relinquish control in an interpersonal setting, a tendency to live in the present rather than in the past or future, and to rely on feelings rather than thinking in making decisions, a capacity for dramatic affective states, a tendency toward compulsive compliance to the demands of others and, consistent with this, an inner core of self-doubt and sense of worthlessness.

The usefulness of hypnotizability assessments in differential diagnosis has been corroborated by Frankel and Orne (17), who found that phobic patients were more hypnotizable than cigarette smokers on the Stanford Hypnotic Susceptibility Scale (18). Spiegel and his coworkers have demonstrated a statistical relationship between certain patterns of performance on the Hypnotic Induction Profile (19, 20) associated with little or no hypnotizability and the existence of serious psychopathology, including schizophrenia (21). A response on this test consistent with high hypnotizability has been reported to be associated with a vulnerability to neurotic disorders, especially hysteria, among the highly hypnotizable (22). Frankel (23) interpreted the symptoms of two phobic patients with relatively high scores on the Hypnotic Induction Profile as spontaneous trance phenomena.

While some workers maintain that schizophrenics are at least as hypnotizable as the normal population (24), the trend of research in this area within the past 15 years has indicated generally lower hypnotizability and the absence of very high hypnotizability among chronic schizophrenic patients (25–28). These findings are consistent with our clinical experience with the Hypnotic Induction Profile. We have observed that as a group schizophrenic and seriously depressed patients frequently show "decrement" patterns on the test, indicating little or no hypnotizability, while healthier individuals, usually outpatients, obtain higher "intact" scores. Disorders of thought or mood seem to interfere seriously with the kind of concentration measured by this test of hypnotizability. Thus, it seems plausible that measurement of the capacity for

attentive, intense focal concentration with diminished peripheral awareness that is characteristic of the hypnotic state may prove useful in discriminating between a psychosis of hysterical origin and schizophrenia or other more malignant psychiatric disorders.

The literature we have cited on hysterical psychosis has described a particular proneness in these individuals to comply with direct or implied environmental cues. In particular, some hysterical psychoses may be attributable to identification with a psychotic person close to the subject. This proneness to identification is a recognizable part of the syndrome of high hypnotizability (16). There have been several suggestions, notably the contribution of Laing (29), that psychoses in general and schizophrenia in particular are means of communicating with the environment and that so-called psychotic symptoms are really not so insane when understood in terms of the suffering inflicted on an individual by the people around him. This existential approach raises fundamental questions about our assumptions of sanity and madness, but it seems to fly in the face of genetic data, which suggest that there may be an inherited predisposition to schizophrenia (30). It may well be that an existential analysis is most appropriate to those individuals with hysterical psychosis, who seem very sensitive to clues from the environment. Interestingly, the genetic data of Rosenthal and associates (30) point to a distinction between those individuals with chronic and borderline schizophrenia, who seem to have a genetic loading for the disease, and those with acute schizophrenia, who do not.

Our thesis is that the phenomena associated with the syndrome of hysterical psychosis may be simplified and understood best by reference to the profound hypnotic trance states of which such individuals are capable. From this point of view such hysterical symptoms as fugue states, amnesia, and hallucinations are understood as spontaneous, undisciplined trance states. Some individuals, in the face of dramatic stress within their family, at their job, or social pressure of other kinds may succumb to a psychotic form of communication which is different from schizophrenia in phenomenology, course, and prognosis. The following case examples are illustrative.

CASE STUDIES

Case 1. Michael, a 15-year-old boy, was agitated and combative when he was brought to a hospital emergency room. He was placed in four-point restraints. He told the admitting physician that he was "possessed by demons of Satan." Michael reported that when he was lying in bed at home that evening he began to feel "shaky." He was then amnesic for the events that followed until he came to the hospital. His family noted that he began speaking "in a bizarre, awful voice, uttering obscenities, grunting, growling, and sniffing like a wild animal." They became frightened and attempted to hold him down, but he struggled with them. The family reported that he had "superhuman strength" while in this state, and the police were called to help get him to the hospital.

Michael and his twin are the youngest of five siblings in a middle class, religious family. Neither the patient nor anyone in his family had psychiatric history. Three months before his admission, Michael was moved to the bedroom of an older, sexually active sister, who was away at college during the week but returned on the weekends, sometimes with her fiance.

Ten weeks before admission, Michael became involved with his first girlfriend. This relationship lasted only 6 weeks, at which point his girlfriend left him in favor of one of his friends. He felt quite despondent over this, but was unable to verbalize his feeling until a week before his admission, when he took a butcher knife from the family kitchen, walked to his girlfriend's house, and made a superficial laceration on his arm. His girlfriend rushed him to her church, where he was told by a trusted minister that this was Satan's work.

When he was admitted one week later he was well oriented in all spheres and cooperative. He displayed goal-oriented speech, his affect was flattened, but he was noted as being mildly depressed. His memory was intact except for the events that had led to his hospitalization. He had suicidal ideation without intent and without vegetative signs of depression. He denied homicidal intent and did not have gross paranoid thought patterns. He said he had no hallucinations and was free of delusional thinking, with the exception of the thought that he might be "possessed by demons." His proverb interpretation was concrete, religiously oriented, and personalized. He was admitted to the hospital, diagnosed as having psychotic depression, and treated with trifluoperazine in doses of 10–20 mg/day. During his hospitalization, he was quiet and cooperative and did not show evidence of bizarre behavior at any time other than during family visits. The ward staff considered him a paranoid schizophrenic after he received a full medical and endocrine work-up that revealed no abnormality.

A week after admission, Michael was examined using the Hypnotic Induction Profile (HIP). His initial score was 4–5 on the 0–5 scale, and when tested 2 weeks later, his score was 5. He was capable of a profound trance state and was able to regress to the past as though it were the present. When regressed to the time of his initial possession episode, he began to relive it with dramatic affect, screaming and talking in a strange voice about his Satanic possession. The patient was taught to bring on one of these attacks and was told that he should practice bringing them on because he would gradually learn to modify them and they would become milder and milder. He was discharged from the hospital 2 days later. He did well at home at first and was quite proud of his ability to bring on the attacks. His family was counseled not to be frightened of him, to use calm reassurance, and to stroke him on the forehead, a signal which had been used during the hypnotic regression to end his reliving of the possession state.

Michael had a second brief hospitalization. While at home, he began crawling on the ground, barking like a dog, and appeared unable to bring himself out of the state. His mother did not panic and said to him sternly: "I don't care who you are, come to the table and eat your dinner." He concurred, lapping milk out of his glass and refusing to use silverware. Later, he became more excited and was returned to the hospital. He went through an episode of wild screaming and laughter and was instructed to put himself into a trance state and bring himself out of it when he was ready. He came out of this state and became quite coherent and quiet within a few minutes. Several days later, he was discharged from the hospital to be followed in family therapy.

One week after Michael's hospital discharge and 1 month after antipsychotic medication had been instituted, it was discontinued because of sedative side effects and the absence of any evidence that his improvement was related to the medication.

Further interventions were made with the family. Michael was moved to a bedroom of his own and the family was urged to focus less attention on him, particularly for his episodes of possession, and to reinforce other, more desirable behavior. The concordance of Michael's psychosis with his environment is intriguing. The family believes in demonic possession. Michael had been reading a Satanic bible and was quite interested in the occult. The information elicited during family therapy revealed that in many ways Michael was quite resentful of the family's intense emphasis on religion. One interpretation is that Michael's psychosis was his way of sayng, "OK, if you want religion, this is what you will get." This had been a chronic issue with him. The acute pressure that led to the temporary ego fragmentation and eruption of unconscious material seems to have been the combination of his being forced into intimate contact with his older sister and her sexuality and his loss of a first girlfriend. The patient could only view such a situation as demonic and in a sense he was possessed by forces beyond his control. His illness succeeded in getting him out of the oversexualized family situation in the short term and then getting him out of his sister's room in the long run.

What is particularly striking about this case is the patient's rapid and dramatic decompensation, his psychotic symptoms, and the rapid recompensation, which was apparently unrelated to the use of neuroleptics. The patient was clearly responding to intense family pressures, and he proved repeatedly to be highly hypnotizable. Although he was psychotic and delusional, his affect was atypical in that he was capable of forming good rapport in the intervals between his states of possession, and he showed periods of intense and dramatic affect. However, he was delusional and his thinking could easily be described as autistic, at least in certain areas.

At 1-year follow-up, Michael is doing well without medication or therapy.

Case 2. Ms. A, a 31-year-old separated working mother of four, first came to our attention after she was admitted to the hospital following a suicide gesture, the ingestion of a small number of aspirins. This action was precipitated by a sexual advance from a man she had recently met. She was a well-groomed woman who displayed exaggerated affect while giving an excessively detailed and dramatic history. She was oriented in all spheres, her memory was intact, and she was judged to be of above average intelligence. She was felt to be engaging and seductive.

Ms. A showed no evidence of delusional thinking and her associations were not loosened. She did, however, admit to both auditory and visual hallucinations, which took several forms. She reported being able to "see through people," i.e., she saw visual representations of her feelings about them. For example, she reported seeing flames burning inside of certain men. Ms. A also reported that before she married her husband she visualized and spoke with her husband's deceased father about the advisability of marrying his son. More recently, she said that when she was driving alone in her car, another woman would appear and the two of them would discuss whatever was bothering Ms. A. The imagined woman was noted to be much different from Ms. A in voice, appearance, and self-confidence. Ms. A also had a long history of numerous complaints such as headaches, low back pain, allergies, and "hypoglycemia," with no apparent organic basis.

The Hypnotic Induction Profile showed Ms. A to be highly hypnotizable (3–4 on the 0–5 scale). Individual psychotherapy was begun and no medications were prescribed. The patient was hospitalized for 2 weeks and discharged without medication to be seen weekly for psychotherapy.

The focal point in therapy was Ms. A's difficulty entrusting herself with any real independence. She had been prone to allow others to make decisions for her, although she became increasingly unhappy over this situation in her marriage. Her ambivalence over the sexual advances she received after her separation from her husband led to situations wherein consciously unwanted sexual involvement was expected of her. Her desire to please and her mistrust of her own judgment made this pattern very difficult and anxiety-provoking, and in this context she developed hallucinations and suicidal ideation. She was helped to see that her desire to please and her self-doubt were part of her character style and in certain interpersonal situations could be understood as spontaneous trance states. By learning to control her entry into and exit from the trance state, Ms. A was then able to control her anxiety about making decisions without resorting to hallucinations and suicide gestures as defenses. Hypnotic regression was used to help the patient relive stressful experiences and her reactions to them. As she reexperienced the anxiety and hallucinations, she was taught how to think through alternative responses and make choices independent of the desires of people around her.

When therapy terminated 5 months later, Ms. A had decided to divorce her husband, had moved out on her own, and had gained steady employment. She was also enjoying an active social and sexual life.

In this case, the use of hypnosis was primarily diagnostic. The patient had psychotic symptoms but was not treated as schizophrenic or psychotically depressed. Her high hypnotizability (a repeat HIP score 2 months later was 4–5) as well as her full affect, her well-groomed appearance, her long marital history, and the lack of positive psychiatric family history all led us away from the more malignant diagnoses. With this perspective, her symptoms were seen as nonverbal communications—she hallucinated flames inside men rather than discussing their sexual advances or more directly understanding and confronting her own sexual wishes. She was helped to structure her world so that she would have more control over her involvements and would value her own judgment and wishes more highly.

DISCUSSION

The two individuals we have described share several characteristics: relatively young age, a history of good premorbid adjustment, a rapid and florid decompensation related to environmental stress, and high hypnotizability. In each case, there was a dramatic symbolic content to the psychotic symptom. Michael was possessed symbolically by the devil but actually by the intense incestuous wishes that his living situation accentuated. Ms. A hallucinated flames inside of men, which represented sexual arousal. The psychotic symptoms can be understood in Chodoff's sense (16) as a means of nonverbal communication about the individual's plight.

Hypnosis was used as part of the differential diagno-

sis and also in the course of treatment. Each of these subjects was found to be highly hypnotizable, on the Hypnotic Induction Profile, and this trance capacity was used to demonstrate to the patients their ability to bring on their own symptoms and thereby to master them. Such patients are often extremely sensitive to environmental cues. It was necessary to remove Michael from the sexual stimulation within the family in order to maintain lasting control over his symptoms. Ms. A was taught to see her symptoms in a new light as a dramatic means of expressing a conflict over her change in marital status and sexual experience.

Because these individuals are so compliant, they can easily slip into a pattern of chronic psychological dysfunction if that is the expectation of their therapist and treatment environment. Thus, it is particularly important to identify such patients early. In these cases, even in the absence of the test of hypnotizability a poor response to antipsychotic medication, as in Michael's case, should suggest the possibility of alternative diagnoses to schizophrenia. Such individuals fall into the group of psychotic patients who are considered to have the best prognosis, with good premorbid adjustment, severe environmental stress, rapid decompensation, and relatively rapid recompensation. It is possible that the assessment of hypnotizability will be of significant usefulness in selecting those psychotic patients who will respond to individual and family psychotherapy and who are not in need of antipsychotic medication. Statistical research on the subject with a much larger number of patients is planned.

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Research and Development of Naltrexone: A New Narcotic Antagonist

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The author reviews the history of federally supported research in the field of narcotic antagonist therapy, focusing on social, political, and governmental issues. He describes the criteria for establishing an optimum narcotic antagonist, the legal guidelines for drug development, and the currently available narcotic antagonists. Research supported by the National Institute on Drug Abuse has indicated that naltrexone is the most promising drug in this category. The author discusses the safety and clinic use of naltrexone, the status of the NIDA naltrexone program, and plans for future development of the drug.

THE CURRENT naltrexone research program supported by the National Institute on Drug Abuse can be traced developmentally to its embryonic beginnings in the mid-1960s. At that time, Martin and co-workers (1) at the Addiction Research Center in Lexington, Ky., initiated a series of studies on the use of narcotic antagonists to treat opiate dependence. The studies were a practical outgrowth of the theoretical formulations elaborated by Wikler (2, 3) over the preceding years. The studies showed that a narcotic antagonist could effectively block the euphorigenic and dependence-producing properties of opiates in man. Furthermore, the antagonist produced neither physical dependence nor abuse potential. This was important because previous treatment drugs had the liability of producing their own degree of addiction.

These early clinical studies of narcotic antagonists might have faded into textbook obscurity had it not been for a number of concurrent social and political events. During the 1960s, experimental and compulsive drug use became widespread. All manner of substances appeared on the street market, and many individuals sought refuge in the blissful escape they supposedly provided. For many, one dead-end to which these pharmacologic avenues led was heroin addiction. Consequently, by 1970 the use of heroin, both at home and among our military personnel abroad, had reached alarming proportions. This situation provided the necessary impetus to both the executive and legislative branches of the government to authorize funding for expanded research on and treatment of opiate dependence.

On June 17, 1971, President Nixon signed a bill authorizing the creation of the Special Action Office for Drug Abuse Prevention (SAODAP) to coordinate the various resources of the federal government to check the continuing spread of illicit drug abuse. These resources previously had been scattered across more than 14 different agencies. In 1972 the Congress passed the Drug Abuse Office and Treatment Act, which was signed on March 21 (P.L. 92-255, section 224). Among the numerous provisions of the law was substantial financial support for the expansion of research on "long-lasting, nonaddictive, blocking, and antagonist drugs or other pharmacological substances for the treatment of heroin addiction." With these substantial mandates, SAODAP's first director, Dr. Jerome Jaffee, set the development of a safe and effective narcotic antagonist as one of the highest priorities of the new office.

THEORETICAL BASIS OF NARCOTIC ANTAGONIST THERAPY

Pharmacologically, a narcotic antagonist is a substance that has the ability to block the euphorigenic and dependence-producing properties of opiates (4). It is currently theorized that the antagonist accomplishes this feat by virtue of its structural similarity to narcotics. Thus, antagonists can occupy the same opiate receptor sites in the body as narcotics, thereby producing competitive inhibition of narcotics. Different narcotic antagonists also have differential abilities to produce both antagonistic and agonistic action. These differential properties are, of course, important in choosing the proper narcotic antagonist for therapeutic use. It should be noted that a pure narcotic antagonist differs greatly from a drug such as disulfiram. When alcohol is ingested by an individual who is taking disulfiram he or she then experiences a violent physical reaction that can be life-threatening. When heroin is injected in a dose that is completely blocked by the dose of a narcotic antagonist, there is no phys-

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ical reaction. Of course, an individual may attempt to overcome the blockade by using too much heroin, which can lead to a fatal overdose.

This unique class of antagonist drugs thus formed the basis of a potential treatment modality as outlined by Wikler (2, 3) and Martin and associates (1). These researchers postulated that operant conditioning plays an important role in initiating and perpetuating heroin use. Initially, the euphorigenic properties of narcotics probably act as strong reinforcers of what is conceptualized as "drug-seeking behavior" in the opiate-dependent individual. Thereafter, tolerance to the narcotic develops and slowly reduces the euphoric effects. In addition to the pursuit of pleasure (the euphoric effects), there is now within the individual a growing awareness of the need to avoid pain (the abstinence syndrome). This avoidance of the discomforts of abstinence also perpetuates the drug-seeking behavior. Finally, a hypothesized "conditioned abstinence syndrome" apparently can be precipitated by environmental stimuli that have been associated with opiate dependence in the past. Occasionally, dependent individuals have described the onset of withdrawal symptoms after opiate detoxification as a result of merely coming into contact with their previous environment. This conditioned abstinence syndrome may be characterized by increased reactivity to stimuli, prolonged abnormal autonomic responses, feelings of dysphoria, and often an intense "craving."

It was quite logical to conclude that the narcotic antagonist could be used to control these various determinants of opiate-seeking behavior. Since the antagonist would block not only the euphoria but also the physical dependence produced by opiates, reinforcement of conditioned abstinence through reestablishment of unconditioned physical dependence would be eliminated and gradual extinction of the response would occur. Eventual extinction of opiate-seeking behavior would be the expected consequence of blockade of the two primary reinforcing properties of opiates, namely, production of euphoria and suppression of the opiate abstinence syndrome, both unconditioned and conditioned.

The protection afforded by the antagonist would provide the needed time to aid the detoxified individual in altering his life course. In a close and humane psychotherapeutic milieu, the individual could then learn to regain control over his own destiny. He could begin to develop greater internal controls and greater independence and to extricate himself from the external web of drugs and environmental pressures that had previously ruled his life.

THE DEVELOPMENT OF AN OPTIMUM NARCOTIC ANTAGONIST

In light of the sociopolitical milieu and the enticing theoretical notions about the antagonists, we can easily understand why the development of a safe, effective antagonist was of the highest priority for SAODAP in 1971. SAODAP directors also recognized that the selection and development of such an antagonist was of no burning interest to the pharmaceutical industry. The projected expenditure of research and development funds and time seemed to outweigh the projected returns from what appeared to be a limited market.

By law, any new drug must pass through rigorous and controlled testing in several animal species as well as in humans before it can be marketed. The testing is divided into a preclinical phase and three clinical phases. In the preclinical phase, the gamut of toxicity studies should be carried out on at least two different animal species. If the drug proves to have a sufficient margin of safety in these toxicity studies, it may then be introduced into man for the purpose of gathering safety and efficacy data. Phase 1 of the clinical studies deals with the basic clinical pharmacology of the drug in man. This covers such areas as dosage levels, absorption rates, and metabolites. Phase 2 represents limited and quite controlled clinical trials intended to demonstrate the safety and relative efficacy of the drug. For reliable results, it is desirable that these studies be carried out within a double-blind placebo design. Phase 3 represents both controlled and uncontrolled clinical investigation in a much larger group of patients. The successful completion of this phase and the demonstration of drug efficacy is the final step before a new drug application (NDA) is submitted to the Food and Drug Administration (FDA). With approval of the NDA, the drug then is eligible for marketing. Of course, there is a constant financial risk involved in this process because development may be halted at any point on the basis of unacceptable toxicity or lack of efficacy.

It was with this long and complex procedure ahead that the federal government, through SAODAP and the Division of Narcotic Addiction and Drug Abuse (DNADA), undertook the development of a safe, effective narcotic antagonist. A research plan was initiated by DNADA in September 1971 to help organize such an efficient development. In this plan were outlined the necessary preclinical research, the procedures for clinical testing, and the cost and personnel estimates. In addition, the optimum characteristics of narcotic antagonists were described as follows:

1. Ability to antagonize the euphoric effects of opiates.
2. Absent or minimal agonistic effects, especially unpleasant ones.
3. Not physically addicting.
4. No increasing tolerance to its antagonistic actions.
5. Absence of serious side effects and toxicity even in chronic use.
6. Ease of administration, i.e., no surgery or painful procedure involved.
7. Long-lasting or moderate duration of antagonist effects.

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8. Absent or low abuse potential.
9. Reversible effects in case of medical emergency.
10. High potency, to allow administration of small amounts in a biodegradable vehicle.
11. Ease of availability and reasonable cost.
12. Therapeutic efficacy in the treatment of narcotic addiction.

By early 1972 there existed several antagonists at various stages of development, the purest of which was naloxone. Naloxone, which had been available as a narcotic antagonist for many years, seemed to be a potent antagonist and at that time showed almost no agonist action. Its main drawbacks as a therapeutic agent in opiate dependence were its high cost, the difficulty in synthesizing it, its very poor oral absorption rate, and, particularly, its short duration of action in the body. Naloxone had been approved by the FDA for short-term use in humans as an antidote for opiate overdose. In spite of its drawbacks, naloxone had met with limited success as an adjunct to treatment. This seemed encouraging for narcotic antagonist treatment in general.

Concurrently, another promising and potent antagonist, cyclazocine (5–7), was being developed. This drug demonstrated a longer duration of action (up to 24 hours with a 4-mg dose). However, cyclazocine had drawbacks—strong agonist properties including psychotomimetic effects and quite unpleasant feelings described as dysphoria. Despite the tolerance that develops to these effects, cyclazocine was not well received by the addict volunteers and soon acquired a bad street reputation. However, cyclazocine was successful in the treatment of some individuals, and the drug is still being used in certain clinics in New York City.

Three other compounds, designated as M-5050, BC-2605, and EN-1639A, were in the early stages of animal and human testing at this time. One of these, EN-1639A, seemed to be a potent antagonist and did not show the dysphoric and unpleasant side effects of cyclazocine (8). The drug, which came to be known as naltrexone, had a good duration: 50 mg seemed able to block narcotic action for 24 hours. By late 1972, there was a substantial supply of the drug available for testing. By mid-1973 it became evident that naltrexone fulfilled the criteria for an optimum narcotic antagonist to a greater degree than any of the other available substances (9–13).

In addition to the research progress that was being made, there were also administrative changes occurring within the government. In 1973, the Division on Narcotic Addiction and Drug Abuse was separated from NIMH and expanded into the National Institute on Drug Abuse (NIDA). Thus, from 1973 to 1974, NIDA and SAODAP shared the responsibility for the development of the narcotic antagonists in general and of naltrexone in particular. By mid-1974, as SAODAP was being phased out of existence, the entire direction and monitoring of the naltrexone research program fell to the NIDA Division of Research.

NIDA-SUPPORTED NALTREXONE RESEARCH

From 1973 to 1974, NIDA supported 26 grants and contracts in preclinical and clinical studies directly related to narcotic antagonists. This support totaled over $5 million. Approximately 17 of these grants and contracts dealt with the use of naltrexone in clinical situations, and they fit together into a rather loosely knit naltrexone research program. Five of these research clinics were selected to participate in the double-blind placebo study of naltrexone that was being conducted by the National Academy of Science (NAS). It was planned that such a study would satisfy in an elegant manner the phase 2 requirements for new drug development and would demonstrate the safety and relative efficacy of naltrexone when compared to placebo. The five NAS clinics in the double-blind study followed a standardized group of three research protocols. Each protocol involved subjects with a different type of opiate dependence. The Baltimore and New Haven clinics used only "postaddicts" in their research, the Detroit and Sepulveda clinics used only methadone maintenance addicts, and the St. Louis clinic used only "street addicts."

By contrast, the remainder of the grants and contracts consisted of a variety of controlled and uncontrolled clinical trials with naltrexone (14–20). Researchers in this group were free to use different protocols, different treatment settings, different types of dependent subjects, and different research questions. This group of grants and contracts was called "NIDA clinics," for want of a better title. These NIDA clinics were later divided into a group of "open clinical naltrexone studies" and a group of "behavioral naltrexone studies" in an effort to bring greater order and specificity to the overall naltrexone program. The former category consisted of studies that tested naltrexone within a variety of clinical contexts, and the latter category consisted of those which specifically attempted to test the original behavioral formulations discussed above.

All of these studies underway by 1974 were thought by NIDA to represent phase 2 testing, i.e., naltrexone was receiving exposure in limited clinical populations. In phase 2, one of the chief responsibilities of NIDA was to provide a watchful eye over the safety aspects of this drug when it was administered to humans. Consequently, a tight monitoring system had to be devised if the limited staff at NIDA was to function properly in detecting any ill effects of the drug. It was therefore decided to establish the same kind of monitoring for the NIDA research clinics that was in use at the five clinics in the NAS study. Biometric Research Institute (BRI) of Washington, D.C., was providing the monitoring and statistical capabilities for the NAS study and had developed a number of forms in conjunction with the NAS Committee on Evaluation of Narcotic Antagonists to carry this out. NIDA therefore contracted with BRI to provide a similar monitoring function for the NIDA clinics. This consisted of gathering

information on the monthly laboratory records, monthly physical and psychiatric summaries, weekly symptom checklists, and the daily treatment records from the various open clinical and behavioral NIDA studies. Thus, by early 1975, NIDA was able to gather a large quantity of both safety and efficacy information into the central data bank of BRI and to keep constant and close watch over any potentially unpleasant or harmful effects of the drug.

Up to the time this paper was written, no dysphoria or other undesirable psychic effects from naltrexone had been reported. The question had arisen in the past whether naltrexone caused an increase in blood pressure. According to the collective data, there seems to be a small (2–3 mm Hg) but not statistically significant rise in both systolic and diastolic pressure after the initial administration of naltrexone. However, by four to six weeks, blood pressure returns to baseline, and in many cases both systolic and diastolic pressure decreases by 2–3 mm Hg (reference 21 and personal communication from R. Resnick, December 1975). The only occasional side effect seems to be abdominal and gastrointestinal discomfort in some subjects. When this was found during the early stages of treatment, it was attributed to minor withdrawal symptoms because opiates were presumably still in the addict's system. However, these symptoms also have been reported later in treatment. Some researchers have found that these symptoms are in some cases relieved by antacids or by administering naltrexone to the addict after he has eaten. Thus, naltrexone may be a gastric irritant for some addicts.

However, to date there have been established no serious lasting side effects directly attributable to naltrexone. This antagonist appears to be a rather safe chemotherapeutic agent for the treatment of opioid dependence.

It is intriguing to consider what interaction naltrexone may have with the endogenous opiate-like compounds that have recently been isolated (22–24). Could the abdominal symptoms be related to such an interaction? If an individual who is being maintained on naltrexone has a quantity of this endogenous substance secreted into his system and it is, in fact, opioid in nature, is it not possible that some physical effects will be observed? This symptomatology might include minor withdrawal-like symptoms characterized by the abdominal discomfort described above. However, another explanation might be that naltrexone has some agonistic properties. Recent animal data, for example, document that even naloxone, thought to be a "pure" narcotic antagonist, has agonistic properties.

THE NALTREXONE PROGRAM: PRESENT AND FUTURE

Phase 2 of naltrexone testing has officially ended. The final reports from the CENA committee on the NAS study and from BRI on the NIDA studies, which were due in May of 1977, were submitted to the FDA for final approval to proceed to phase 3 testing. They have not been published as yet in the public sector. By the publication date of this paper, phase 3 of naltrexone testing should be well on the way to completion.

The preliminary data analysis certainly underscores the safety of naltrexone and also points to some measure of therapeutic efficacy. In the NAS study, 192 subjects received at least one dose of study medication—94 on naltrexone and 98 on placebo. The number of dropouts throughout the nine-month double-blind study was, of course, quite large—larger than expected. Nevertheless, at the end of nine months, approximately 7% of the subjects remained. During the middle months of the study (from $2^1/_2$ to $5^1/_2$ months), there appeared to have been a substantial difference in retention between the naltrexone group and the placebo group.

In addition to the NAS study, about 1,005 individuals have taken at least one dose of naltrexone in the other NIDA-sponsored clinics. The resultant retention curve of all these clinics combined is very similar to that seen in the NAS study. The final nine-month retention figure of 5% is somewhat lower than in the NAS study.

It seems that naltrexone is of long-term benefit to only a small percentage of addicted individuals. However, this small percentage represents quite a large population when one considers the total number of addicts in the United States. What may prove to be of greater therapeutic interest is naltrexone's 60% and 40% retention rates at one and two months. This fact may lead us to rethink the best way to use naltrexone in a treatment setting. In addition to long-term chemotherapeutic maintenance, we may begin to use it in conjunction with short-term crisis intervention or with behavioral techniques. Furthermore, naltrexone might successfully be used in a preventive contingency manner, i.e., the addict could ask to be put on the drug when he felt the need arise. Other factors that may be crucial to drug efficacy and that deserve further research include attitudinal, environmental, and sociocultural variables in the clinic personnel and in the addicts treated. In other words, is there a subgroup of addicts that can be described by these variables, as well as by personality variables, who would be candidates for naltrexone therapy?

Some of these questions hopefully will be addressed in phase 3 of the research. The possibility is being explored that Endo Laboratories, the pharmaceutical company which holds the patent on naltrexone, will take over the phase 3 development task. NIDA would like to have that phase begin soon so that naltrexone can reach the marketing stage as quickly as possible. As sentiment mounts against other dependence-producing agents such as methadone, we may well be turning to a therapeutic program that includes naltrexone as a pivotal component. It is NIDA's hope, therefore, that further research will continue to expand naltrexone's efficacy and will establish the place

of naltrexone therapy within the overall treatment approach to opiate dependence.

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Emergency Room Medical Clearance: An Educational Problem

BY MICHAEL P. WEISSBERG, M.D.

The term "medically clear" has a greater capacity to mislead than to inform correctly. The overuse of this term, especially in emergency room settings, may indicate difficulties in medical education and in the consultation/referral process between psychiatry and other specialties; further, it results in poor patient care. Nonpsychiatric physicians may prematurely refer patients as medically clear because of their unfamiliarity or discomfort with clinical psychiatry. Psychiatrists often ask for medical clearance of patients to hide their discomfort with or antipathy toward clinical medicine. The use of emergency room settings for interspecialty collaboration and training helps minimize the underlying difficulties that lead to the use of this term by fostering psychiatric skills in nonpsychiatrists and a sense of medical identity in psychiatrists.

THE USE of the label "medically clear" in emergency room settings hinders patient care by impeding the flow of information between psychiatric and nonpsychiatric personnel. The imprecision and varying meanings and uses of medical clearance are symptomatic of problems in medical education. At times, the medically clear label reflects and is used to obscure psychiatry's discomfort with medical problems and medicine's discomfort with psychiatry. The purpose of this paper is to examine some of the meanings and uses of medical clearance and to suggest some solutions for the educational problems that underlie this phenomenon.

The emergency room is an ideal but often neglected educational setting. Few settings are as appropriate for learning about interdisciplinary collaboration. However, when emergency room duty is treated by training programs as nothing more than a service obligation, residents and interns may invest themselves minimally. Their work may become sloppy and patients may be "disposed of" with inadequate diagnosis and treatment. In this sort of atmosphere medical clearance is a common reason for referrals of patients to the psychiatry service.

The term "medically clear" sounds legitimate and precise, especially to the inexperienced clinician. However, a thorough search of the literature revealed no information on the origins of the term. The label is applied in at least three situations when patients are referred to the psychiatric service.

1. It is thought that there is no medical illness present. An illustrative case is described below.

Case 1. A 26-year-old teacher complained of anxiety and spells of confusion. She was in the midst of divorce proceedings and had been disowned by her family because she was living with a man they considered beneath her. She had had two previous psychiatric contacts. The patient was medically cleared, diagnosed as having anxiety attacks, and referred for further psychiatric care. On closer examination, she also related some visual difficulties and problems with speaking and calculation. She was subsequently diagnosed as having had a stroke from which she later recovered.

2. A medical illness is known to be present but is not thought to be the primary cause of the patient's symptoms, as is shown in the following case.

Case 2. A 56-year-old woman with a history of chronic alcoholic liver disease was brought to the medical emergency room by relatives because she had become increasingly agitated since the sudden death of her husband 2 days before. The previous night the patient had misidentified her brother as her dead husband and had defecated in a trash can. She was medically cleared and referred to the psychiatry service with a diagnosis of acute grief reaction. The patient had a difficult time attending to the interview; she was intermittently agitated, disoriented to time, and unable to remember more than one object out of three after 5 minutes. The psychiatrist ordered a urinalysis because he noted slight scleral icterus. The urine contained bilirubin. After the patient was admitted to the medical service she was diagnosed as having fulminant viral hepatitis superimposed on chronic alcoholic liver disease. The patient's organic brain syndrome was making it impossible for her to grieve.

3. It is thought that the medical illness that was present no longer needs medical treatment, as illustrated below.

Case 3. A 26-year-old single unemployed man was admitted to the emergency room after he took an unknown quantity of pills because of an argument with his girlfriend. He was given gastric lavage and after an unsuccessful attempt to identify the medication was referred to the psychiatry service as medically clear. The patient was somewhat somnolent

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but could speak coherently. Although he was oriented, he had a slight defect in memory. His pupils were dilated and his pulse rate was elevated. The psychiatrist felt the patient was suffering from an anticholinergic syndrome and should be given cardiac monitoring in case he had arrhythmias. The medical resident agreed only after the pills were identified as amitriptyline.

Misdiagnosis and premature referral may occur with or without the patient being labeled medically clear, but the term does have an authoritative ring and implies exhaustive study. However, it does not convey what conditions were considered and how they were ruled out; it implies that everything was done and nothing was found. Thus, this designation can offer false assurance to the next professional who sees the patient and thus impedes the evaluation of the patient. The psychiatrist may too often be willing to accept his colleague's authoritative pronouncement rather than exposing himself to criticism, perhaps because he feels his medical skills are rusty.

WHY MEDICAL CLEARANCE?

If the medical clearance label is imprecise, why is it used by medical services and so frequently asked for by the psychiatry service? Perhaps it indicates discomfort in the relationship between medicine and psychiatry and each specialty's ignorance—usually feigned—of the other's field. These factors may result from a deficiency in medical education and a poor sense of medical identity in psychiatry.

Medicine's Contribution to Medical Clearance

Little attention is paid to the consultation and referral process in undergraduate and graduate medical education: some major medical textbooks (1) do not even mention it.

A physician's feelings of intolerance, frustration, or decreased self-esteem can interfere with patient care. If the physician feels that his competence is threatened he may minimize physical findings and tell the patient that his or her problems are "all in your head" (2). In the emergency room, the physician who becomes frustrated with a patient may refer him or her prematurely as medically clear. Further, there is an all too common belief that alcoholics, drug abusers, and suicidal individuals create their own diseases and are therefore less entitled than others to sympathetic and thorough care.

The nonpsychiatrist's unfamiliarity (and in some cases discomfort) with clinical psychiatry also promotes imprecise thinking in this area. For example, a patient with a preexisting psychiatric diagnosis is likely to be referred prematurely (e.g., case 1). Also, many physicians have difficulty conducting or interpreting the mental status examination (e.g., cases 1-3) and distinguishing between functional and organic causes for alterations in mood, thinking, or behavior (3). Even though many physical conditions influence thinking, mood, and behavior, a patient who "acts funny," is frightening, or is frustrating may be referred prematurely as medically clear (4).

Psychiatry's Contribution to Medical Clearance

Some psychiatric residents are uncertain about their status and competence as physicians, and others seem to believe they must "unlearn" medicine to become psychiatrists (5). At times they seem to feign ignorance when it comes to medical matters. Frequently a beginning resident discussing a patient who has been medically cleared ignores such elementary findings as vital signs and does not read thoroughly the medical workup, something that would never have occurred on medical rounds just a few months earlier. This active denial of medical expertise seems to be supported by Greden and Casariego's finding that 42% of psychiatric residents felt that they were psychotherapists first and physicians second (6). Only 39% of the residents in that study said they would do a physical examination of patients they planned to treat psychiatrically. These beliefs were either present before the residency began or developed soon afterward.

Psychiatrists' overvaluation of the medical opinions of other physicians seems to lead them to suspend their own clinical judgment, as is illustrated in the following case.

Case 4. A middle-aged alcoholic man came to the emergency room complaining of restlessness and anxiety. He was medically cleared as showing no evidence of severe alcohol withdrawal, despite an elevated temperature and pulse. However, within a few minutes of his admission by a psychiatric resident he showed rapidly increasing agitation. In a few minutes the patient was grossly delirious.

When this case was reviewed on psychiatric rounds the next morning, the psychiatric resident said he did not feel competent to question the medical clearance, although he had recently had excellent training in a city hospital emergency room as part of his internship. Because of his sense of medical inferiority, he ignored the ominous signs of elevated pulse, temperature, and a rapidly deteriorating course. He forgot that the consultant has the opportunity to make the last mistake.

Others have observed this tendency to repudiate medical identity by some psychiatrists in training. At the same time, older and presumably wiser heads question whether psychiatry should view itself as a medical specialty (7-9). Psychiatry's move away from medicine has been especially prominent in the community mental health field (5). Why does this distancing occur? For some residents it is a result of an original antipathy toward medicine, a problem that was unfortunately compounded for several years by the discontinuation of the internship requirement. Many psychiatric residency training programs seem to have ignored this problem. Gurel (10) reported that only 43% of the residency programs he polled offered interdisciplinary collaboration with medical colleagues, and that collaboration was very rudimentary. There was no mention of collaboration in emergency room work. Further-

more, some supervisors of psychiatric residents do not feel that it is their responsibility to consider, let alone to teach about, the medical problems of patients their residents see. Some supervisors probably screen out of their own practices patients with such problems and hence are unfamiliar with, and presumably uninterested in, medical matters.

Organization of Services and Medical Clearance

Physical separation of clinical areas decreases the possibility of cooperation and collaboration between psychiatric and nonpsychiatric colleagues. Emergency psychiatric services may be located far from the general emergency room, which further promotes an "either/or" diagnostic evaluation of patients. Many psychiatric and medical emergency services are staffed by overworked and inexperienced physicians who may quickly retreat to defensive postures when dealing with other services or difficult patients (11) and are more likely to hide behind the medically clear label.

SOLUTIONS

Problems in the consultation/referral process as exemplified by the medically clear label can be divided roughly into educational, organizational, and attitudinal shortcomings. However, if attention is paid to educational and organizational problems, changes in attitudes tend to follow.

Departments of graduate medical education should realize that the emergency room provides an ideal setting for interspecialty collaboration and training. The emergency room activity of each specialty should be integrated with that specialty's educational program rather than being regarded as a necessary and annoying service obligation. We have found that the institution of evening and weekend teaching rounds, in which the vast majority of senior psychiatric faculty are voluntary participants, has gone a long way toward integrating the emergency psychiatric service into the rest of the department. A full teaching program in emergency psychiatry (weekly supervision and conferences, daily work rounds) fully exploits the educational resources of the emergency room and leads to its being regarded as an important part of the residency training program. Residents feel supported and their efforts pay off in enhanced learning. An added benefit is that psychiatric faculty members have a regular opportunity to hone their diagnostic skills in a medical environment.

Some problems in the consultation/referral process may result from a shaky medical identity, and this factor should be considered in the selection of psychiatric residents. Efforts should be made to screen out applicants who are escaping from medicine and to encourage those who see psychiatry as a medical specialty. Supervisors of psychiatric residents should be alert to the active denial of medical expertise seen in beginning trainees.

Supervisors may also help residents by encouraging them to examine patients and to think through diagnostic possibilities before asking for consultation. This does not imply that the trainee (or faculty member) must stay current with the literature of all major specialties, but he should feel comfortable touching patients, examining them, and integrating his findings into an understanding of his patient's total difficulties.

Patients are also less likely to be "dumped" as medically clear if trainees witness cooperation and mutual respect among staff of different specialties. We have found that the morbidity/mortality conference, which members of many specialties attend, is an ideal vehicle for exploration of the consultation/referral process and for open discussions of difficult cases. Informational matters, such as the diagnosis and management of organic brain syndromes, depression, suicide, grief, hypochondriasis, etc., are presented at weekly seminars by psychiatric staff to nonpsychiatric house officers, medical students, and emergency room personnel.

Physical integration of services also reduces the potential for erroneous "either/or" diagnoses and the use of medical clearance. In order to foster further collaboration and to minimize areas of potential friction between specialties, the department of psychiatry in our facility has created a four-bed combined medical-psychiatric observation and short-term treatment unit in the emergency room. This integrated facility has made it unnecessary, for example, to decide precisely when an overdose patient becomes the responsibility of the psychiatric service.

CONCLUSIONS

The overuse of the term "medically clear" may indicate shortcomings in medical education and in the organization of emergency services. The consultation and referral process is a complex and often delicate form of interprofessional communication. Use of a shorthand phrase raises opportunities for misdiagnosis, premature referral, and misunderstanding. These mishaps can be minimized, and trainees' educational experience enriched, if directors of training programs pay attention to the educational and organizational factors that encourage the inappropriate and/or automatic use of the term "medically clear."

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Incidence of a History of Incest Among 18 Female Psychiatric Patients

BY ALVIN A ROSENFELD, M.D.

The author ascertained the frequency of reports of a history of incest among the 18 female psychiatric outpatients he first evaluated or treated in a one-year period. Six of these patients (33%) reported a history of incest. The author suggests that if this figure reflects actual prevalence, previous estimates of a history of incest among female psychiatric patients may have been mistakenly low. He discusses the implications and limitations of the statistics.

IN 1955 WEINBERG (1) stated that the official estimate of the incidence of incest was approximately one case per million population. Many professionals realized that this figure was low because it referred to cases reported to the court, but few expected a significantly higher prevalence. However, since 1955 many authors have been surprised to find a large number of cases of incest among female psychiatric patients. For instance, Lukianowicz (2) discovered that about 4% of all of the female psychiatric patients seen in County Antrim, Ireland, reported a history of incest. Browning and Boatman (3) found that 3.8% of all new cases seen at a child psychiatry clinic in Oregon had a history of incest; all but 1 were females. Similar observations have been made in psychiatric centers in other geographic locations (4, 5).

Studies of women with severely deviant social behavior have yielded even higher frequencies of a history of incest. For instance, among a population of delinquent adolescent girls confined to a state training school, 15% reported having had sexual relations with their fathers or stepfathers (6). Early in this century, a study found that 51 of 103 women arrested by the Chicago vice squad reported that their first sexual experience occurred with their fathers (7). As my associates and I have mentioned (5), it is difficult to assess to what extent the reports of incest among women with a history of socially deviant behavior are influenced by the complex motivational issues involved in the retrospective confession of a traumatic history to authority figures. Nonetheless, incest clearly occurs far more often than previously believed; yet many clinicians continue to consider incest a rare phenomenon.

Most studies of incest have derived incidence figures by adding up the numbers of spontaneous patient reports. To expect that this is an accurate incidence would be to assume that incest is usually reported when it occurs. Few experts believe this. However, most clinicians fear that direct questioning regarding a history of incest might elicit false or fabricated reports from suggestible and compliant patients who sense the therapist's interest. Although this possibility exists, Shengold (8) has noted that patients who have an actual history of incest "wish . . . very strongly that their memories could be regarded as fantasies" and may alter or withhold their history. A clinician who regards a patient's incestuous memories as fantasy may communicate this attitude to a compliant patient, who will then attempt to go along with the therapist's assumption and not report the incest.

In the present study I will describe the frequency of reports of a history of incestuous involvement among all of the female psychiatric patients I have seen in private practice in a one-year period. During this period I used a combination of active alertness to the possibility of incest and more direct questioning. I minimized suggestion to the best of my ability. In this study incest is defined as overt sexual activity involving genital contact between people too closely related to marry. Cases of genital contact for other reasons, such as hygiene, are excluded. Authors who use the term "incest" only when coitus is involved might not consider some of the cases that I would include as incest (e.g., cases of genital fondling). However, I feel that the definition of incest as coitus only is too limited. My associates and I (5) have discussed this issue.

SUBJECTS

Eighteen of the 21 female psychiatric patients I evaluated in a large group and private practice during 1976

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are included in this study. Three of my female patients were excluded from the study. One woman came for one evaluation session to discuss whether she should seek therapy for her young granddaughter who was not in her care; this women did not wish to discuss anything about herself. A second woman was disorganized, incoherent, and incapable of giving a history; she required immediate hospitalization. A third woman had a history of incest, but my notes were lost. I decided to omit this case from the sample because it could not be documented at the time of chart review.

Seventeen of the 18 patients in the sample were randomly assigned to me by the clinic secretary, who took referral calls and made her assignments on the basis of the clinician's having time available. She had no knowledge of my research and usually had only general demographic and insurance data about the new patient. The remaining patient had met me twice in a way related to my general psychiatric work with children. She specifically requested me as therapist. In a year of twice-weekly therapy, this patient never revealed that she had any knowledge of my work on incest, almost none of which had been published at the time she was in therapy with me.

All of the patients paid for the psychiatric evaluation and, in many cases, for psychotherapy with personal funds, private insurance, or Medicaid. For reasons extraneous to the study, none was covered by CHAMPUS. Fourteen of the women were self-referred or referred by another physician. Two were sent by a social service agency, and 2 others were referred by the court. Thirteen were white, and 5 were black. Thirteen were adults, 3 were adolescents, and 2 were children.

METHOD

In 1977 all of the charts of female patients first evaluated in 1976 were reviewed for a history of incest. I had obtained information in various ways. In some cases, it seemed feasible and appropriate to ask abut an incestuous history while taking a routine sexual history during evaluation. I asked about the sexual attitudes of the patient's subcultural group, extended family, and parents. If the information suggested overt sexual activity in the family, I asked further questions. In other cases, I felt this method was not appropriate or there was no reason to suspect an incestuous history. In these cases I inquired about the possibility of incestuous involvement if material presented during the course of therapy suggested it. For instance, one patient (patient 4) had unusually intense, overtly sexual dreams about a "father figure." When asked about her actual relationship with her father, she revealed an incestuous history. By the time of chart review, most of the patients had been asked about incest. However, I failed to get a sexual history from one young, manipulative adolescent (patient 7) who required hospitalization following a suicide attempt. Since she was coherent and could provide historical data, she is included in the sample.

The social class of the family of each patient was noted to investigate the common impression that incest is primarily a lower-class phenomenon. I estimated social class on the basis of the parents' jobs and the family's social and educational status. This assessment is therefore subjective; no formal assessment was made. In one case (patient 6), the patient's mother was functioning at a social level below that of her family of origin; since the grandparents had assumed primary responsibility for child-rearing, including housing, the child was considered to be in the grandparents' social class.

Intellectual deficits in 2 patients were determined by formal psychological testing.

FINDINGS

Table 1 summarizes the demographic data for the 18 patients. If the case was referred by the court or a social service agency, this is indicated as part of the chief complaint. Nine of the 18 patients were from lower- to lower-middle-class families, and 9 were from middle- to upper-middle-class families.

Six of the patients (33%) gave a positive history of incestuous experiences (patients 1–6 in table 1). Tables 1 and 2 summarize the data on these patients. There was a great deal of marital discord between the parents of these 6 patients which seemed an integral part of the "setting" of incest in these cases. The adults involved were biological fathers in 3 cases and a grandfather, a brother, and an aunt in the other 3. All of the adult participants were full-time residents in the same home as the child at the time incest occurred. All 6 of the cases of incest came from 14 patients who were self-referred or referred by another physician. All were white. Three were from the middle- or upper-middle class, and 3 were from the lower- to lower-middle class. Five were adults and 1 was a young child. None was judged to be psychotic. Only 1 patient revealed the history spontaneously. Two reported incest when asked about it during evaluation, 2 reported it later in the course of therapy when asked, and 1 young child told her grandmother some time later in therapy. Five were seen in psychotherapy, 4 for a period of more than 6 months. Once the accusation had been made (often with great reluctance) none withdrew the charge or changed it substantially.

COMMENT

I could not determine with certainty whether any of my patients who reported a history of incest fabricated the history or how many withheld it. However, it is noteworthy that none of the 4 patients referred by the court or a social service agency fabricated a history of incest to elicit sympathy or to affect her status in the

TABLE 1
Summary of Data on 18 Female Psychiatric Patients

Patient	Age Range	Social Class	Race	Diagnosis	Chief Complaint	History of Incest
1	24–29	Upper-middle	White	Hysterical character disorder	Difficulty concentrating	Positive
2	Over 40	Lower	White	Hysterical character disorder	Marital and sexual difficulties	Positive
3	30–35	Lower-middle	White	Hysterical character disorder	Marital and sexual difficulties	Positive
4	30–35	Upper-middle	White	Hysterical neurosis	Marital difficulties	Positive
5	18–23	Lower-middle	White	Hysterical neurosis	Intense murderous fantasies	Positive
6	Under 12	Middle	White	Adjustment reaction of childhood	School phobia	Positive
7	12–17	Middle	White	Hysterical character disorder	Suicide attempt	Negative
8	24–29	Upper-middle	White	Obsessional character disorder	Problems with girlfriends and boyfriends	Negative
9	24–29	Lower-middle	Black	Hysterical character disorder	Sexual and marital difficulties, depression	Negative
10	30–35	Lower-middle	Black	Depressive neurosis	Marital difficulties	Negative
11	12–17	Lower-middle	White	Character disorder	Court referral; legal difficulties	Negative
12	24–29	Upper-middle	White	Ambulatory schizophrenia	Severe anxiety	Negative
13	24–29	Lower	Black	Character disorder; borderline retardation	Court referral; legal difficulties	Negative
14	12–17	Lower-middle	Black	Retardation	Social service referral	Negative
15	18–23	Middle	White	Hysterical character disorder	Sexual and marital difficulties	Negative
16	24–29	Lower	Black	Hysterical character disorder	Difficulties with family and placement; postlegal difficulties; social service referral	Negative
17	30–35	Middle	White	Hysteria	Depression, sexual and marital difficulties	Negative
18	Under 12	Middle	White	Oppositional character	Sadness	Negative

TABLE 2
Summary of Data on 6 Female Psychiatric Patients with a History of Incest

Patient	Adult Involved	Incestuous Act	Duration of Incest	Age of Patient When Incest Started	How History Revealed	Duration of Therapy
1	Father	Masturbation, cunnilingus, fellatio, and exhibitionism	17 years	8	Spontaneously	More than 6 months
2	Brother	Intercourse	Once	9	During evaluation	None
3	Father	Attempted rape (stopped before penetration)	Twice	13–16	During therapy	4–6 visits
4	Father	Mutual masturbation	Once	12	During therapy	More than 6 months
5	Maternal grandfather	Genital manipulation of child	Unclear—several months to 1 year	5	During therapy	More than 6 months
6	Aunt	Cunnilingus	Weeks to months	5	During therapy, to grandmother	More than 6 months

referring agency. Since most of the patients who reported a history of incest were seen in weekly therapy of at least 6 months' duration, I had an opportunity to observe whether any reports changed significantly over time and whether any were elaborated or withdrawn.

A surprising percentage of this small random sample of female psychiatric outpatients revealed a history of incest when asked. Only 1 patient reported the history spontaneously. Since not all of the patients were seen over an extended period of time, and since half of the patients revealed the history only later in therapy, this is the lowest possible estimate for this sample.

Since 4 of the 6 women who reported a history of incest came from families native to a particular geographic area, it is possible that incest is more common in this geographic area than in others. Although there have been no previous reports of this, two other clinicians in the area commented on the high local incidence of incest, which they had not seen in different

geographic locations. Other clinicians, however, said they saw no more cases of incest in this area than they had seen elsewhere. However, Proctor (9) reported a high incidence of hysteria in children in a neighboring state. He felt this had some connection with the area's religious, antisexual attitudes, combined with practices resulting in early overstimulation of the child, such as permitting the child to sleep in the parents' bed over a period of a number of years. He did not indicate that there were any more overt sexual interactions between the parents and children involved in the symptom formation.

The frequency of incest found in the present study does not appear to be connected to the social class of the patient's family, but it is impossible to know whether this reflects differential reporting patterns. In addition, I have never seen a case of incest involving blacks. Whether this reflects reality or is an artifact of case selection is hard to assess. The *California Sexual Deviation Research* (10) noted lower than expected numbers of blacks among imprisoned sex offenders in the 1950s: although blacks made up 20.5% of all men received at the state prison reception center, they were only 9.9% of all the convicted sex offenders and only 5.6% of all those who committed sex crimes against children. This could be an artifact if, for instance, sex crimes against children were less frequently reported in the black community or incest was less common among blacks because there were fewer intact families. According to a private communication, those who work in black communities see a significant number of cases of incest among blacks. If there are actual differences in prevalence of incest, it again highlights the variability in incest rates among different subcultural groups.

There has been a historical question of whether patient reports of incestuous experiences might be fantasy. I cannot determine with certainty whether the reports of my patients are true because only corroboration by a witness could provide indisputable proof. However, I think that many, if not all, of these reports reflect reality. My associates and I have discussed the basis for this opinion elsewhere (11). Even if half of the reports were false, there would still be an unusually high incidence (17%). If these figures do not reflect a local peculiarity, previous estimates of a history of incest among female psychiatric patients may have underestimated the actual incidence because they relied on spontaneous patient revelations rather than on more direct questioning. Many patients are extremely reluctant to reveal a history of incest, particularly if they feel the evaluator will be shocked or judgmental. All of the patients in this sample had told no one, or at most only one or two close relatives, of the experience. Although it may be possible to "suggest" this history to patients, I tried to avoid this to the best of my ability.

If my conclusion is accurate, it raises the question of whether Freud's early theory that sexual molestation in childhood causes hysterical symptomatology had some merit. This question is particularly pertinent as we grow more knowledgeable about family dynamics and the role of parental seductiveness that stops short of actual incestuous activity. This situation can create striking hysterical symptomatology in the absence of overt action; I have discussed this more fully elsewhere (12, 13).

The effects of the early sexual experiences of these patients are difficult to ascertain. None of the patients was overtly psychotic, and, in terms of social adjustment among the adults, most seemed to have made adequate adjustments, usually similar to those of their parents. There was a high level of marital discord, sexual dissatisfaction, and hysterical characterological disturbance among the patients who had experienced incest. However, it is difficult to assess whether this was related to incest because the patients without a history of incest revealed a similar range and type of symptoms. The effects of incest may be more subtle and related to object relationships (5).

Four of the 5 patients who had experienced incest and who entered psychotherapy benefited (in varying degrees) from therapy. Most of my therapeutic work with these and other patients with a history of incest focused on issues of identity, independence, feelings of safety (14), and the modulation of interpersonal distance (15), not on sexuality. None of the patients in therapy seemed to use her incestuous past to obtain sympathy, although some were seductive in the earlier phases of therapy. Knowing of their past incestuous experience was helpful in understanding some of their feelings and transference reactions.

CONCLUSIONS

It is impossible to make any significant generalizations about the effect of incest on ego structures and object relationships on the basis of this small sample. Only further research will ascertain the effects of early overt sexual experience in the family specifically related to the sexual experience, as opposed to the more general outcomes of grossly disturbed family relationships noted in many incestuous and nonincestuous families. In any case, if therapists become alert to the possibility of a history of incest among their patients, they may be more effective and empathic in their therapeutic endeavors with these patients.

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Objective Personality Changes in Residents of a Therapeutic Community

BY JOEL G. SACKS, D.S.W., AND NORMAN M. LEVY, M.D.

The authors tested several groups of drug-abusing young adults who were residents of a drug-free therapeutic community. Three instruments were used: staff ratings, sociometric ratings by members of the community, and MMPI scores. All three measures showed objective evidence of decreasing psychopathology correlated with length of time in treatment, demonstrating the effectiveness of the therapeutic community in the rehabilitation of drug abusers. Individuals who left before completing treatment had MMPI scores indicating inability to develop social relationships; this elucidates a probable cause of the dropout phenomenon.

THE DRUG-FREE RESIDENTIAL therapeutic community is one of the two major methods for treating drug-abusing young adults; the other is methadone maintenance (1, 2). Based on a concept of self-help, the therapeutic community uses positive peer pressure and informal counseling procedures as instruments for personality change. Values that make possible a socially acceptable lifestyle are inculcated by a staff of paraprofessionals (ex-addicts) through a system of rewards and punishments and by the residents themselves through encounter groups that confront individuals with their antisocial attitudes.

This study was done in an attempt to find answers to the following questions: 1) Does the therapeutic community actually produce changes in the personality characteristics of its residents? Although there have been anecdotal accounts of dramatic characterological alterations in individuals who have gone through a therapeutic community (3), it is difficult to find hard data to confirm such changes. 2) Why do some residents drop out within a few weeks of admission and others after 6 or 8 months, while others remain for the complete program of 14 to 18 months?

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METHOD

Sample

The Renaissance Project, which is modeled along the lines of Phoenix House (4), serves a predominantly suburban population of drug-abusing young adults. They are drawn from the Westchester County, New York, area. Their median age is 21. They are primarily white (60%), male (85%), and Catholic (45%). Most come from blue-collar, lower-middle-class families (70%). The characteristics of this population have been described in detail elsewhere (5). The majority have used heroin or other opiates.

Design

Because of our stated purposes, a panel study entailing the measurement of residents at two different times was designed and carried out. Over a period of 14 months there were four testing periods, which produced pre- and posttest measures for three different resident groups: 1) group 1, tested in June and October, 1974 (N=20), 2) group 2, tested in October 1974 and March 1975 (N=16), and 3) group 3, tested in March and August, 1975 (N=14). No resident was in more than one group.

The cumulative total of residents tested by this procedure at least once was 114. In addition to the 50 residents who were tested twice, 64 residents received an initial test but did not remain in the program long enough to be retested. There were 44 dropouts in this group. A cross-sectional analysis was conducted on the entire sample of 114 to supplement the test-retest analysis and to offer opportunities to corroborate its findings.

The 114 subjects were divided as follows: cohort 1, 29 residents whose average time in the program at their initial testing was 3 months (range, 0–241 days); cohort 2, 20 residents whose average time in the program was 12 months (range, 242 days–1½ years); cohort 3, 21 residents in the final phase of the program, graduates, and senior staff people, whose average time in the program was 20 months (range, 19–24 months); and the fourth group of 44 dropouts whose average time in the program was 9½ months.

Assessment Procedures

At each test period three assessment procedures were used: staff ratings, sociometric ratings, and the full-scale MMPI. For the staff ratings the staff was

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TABLE 1
Changes in Test Scores of 50 Therapeutic Community Residents Over Time

Measure	Group 1 (N=20)[a]				Group 2 (N=16)[b]				Group 3 (N=14)[c]			
	Time 1	Time 2	Mean Change	p	Time 1	Time 2	Mean Change	p	Time 1	Time 2	Mean Change	p
Staff ratings	14.3	16.7	2.4	<.001	15.2	15.6	0.4	.276[d]	14.4	15.3	0.9	.088
Sociometric ratings					101	115	14	.014	97	108	11	.016
MMPI scales[e]												
Infrequency (F)	75.8	69.8	6.0	.012	68.7	62.0	6.7	.012	75.1	66.8	8.3	.008
Depression (D)	69.5	62.4	7.1	.017	68.8	63.1	5.7	.026	75.5	69.1	6.4	.053
Psychopathic deviate (Pd)	79.1	79.3	0.2	n.s.	80.2	72.2	8.0	.001	81.8	75.4	6.4	.026
Paranoia (Pa)	69.3	64.1	5.2	.043	62.5	61.9	0.6	n.s.	71.8	66.1	5.7	.048
Lie (L)	44.4	45.7	1.3	n.s.	42.8	46.4	3.6	.0005	44.4	48.9	4.5	.030
Introversion (I)	57.0	55.4	1.6	n.s.	60.8	56.5	4.3	.01	64.6	55.7	8.9	.0005
Ego strength (Es)	43.1	47.9	4.8	.005	45.1	52.1	7.0	.006	38.7	46.3	7.6	.030
Maladjustment (Mt)	81.5	76.9	4.6	.090	80.9	72.7	8.2	.020	88.8	77.4	11.4	.002
Anxiety (A)	61.4	58.8	3.6	.085	63.3	57.9	5.4	.003	67.3	59.3	8.0	.005
Prejudice (Pr)	60.3	59.3	1.0	n.s.	59.9	55.3	4.6	.036	65.2	59.7	5.5	.034
Status (St)	54.8	52.6	2.2	n.s.	53.0	56.8	3.8	.060	48.5	53.8	5.3	.016
Iowa manifest anxiety (At)	64.3	62.1	2.2	n.s.	63.8	59.1	4.6	.013	71.7	64.3	7.4	.012
Social desirability, revised (So-r)	19.4	23.7	4.3	n.s.	18.1	29.3	11.2	.01	9.9	23.6	13.7	.01

[a]4-month interval between tests.
[b]6-month interval between tests.
[c]5-month interval between tests.
[d]n.s.
[e]Only those scales which showed a significant difference for two or more test groups are listed.

asked to rate resident performance in the five areas of work function, relationships to peers, relationships to staff, participation in therapy programs, and general attitude. Each of these areas was rated on a 5-point scale according to which a rating of 5 signified "very good," a rating of 3 signified "fair," and a rating of 1 signified "very poor." The sum total of ratings received became the resident's score for the time period.

The sociometric procedure consisted of asking the residents to write in the names of peers who in their judgment fell into any of the four following social categories: 1) a person the rater would like to attend a party with, 2) a person the rater considered happy in the program, 3) a person likely to be a leader, and 4) a person considered most unhappy. Each time a resident's name was written in a given category he or she received 1 point. If that resident's was the first name cited, he or she received 2 points; the score was positive for the first three items and negative for the fourth. The total score was the algebraic sum of all scores obtained.

The full-scale MMPI has been used extensively in studying drug addicts and drug abusers. We used the computerized version, which was scored electronically by a testing service.

RESULTS

Table 1 summarizes the results of the test-retest study. Each of the three groups underwent change in the direction of improved adjustment and reduced pathology. Moreover, each of the independent assessment procedures provided confirmatory evidence of this change. Not only was there significant improvement on MMPI scales for each group at the time of retesting, but the staff ratings and sociometric scores also tended to show improvement. One-tailed correlated t tests were used to determine significance.

For each test group the staff ratings at the second testing were higher than at the first. In group 1, the difference of 2.4 points was highly significant (see table 1). For group 3, the .92-point difference approached significance. Only in the case of group 2 was the difference slight and not significant. It is interesting to note that the staff ratings provided the most conservative picture of the degree of change.

Sociometric ratings were available for both testings for groups 2 and 3 (see table 1). Both of these groups showed a large change that was quite significant; for group 2 the second sociometric rating was 14 points higher than the first, and for group 3 it was 10.8 points higher.

Finally, for all groups there was a pronounced pattern of significant change on many but not all of the MMPI scales. These changes were positive and can be interpreted as being in the direction of reduced symptomatology and improved functioning. Thus all three groups showed significant or almost significant reductions in indicators of pathology or, conversely, improvements on measures of health on the infrequency, depression, ego strength, and maladjustment scales. Two of the groups showed a significant improvement on 12 additional scales. These included subtests considered to represent measures of pathology: the anxiety, Iowa manifest anxiety, and schizophrenia scales. In general, we found that changes occurred in the same areas reported by DeLeon and associates (6),

who believed that they represented change in pathological signs.

In the cross-sectional analysis, 8 MMPI scales showed differences of 12 to 18 points between cohort 1 (whose average time in the program was 3 months) and cohort 3 (average time, 20 months) and another 10 differences of 7 to 10 points. In almost all cases the direction of change was in favor of cohort 3, who spent the most time in the program.

These findings seem to suggest a relationship between exposure to the therapeutic community and reduction in pathological symptoms.

In general, dropouts tended to perform in the way one might have expected given their average time in the program (9½ months). Their average scale scores tended to fall in between the ones obtained by cohorts 1 and 2, whose average time in the program was 3 and 12 months, respectively. However, the dropouts scored lower than expected on 4 subscales, 3 of which were found to have no significant correlation with time spent in the program. The fourth was the social introversion (SI-O) scale, which was found to correlate strongly and significantly with the actual sociometric ratings ($r=.33$, $p=.004$); this scale also had one of the strongest correlations with time spent in the program ($r=.46$, $p=.001$).

DISCUSSION

The results of this study show clearly that drug abusers involved in our therapeutic community underwent personality changes in the direction of diminished pathology, and that the longer they stayed in treatment the greater were the alterations according to the MMPI and other instruments. The general configuration of the profiles is quite similar to those obtained by earlier researchers (7, 8): typically, the scores on the psychopathic deviate and hypomania (Ma) scales were above 70 and represented the peaks of the curve.

In our study, the performance curve of the 20 residents who stayed in the program for an average of 12 months approached that of the 21 graduates, but the pattern of the 29 residents who stayed in the program for an average of 3 months had some interesting differences. In the latter group, there was the same tendency to peak at the psychopathic deviate and hypomania scales, but this group also had high scores on the depression and schizophrenia scales. This suggests either of two possibilities: 1) the graduates, being an older generation of drug abusers, had a different pattern of disturbance than the younger group of abusers, or 2) some of the scales, particularly depression and schizophrenia, are more likely than others to change.

A recent study by Zuckerman and associates (9) demonstrated results similar to ours. In the group closest to our sample, which they termed "male hard drug," the pretreatment profile was very much like that of our residents who stayed for only 3 months, with peaks at the depression, psychopathic deviate, schizophrenia, and hypomania scales. The posttreatment profile of this group showed marked drops in the depression and schizophrenia scales, a slight reduction in the psychopathic deviate scale, and virtually none in the hypomania scale, again like our graduate profile. This tends to confirm the suggestion that certain personality elements are selectively affected by involvement in the therapeutic community.

Of particular interest in our study is the lack of any change in the hypomania scale and the MacAndrews scale, which measures tendency toward addiction. These findings suggest that since 2 of the scales that distinguish the addictive profile do not show any improvement, the basic personality substratum of the drug abuser may not be influenced by treatment despite amelioration of symptoms in other areas.

The second major concern of our study was an attempt to elucidate the dropout phenomenon. The dropouts were expected to demonstrate pathological signs to a greater degree than those who stayed in the program when their respective first-test MMPI subscores were compared. In effect, we assumed that the more disturbed members of the community could not be reached by the program and thus would leave it. Instead, we found that the dropouts, in keeping with their average time in the program, tended to fall between the beginners and the residents who stayed in the program. Thus, rather than appearing to be more disturbed than the others, the dropouts seemed to be a group that made some progress and then terminated.

There was one significant exception to the general trend, namely the performance of dropouts on the social participation scale. Here the dropouts did only as well as the beginners. In other words, although the general tendency was to improve somewhat in most areas, in this particular dimension they had not improved at all. This suggests that the crucial factor in the dropout phenomenon is not the overall degree of disturbance but the extent to which the resident is socially accessible to the group experience and capable of achieving some status in relation to peers and other community members.

CONCLUSIONS

Our study demonstrates that there is a definite correlation between length of time in a therapeutic community program and decrease in objective indicators of pathology on the MMPI, staff rating form, and sociometric form. These findings suggest that the therapeutic armamentarium of a therapeutic community such as Renaissance Project is effective in treating drug abusers. Further research is needed to determine the reasons that certain MMPI scales, and their concomitant personality elements, are preferentially improved by involvement in the therapeutic community.

The fact that certain scales on the MMPI that correspond to the underlying character structure of drug abusers did not change suggests that the basic person-

ality configuration was untouched while peripheral pathology was modified, so that the drug-abusing tendency was effectively suppressed. Opposing influences outside the therapeutic community might be expected to have the reverse effect, intensifying psychopathology and causing drug-abusing behavior to recur.

Individuals who dropped out of the program before completion differed from the others mainly in their inability to develop social relationships, as indicated by lack of improvement on a particular MMPI scale that correlated highly with ratings. Thus, initial testing to discover potential dropouts using these instruments would allow extra effort to be brought to bear on such individuals to reduce the risk of their dropping out.

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Cognitive and Affective Responses to Lithium in Patients with Organic Brain Syndrome

BY KENNETH H. WILLIAMS, M.D., AND GERALD GOLDSTEIN, PH.D.

The authors describe a series of patients with organic brain syndrome who showed a dramatic clinical response to lithium carbonate therapy. None of the patients had been diagnosed as manic-depressive. Most had extensive psychiatric treatment experiences and had been given both affective and cognitive diagnoses. Six of the eight patients also qualified for the diagnosis of alcoholism. They had been treated with a wide variety of psychotherapeutic medications. Lithium was found to be rapidly and dramatically effective in patients with static lesions of the central nervous system who showed a combination of dementia and agitated depression.

YOUNG AND ASSOCIATES (1) reported on three patients who had manic-depressive illness in combination with organic brain syndrome and who responded well to lithium treatment. The purpose of our report is to summarize the treatment of 10 patients with organic brain syndrome but without manic-depressive illness, 8 of whom also responded to lithium. The organic brain syndromes were well documented and were associated with chronic brain disease of a static or slowly progressive type. Six of the patients were alcoholics (although this was the primary diagnosis for only 2 patients), all had a significant degree of dementia and had a sleep disturbance, and most had a mood state characterized by episodic agitation and dysphoria. Motor restlessness, manifested by pacing or similar rapid, repetitive activity was common.

The effectiveness of lithium in treating patients who have agitated depression, or "mixed mood state," has been documented by Himmelhoch and associates (2). There is also an extensive literature, which has been reviewed by Schou (3) and Kline (4), on the use of lithium to treat alcoholics. Lithium therapy for organic brain syndrome has been discussed by Rosenbaum and Barry (5), Mehta (6), and Taylor and Abrams (7). Jefferson and Greist (8) reviewed the findings for both of these diagnostic groups and concluded that lithium seemed to be effective in alcoholics when a bipolar affective disorder or other cyclic mood disorder was also present and that the organic brain syndrome research was somewhat contradictory but generally positive. Our study was stimulated by this literature and by our experience with the first patient we will describe, whose affect and cognition improved dramatically with lithium after having shown no significant change with other psychotropic medications.

METHOD

Pertinent data on the 10 patients are presented in table 1. The patients were all hospitalized in a large Veterans Administration neuropsychiatric hospital on a ward that specialized in evaluating and treating organic brain syndrome. The ratings in the table are based on clinical observations made by the ward team.

All patients had symptoms of depression, agitation,

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The papers in this section are grouped around a specific topic. Publication here does not, however, imply that the Editor considers this material to constitute a comprehensive analysis of the topic.

TABLE 1
Major Clinical Features of Lithium Responders and Nonresponders with Organic Brain Syndrome

Case Number	Past Psychiatric Diagnoses	Depression	Agitation	Sleep Disorder	Alcoholism[a]	Abnormal CT Scan
Lithium responders						
1	Korsakoff's syndrome	Moderate	Severe	Moderate	Yes	No
2	Schizophrenia, catatonic type; subarachnoid cyst	Severe	Severe	Mild	Yes	Yes
3	Schizophrenia, chronic undifferentiated type	Mild	Severe	Moderate	Yes	Yes
4	Cerebrovascular accident	Moderate	Severe	Severe	Yes	Yes
5	Depression	Moderate	Moderate	Severe	Yes	No
6	Psychotic depression, brain atrophy	Severe	Severe	Severe	No	Yes
7	Schizophrenia, paranoid type; atypical dyskinesia	Mild	Severe	Mild	No	No
8	Korsakoff's syndrome	Mild	Severe	Moderate	Yes	—
Lithium nonresponders						
9	Organic brain syndrome, cardiac arrest	Mild	Severe	Mild	No	Yes
10	Organic brain syndrome, Alzheimer's disease	Moderate	Moderate	Mild	No	Yes

[a]The presence of alcoholism was assessed by record reviews and social histories done on the ward. In some cases alcoholism was not formally diagnosed but was clearly indicated by the records and/or information provided by the patient or relatives.

and sleep disorder. The sleep disorders consisted primarily of difficulty falling asleep, which extended in 1 case to lengthy periods of hyposomnia. Nine patients received CT scans, 6 of which were reported as abnormal. In 4 cases the abnormality was generalized atrophy, but in the other 2 cases there was substantial focal brain damage, resulting in 1 case from a cerebrovascular accident and in the other from a subarachnoid cyst. The 2 patients with substantial damage had received neurosurgical treatment many years previously. Because the patients had long-term chronic illnesses, they had been given numerous anticonvulsant, antipsychotic, and antidepressant medications, none of which was effective—their symptoms persisted and they continued to require hospitalization.

Lithium treatment was carried out in a conventional manner. Before initiation of treatment, patients were given thyroid studies, EEGs, ECGs, and other routine laboratory procedures. The starting dose was typically 900 mg/day, which was raised after a brief period to 1200 mg/day. Blood levels were monitored periodically, and treatment was continued on an indefinite basis. The patients have been maintained on lithium for over a year without notable adverse consequences. When patients were discharged they were advised to continue with the lithium and to report back regularly for monitoring of blood levels.

RESULTS

Of the 10 cases we have described, 6 had clear and dramatic positive responses to lithium. Two patients had partially positive responses that can be viewed as equivocal successes. Lithium treatment was also attempted with two other agitated patients with organic brain syndrome who did not respond at all. One of these patients had experienced acute massive diffuse brain damage associated with cardiac arrest, and the other had advanced Alzheimer's disease. Without a detailed analysis of each case, it is difficult to describe the varied and frequently dramatic positive responses to lithium among these patients. Space limitations do not permit full documentation here[1]; briefly, changes included marked reduction of agitation and pacing, remarkable improvement of memory, particularly in one of the Korsakoff patients, improvement of speech in an aphasic patient, and essential disappearance of abnormal movements in a patient with a severe dyskinesia that resembled Huntington's chorea. We will describe 3 of the more dramatically successful cases.

Case 1. Mr. A, a 52-year-old widowed retired man, was admitted to the hospital after he threatened his son with a knife while he was intoxicated. Mr. A had a history of episodic, unpredictable, violent behavior, multiple hospitalizations, amnesia, confabulation, and denial. He had a 3-year history of difficulty sleeping and a 15-year history of alcoholism. In spite of medical complications associated with drinking, he was unable to remain abstinent and frequently expressed a desire to drink. His medical history included peripheral neuropathy, alcoholic liver disease, and a surgically treated peptic ulcer.

Examination revealed spider angiomata and gynecomastia. He was disoriented to time and place, had a broad-based gait, and peripheral neuropathy. Positive neurologic signs included bilateral graphesthesia, absence of ankle jerks, resting tongue tremor, and positive palmomental, Myerson, and snout signs. Laboratory tests, including liver function tests, were normal, as were the EEG and CT scan. The patient could not recall recent events and in general had

[1]Detailed case histories are available on request from the first author.

an amnesic disorder quite characteristic of Korsakoff's syndrome.

During the 2 consecutive years of his hospitalization Mr. A remained exceedingly agitated, depressed, and irritable. He refused to do anything asked of him, used abusive language, and frequently struck out at people. He was confused, difficult to direct, unable to care for himself, and had episodes of incontinence. He got in and out of bed several times a night. Treatment with chlorpromazine (up to 1000 mg/day) did not control his agitation or relieve his other symptoms.

After initiation of lithium, improvement was noted within 2 weeks. He became quieter and less restless, and his pacing diminished significantly. His sleep was greatly improved and the depressive symptomatology cleared. The temper outbursts essentially disappeared, and he became cooperative, as exemplified by his willingness to take neuropsychological tests that he had belligerently refused previously. These tests revealed moderately impaired function, with normal-range verbal and performance IQs. His incontinence ceased and his self-care improved. Approximately 1 month after initiation of lithium Mr. A returned from a weekend pass and was able to recount, in good detail, where he had been and what he had done. Two months after the initiation of lithium treatment he was discharged home. Under the care of his family he has continued to take his medication and has not been drinking for 1½ years.

Case 2. Mr. B, a 59-year-old retired railroad employee, had a long history of psychiatric hospitalizations dating back to his early 20s. The diagnosis at that time was schizophrenic reaction, catatonic type. The patient also had a history of alcoholism, with alcoholic blackouts, loss of control, and family, legal, and medical problems secondary to drinking. In addition to delusions and hallucinations, he had been consistently described as violent, combative, assaultive, angry, restless, and irritable. He experienced severe recurrent depressions, went without eating or sleeping for extended periods of time, and had made one major suicide attempt. He could not be managed at nursing homes because of episodes of aggressive behavior. In 1972, he underwent surgery to drain a subarachnoid cyst in the parieto-occipital area of the left cerebral hemisphere. After the operation, he had a chronic brain syndrome and a mixed conductive-expressive aphasia.

CT scan and blood flow studies during the present hospitalization suggested a lesion in the distribution of the left middle cerebral artery, and the EEG showed a right cortical focus. Because of the recalcitrant nature of Mr. B's psychiatric and physical pathologies, he received a variety of medications over the years, all with little success, regardless of the amount or combination used. These included three benzodiazepines, four phenothiazines, one butyrophenone, four antidepressants, two anticonvulsants, an antihistamine, and one barbiturate.

It took about 6 weeks for Mr. B to respond to lithium—before this he seemed to worsen. However, after this initial period, marked improvements were noted. There was a dramatic change in affect, and he was described as being more cheerful and less restless. He became more capable of carrying on conversations and had several productive meetings with his wife. Perhaps most dramatically, his aphasia, which had persisted for 5-6 years, seemed substantially improved. Initially, he could only utter a few agrammatic phrases, but he began to speak in normal sentences, albeit with a stutter and some hesitancy. An aphasia could be detected only by specific testing. In view of these marked improvements in affect and in language, Mr. B was discharged home to live with his wife. He has remained well and abstinent from alcohol for over a year at this writing. He continues to take lithium.

Case 3. Mr. C, a 51-year-old unemployed laborer, had had three psychiatric admissions in 4 years for anxiety and depression. He suffered a "stroke" at age 42, with residual aphasia and blindness in the left eye. Over a period of years right and left carotid endarterectomies were performed, with improvement in the aphasia but a residual blind spot in the left eye. Mr. C began drinking heavily at age 18. He had a history of loss of control, blackouts, hospitalizations for detoxification, and family, job, and legal problems related to alcoholism.

Neurologic examination revealed small, irregular, and sluggishly responsive pupils, facial asymmetry, hypesthesia over the upper and lower extremities, and the previously noted blind spot. CT scan showed a large defect in the distribution of the left middle cerebral artery. Three EEGs were abnormal. Neuropsychological tests revealed moderately impaired functioning and an IQ in the low normal range. The patient was treated with individual psychotherapy, three benzodiazepines, a phenothiazine, three antidepressants, an antihistamine, and two sedative-hypnotics, none of which was effective.

Progress notes before lithium was initiated documented the patient's psychopathology with such remarks as "anxiety attack with shortness of breath," "pulse 116," "preoccupied with suicide," "legs and body in constant motion," and "not able to concentrate or retain." One month after initiation of lithium treatment the patient reported that he felt good and was described as being more relaxed and outgoing. The sleep disturbance diminished and he began to read with good retention. The tremor, quavering voice, and other signs of severe manifest anxiety essentially disappeared. Mr. C was discharged and has been doing well at home for a year. He has not resumed drinking and has no complaints of anxiety or depression. This patient's affective and cognitive responses to lithium treatment were remarkably dramatic, perhaps more so than those of any other patient in the series. This patient also is continuing to take lithium.

CONCLUSIONS

We are not proposing that lithium will be effective in all cases of organic brain syndrome with agitation. Our preliminary impression is that patients with severe and diffuse brain damage may not be good lithium responders. However, neither of our nonresponders reacted adversely to lithium.

It is more difficult to explain why the lithium worked than why it did not. Indeed, Himmelhoch and associates have stated that lithium is contraindicated for manic-depressive patients with low-grade dementias or extrapyramidal disorders.[2] One hypothesis might be that lithium-induced reduction in anxiety and agitation

[2]Himmelhoch JM, Licta SM, May SJ, et al: "Age, Dementia, Dyskinesias, and Lithium Response" (unpublished manuscript).

is responsible for the improvements noted in memory and other cognitive abilities. We feel that this is unlikely for two reasons. First, these patients had been given numerous antianxiety agents in high dosages, with no improvement in their cognitive functioning. Second, the changes in cognition were frequently so dramatic that it would be difficult to attribute them solely to stress reduction.

Beyond these considerations, we are at a loss to explain the positive responses to lithium in the successful cases we have described. However, in view of the surprisingly dramatic results, it seemed imperative to report the findings. A controlled, double-blind experiment could not have been performed, and extraneous unidentified variables may have been at work. Nevertheless, the clinical significance of the findings may be enhanced by the fact that we worked with chronic psychiatric patients with long histories of hospitalization. They had been given a wide variety of antipsychotic and antidepressant medications, singly or in combination, none of which effected the improvement seen with lithium. Furthermore, we have also observed relapses (in these and other cases) when the patients stopped taking lithium or took it irregularly. Thus, in a manner of speaking, many of the patients served as their own controls.

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Cardiac Sinus Node Dysfunction During Lithium Treatment

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Lithium treatment has been associated with a wide range of cardiac complications. The authors report three additional cases of cardiac sinus node dysfunction due to lithium. Two of the three cases were documented to be lithium dependent by the use of Holter monitoring of cardiac rhythm. On the basis of this finding as well as other reports in the literature the authors recommend careful monitoring of the pulse of patients taking lithium as well as ECG monitoring of patients who are over the age of 50 or who have a history of cardiac disease.

LITHIUM TREATMENT has been associated with an increasingly wide range of cardiac complications. T-wave changes on the ECG, myocarditis, and ventricular arrhythmias have all been reported (1-3). Wellens and associates (4) and Wilson and associates (5) each described a case of reversible sinus node dysfunction. The purpose of our paper is to report three additional cases of sinus node dysfunction, two of which were shown by Holter monitoring of cardiac rhythm to be clearly related to lithium use.

METHOD

All of the patients discussed in this paper were treated as inpatients on the metabolic unit of the New York State Psychiatric Institute. The patients met the diagnostic criteria of Feighner and associates (6) for primary affective disorder. Two of the patients met the criteria of Fieve and Dunner (7) for bipolar I illness, and one patient met these criteria for unipolar illness. Patients were studied only after they gave informed consent to all procedures.

In this series sinus node dysfunction was initially diagnosed by routine ECG and then documented to be related to lithium in two cases by use of Holter monitoring. This procedure records 24 consecutive hours of cardiac rhythm, thereby revealing transitory arrhythmias that might not be discovered by a random ECG. The recording instrument itself is small enough that it can be carried around like a handbag, allowing the patients normal activity during the recording period. Exercise ECG was performed with the patients on a treadmill.

RESULTS

Patient 1

Patient 1, a 69-year-old woman with a history of rapid-cycling bipolar I illness, has been followed in our lithium clinic for the past 10 years. Recently she has been maintained on 600 mg of lithium carbonate q.d.

The patient was admitted to the metabolic unit in a euthymic state for a research protocol. She had a history of hypothyroidism but had been euthyroid for many years on 25 μg of triiodothyronine b.i.d. A recently discovered mild elevation of blood pressure was easily controlled by 250 mg of methyldopa t.i.d. Her admission physical examination was normal. Blood chemistries, electrolytes, complete blood count, thyroid functions, and urine analysis were all normal. The lithium level was 1.0 mEq/liter. A routine ECG, read by our attending cardiologist as normal, showed a sinus rhythm with a rate of 83.

Two months after admission the patient was noted to have a pulse of 40; an ECG taken at that time showed a sinus brachycardia of 40 with occasional junctional escape beats. The lithium level was 1.1 mEq/liter, and the patient's only complaint was mild dyspnea on exertion.

Holter monitoring confirmed a sinus brachycardia with long periods of junctional escape rhythm. During exercise the sinus rate appeared to be incapable of accelerating normally and never exceeded a rate of 80 without exit block and subsequent junctional rhythm. No significant ST-T-wave changes occurred. The patient's lithium was then discontinued. The Holter monitor and stress test were repeated after the patient had been off lithium for 18 days. This time the Holter monitor showed a regular sinus rhythm without arrhyth-

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mias, and the stress test showed that the sinus node was able to accelerate to a rate as high as 116/minute without evidence of block.

The patient was then given lithium again; when her level reached 1.0 mEq/liter a third Holter monitoring was recorded. The sinus node dysfunction had returned, as evidenced by long sinus pauses and consequent junctional escapes. The cardiology consultant recommended that if the patient required lithium maintenance a permanent cardiac pacemaker be implanted. The patient recognized that lithium treatment had been essential to her mood stability and agreed to a pacemaker implantation. Two months after the implantation the patient was living at home in a euthymic state and without cardiac symptoms.

Patient 2

Patient 2, a 75-year-old man with a 30-year history of bipolar I illness, has been followed in our lithium clinic for 9 years and has been stable on a maintenance dosage of 1200 mg of lithium q.d. The patient had no clinical history of cardiac disease, but a routine ECG in the clinic showed a normal sinus rhythm with a left bundle-branch block and evidence of an old anterolateral myocardial infarction. His ECG findings had been unchanged for 9 years.

After the patient abruptly went on a salt-restricting diet, he developed ataxia and choreiform movements of the fingers and hands. His lithium level was 1.8 mEq/liter when he was admitted to the metabolic research unit.

Physical examination on admission showed that he was alert, that his blood pressure was 130/80, and that his pulse was 40. There was slurring of speech, gross ataxia when walking, and the choreiform movements previously noted. Blood chemistries, electrolytes, complete blood count, and urine analysis were all normal. An ECG showed a sinus brachycardia of 35, with periods of a junctional escape rhythm. There was a left bundle-branch block configuration and T-wave inversions in leads II, III, AVF, and V4-V6.

The patient was transferred to the cardiac intensive care unit. Atropine (1 mg I.M.) was given without effect. Following 18 hours of hydration the patient's lithium level had fallen to 1.5 mEq/liter and his heart rhythm then stabilized at normal sinus, with a rate of 60. The T-wave inversions in leads V4-V6 persisted. Subsequent ECGs all showed normal sinus rhythm, with rates of 60–80/minute.

The patient recovered uneventfully from this episode of lithium toxicity but refused Holter monitoring. Lithium maintenance was resumed (600–900 mg q.d.), and he was discharged from the hospital with instructions to take his pulse twice a day. Through 2 years of follow-up he had only one episode of transitory sinus brachycardia.

Patient 3

Patient 3, a 53-year-old woman, had a 15-year history of recurrent unipolar depressions. She was admitted to the metabolic research unit for treatment of an acute depressive episode. She had no history of cardiac disease.

Admission physical examination was normal. Blood chemistries, electrolytes, complete blood count, and urinalysis were all normal. ECG was read as normal and showed a normal sinus rhythm, with a rate of 72.

The patient was treated with lithium and tricyclic antidepressants for 3 months without medical complications but also without a satisfactory clinical response. The medications were stopped and the patient was given a course of ECT; moderate improvement followed. Lithium treatment was resumed. After 4 weeks an ECG showed sinus node dysfunction, evidenced by an irregular sinus rhythm with pauses up to 1.6 seconds followed by junctional escape beats. The patient's lithium level was .71 mEq/liter.

Lithium was discontinued and the patient was kept off medication for 16 days. A Holter monitor recording showed a normal sinus rhythm, with a mean rate of 72/minute. Lithium was resumed and stabilized at a level of 1.0 mEq/liter. A repeat Holter monitoring showed that the sinus node dysfunction had returned. The patient was taken off lithium; 2 months later there was no ECG evidence of sinus node dysfunction or other cardiac symptoms.

DISCUSSION

It is known that lithium enters the cardiac cell, displacing cations and creating intracellular metabolic changes, including hypokalemia. These electrolyte shifts are thought to be the physiological processes underlying the T-wave changes seen on the ECG during lithium treatment. However, the mechanism by which lithium influences cardiac conduction is still obscure. Riciutti and associates (8) showed that when doses of lithium as high as 300 mg/kg of body weight were given intravenously to animals, ventricular and atrial arrhythmias as well as prolonged AV node and intraventricular conduction times occurred. Paradoxically, a review of animal studies by Tilkian and associates (9) demonstrated a possible antiarrhythmic effect of lithium. The pathophysiology of spontaneously occurring sinus node dysfunction is not yet established. The pacing cells of the sinus node are in part dependent on calcium ion flux for their generative capacity, and it is possible that lithium-induced sinus node dysfunction may be related to alterations in calcium ion flux.

Tilkian and associates (9) referred to sinus node dysfunction and ventricular arrhythmias as complications that occur rarely in lithium-treated patients. However, in a report on the results of exercise testing and prolonged ECG recordings in 12 patients on and off lithium, the same group (10) observed that 3 patients had increased ventricular ectopy on lithium. It is likely that cardiac arrhythmias are a more frequent complication of lithium treatment than originally suspected, especially in view of the fact that in one earlier case and in

all of the patients reported in this study the sinus node dysfunction was asymptomatic. Furthermore, as the three cases reported here indicate, the complication of sinus node dysfunction can occur during any of the stages of lithium treatment, e.g., the initial phase, after many years of uncomplicated treatment, or during an episode of toxicity. Therefore, we recommend that the pulse of patients on lithium be regularly recorded and that semi-annual ECGs with long rhythm strips be taken on all lithium-treated patients who are over the age of 50 or who have a history of cardiac disease.

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Variable Attenuation of Amphetamine Effects by Lithium

BY BURTON ANGRIST, M.D., AND SAMUEL GERSHON, M.D.

In an open study of 8 subjects, approximately half showed some attenuation of CNS stimulant effects of amphetamine after pretreatment with lithium. Two showed specific blockade of euphoria, with persistence of some CNS stimulant effects. In 3 subjects lithium did not appear to affect the response to amphetamine. Lithium caused significant attenuation of the amphetamine-induced increase in systolic blood pressure for the group as a whole.

TWO REPORTS to date have indicated that in some cases the effects of amphetamine can be diminished by chronic lithium treatment. Flemenbaum (1) retrospectively documented 3 cases in which this occurred: 2 amphetamine abusers spontaneously gave up the drug because of loss of "high" feelings after starting lithium treatment for affective disorder; the third took amphetamine for dieting and noticed a blockade of both subjective affects and anorexia after receiving lithium. Van Kammen and Murphy (2) reported a double-blind placebo-controlled study in which the effects of 30 mg of *d*- or *l*-amphetamine were observed in 9 patients with moderate to severe depression before and after a minimum of 10 days' lithium treatment. They found a 60% attenuation of activation and euphoria induced by the *d* isomer and almost complete abolition of the effects of *l*-amphetamine after lithium administration. We conducted the open study described here to assess the effect of lithium on the response to amphetamine of subjects without overt affective disorder under conditions of direct observation.

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METHOD

Our proposed project was explained to patients admitted to Bellevue Psychiatric Hospital for a variety of reasons who had some familiarity with amphetamine effects. Eight patients who were willing to participate and who provided informed consent were transferred to a research ward, treated as indicated by their individual condition, and then stabilized off all medica-

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tions for 6–24 days before participating in the study. D-Amphetamine (1/2 mg/kg) was given orally twice—before and after 6–15 days' treatment with lithium. The amphetamine response was assessed by the following methods: 1) taking pulse and blood pressure after the patient had been lying quietly for 5 minutes before amphetamine administration and at hourly intervals after amphetamine was given, 2) unstructured clinical notes before amphetamine administration and hourly after, 3) filling out the amphetamine subscale of the Addiction Research Center Inventory (ARCI)—38 true-false items that have been shown to discriminate amphetamine from placebo at the .05 level of significance (3)—before and 3 hours after drug administration, and 4) giving the 100-millimeter line, an index of self-assessment of euphoria, before and 3 hours after amphetamine was given (in 2 cases the ARCI and the 100-mm line were unfortunately omitted at baseline; in these 2 cases raw scores rather than pre-post drug change scores are reported).

These parameters of the amphetamine response "on" and "off" lithium were compared. The study was not blind, and neither lithium placebo nor amphetamine placebo was used.

RESULTS

The parameters of response described above to a fixed dose of 0.5 mg/kg *d*-amphetamine are indicated in table 1, which shows the responses of the patients before and after lithium administration.

Patient 1 was a 19-year-old man with a diagnosis of antisocial personality who entered the hospital because of a mild amphetamine psychosis. The patient had taken 100 mg of *d*-amphetamine "to cause a heart attack." He received lithium for 15 days (maximum dose, 3000 mg/day). At the time of testing, his lithium level was 1.24 mEq/liter. According to the clinical notes, this patient was more talkative, evasive, and histrionic while off lithium. On lithium, he was "in love with mankind" and bought potato chips for the other patients. He said he had felt "crappy" by comparison the last time he got amphetamine: "Wow! I love you for this." The global clinical impression was that he was definitely more euphoric on lithium.

Patient 2 was a 24-year-old man diagnosed as having acute reactive depression and a passive dependent personality. He received lithium for 12 days (maximum dose, 2100 mg/day). His lithium level was 1.59 mEq/liter at the time of amphetamine administration and 1.24 mEq/liter 3 hours after that. Off lithium he was markedly more animated and talkative and was optimistic, confident, and energetic. He had definite euphoria and some palpitations, clenching of teeth, and "tingling" of scalp. On lithium he was irritable and "edgy" and had rapid speech. He preferred to remain withdrawn and felt "physically hyped up, but it's like my engine is revving. The positive feeling and confidence isn't there." He was somewhat self-dramatizing and provocative but had no palpitations or clenching of teeth. The global clinical impression was that euphoria was blocked but dysphoric stimulation persisted on lithium.

Patient 3 was a 53-year-old man with a history of habitual excessive alcohol use who was admitted for detoxification. He received lithium for 6 days (maximum dose, 3000 mg/day). His lithium level was 1.16 mEq/liter at the time of testing. Off lithium he was talkative and euphoric. He noted philosophical concerns: "It's like a religious experience—the exact opposite of alienation." He spoke of John Donne, theories of love, and "the nature of truth." On lithium he felt a "muted effect." He had no "push" toward philosophizing as he had before. He continued to read with pleasure, and his concentration was increased. He appeared relaxed and euphoric. The global clinical impression was that lithium attenuated psychological and cardiovascular amphetamine effects.

Patient 4 was a 35-year-old man with a diagnosis of episodic excessive alcohol abuse who was admitted for detoxification. He received lithium for 6 days. This was discontinued for 24 hours because of possible toxicity, and then lithium was given for another 6 days before testing (maximum dose, 3000 mg/day). His lithium level was 1.0 mEq/liter. Off lithium he appeared calm but more talkative. He said he felt "serious" and "at peace with the world." At times he was slightly tangential. On lithium he stated he felt "calm but energetic." He was talkative, intense, and introspective. At times he had a wide-eyed "stare." The global clinical impression was that there was no clear difference in the effects of amphetamine after lithium administration.

Patient 5 was a 19-year-old woman two took amphetamine and cocaine intravenously and was admitted because of an amphetamine psychosis. The only diagnosis assigned her was drug dependence, specifically dependence on CNS stimulants. She was given lithium for 6 days (maximum dose, 2000 mg/day). Her serum lithium level was 1.14 mEq/liter. Off lithium she had increased libido, increase in "tender" feelings, and emotional lability. She said she felt "terrific." She enjoyed talking with other patients and showed pressure of speech, some tangentiality, and circumstantiality. On lithium, she was calmer, less intense, and in better control: "I can keep my mind on one thing." There was no objective circumstantiality or tangentiality and no increase in libido: "I'd say you gave me half the dose as last time." She felt this dose was "perfect" and wouldn't take more. The global clinical impression was that the effects of amphetamine were attenuated after lithium administration but that there was no specific blockage of euphoria.

Patient 6 was a 49-year-old man diagnosed as having antisocial personality with habitual excessive drinking. He was admitted after causing a disturbance while drunk. He received lithium for 7 days (maximum dose, 2400 mg/day); his serum level was 1.11 mEq/liter at the time of testing. Off lithium, he was more talkative; his

TABLE 1
Response of 8 Patients to Amphetamine[a] Before and After Lithium Administration

Item	Off Lithium			On Lithium		
	Before Amphetamine	After Amphetamine	Change	Before Amphetamine	After Amphetamine	Change
Patient 1						
ARCI score[b]	22	18	− 4	18	29	+11
100-mm line score[c]	29	39	+10	43	56	+13
Systolic blood pressure	112	160	+48	122	160	+38
Diastolic blood pressure	74	110	+36	76	86	+10
Pulse	76	64	−12	72	92	+20
Patient 2						
ARCI score	21	24	+ 3	21	13	− 8
100-mm line score	62	82	+20	40	30	−10
Systolic blood pressure	128	156	+28	136	152	+16
Diastolic blood pressure	64	84	+20	72	74	+ 2
Pulse	88	80	− 8	96	100	+ 4
Patient 3						
ARCI score	21	31	+10	18	31	+13
100-mm line score	67	94	+27	60	86	+26
Systolic blood pressure	102	170	+68	110	136	+26
Diastolic blood pressure	76	98	+22	76	88	+12
Pulse	64	64	0	64	80	+16
Patient 4						
ARCI score	23	18	− 5	13	16	+ 3
100-mm line score	64	82	+18	55	70	+15
Systolic blood pressure	124	146	+22	118	136	+18
Diastolic blood pressure	90	102	+12	82	92	+10
Pulse	96	108	+12	84	104	+20
Patient 5						
ARCI score	24	31	+ 7	12	10	− 2
100-mm line score	48	78	+30	40	44	+ 4
Systolic blood pressure	110	120	+10	116	118	+ 2
Diastolic blood pressure	72	70	− 2	70	64	− 6
Pulse	92	104	+12	68	64	− 4
Patient 6						
ARCI score	25	35	+10	16	36	+20
100-mm line score	62	80	+18	60	89	+29
Systolic blood pressure	110	130	+20	110	132	+22
Diastolic blood pressure	82	82	0	74	90	+16
Pulse	84	92	+ 8	80	92	+12
Patient 7						
ARCI raw score	—	19		—	18	
100-mm line raw score	—	35		—	49	
Systolic blood pressure	120	148	+28	120	142	+22
Diastolic blood pressure	86	88	+ 2	84	94	+10
Pulse	72	76	+ 4	92	104	+12
Patient 8						
ARCI raw score	—	37		—	17	
100-mm line raw score	—	85		—	10	
Systolic blood pressure	114	162	+48	112	140	+28
Diastolic blood pressure	72	96	+24	68	86	+18
Pulse	72	62	−10	84	68	−16

[a]A fixed dose of 0.5 mg/kg of *d*-amphetamine was administered.
[b]The amphetamine subscale of the Addiction Research Center Inventory (ARCI) contains 38 true-false items that have been shown to discriminate amphetamines from placebo.
[c]The 100-mm line is an index of self-assessment of euphoria.

mood was serious and earnest: "My problems are there but I don't worry about them." He felt more "sentimental" and "creative" and wrote a poem about New York City. On lithium, he was again more talkative: "It takes the gloom away. I know the problems are there but they don't bother me." He felt he had increased "fertility power" and an increased sense of smell (he "smelled women"). He said he had "no worries." The clinical global impression was that there was no clear change in amphetamine effects after lithium administration.

Patient 7 was a 29-year-old man who was a heavy user of intravenous cocaine. He was admitted because of suicidal ideation and diagnosed as having a passive-aggressive personality with cocaine dependence. He received lithium for 7 days (maximum dose, 1800 mg/day) and had a serum level of 0.7 mEq/liter at the time of testing. Off lithium he felt he had an increased sense

TABLE 2
Mean Behavioral Scores for 6 Patients and Mean Cardiovascular Measures of 8 Patients

	Off Lithium						On Lithium					
	Before Amphetamine		After Amphetamine[a]		Change		Before Amphetamine		After Amphetamine		Change	
Measure	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
ARCI score[b]	22.6	1.6	26.2	7.3	3.8	6.43	16.3	3.4	22.5	10.8	6.2	10.4
100-mm line score[c]	55.3	14.5	75.8	18.9[d]	20.5	7.1	49.6	9.7	62.5	23.5	12.8	14.4
Systolic blood pressure	115	8.5	149	16.9[d]	33	17.1	118	8.6	139.5	12.7[d]	21.5	10.4[e]
Diastolic blood pressure	77	8.5	91.3	12.7[f]	15	12.6	75.3	5.5	84.3	10.2[d]	9.3	7.8
Pulse	80.5	11.2	88	15.7	8	9.8	80	11.3	88	15.7	8	12.7

[a]A fixed dose of 0.5 mg/kg of *d*-amphetamine was administered.
[b]The amphetamine subscale of the Addiction Research Center Inventory (ARCI) contains 38 true-false items that have been shown to discriminate amphetamine from placebo.
[c]The 100-mm line is an index of self-assessment of euphoria.
[d]Significantly ($p<.01$) elevated over baseline.
[e]Change score significantly ($p<.05$) smaller off lithium.
[f]Significantly ($p<.05$) elevated over baseline.

of smell. He felt "benevolent" and was emotionally labile: "I see other patients talking and laughing and I think of not knowing what friends or family are." On lithium he felt his sense of smell was again increased. He felt more "empathy" and was again emotionally labile: "I feel I want to grab your arm and say 'thank you.' " Ideas seemed "more important." The global clinical impression was that there was no clear change in amphetamine effects after lithium administration.

Patient 8 was a 32-year-old man diagnosed as showing an inadequate personality with alcohol addiction. He received lithium for 8 days (maximum dose, 1750 mg/day) and had a serum level of 1.0 mEq/liter at the time of testing. Off lithium he said he felt "nice," "lively," and "not depressed." He snapped his fingers while talking. He was animated, and his reaction time was decreased. On lithium, he felt "nervous," "jittery inside," and "jumpy": "not at all like last time." His animation was not increased, and his reaction time was not decreased. The global clinical impression was that euphoria was blocked; there was some attenuation of stimulant effects.

Statistical analysis of pooled data (see table 2) indicated that the amphetamine-induced increase in systolic blood pressure was significantly less after lithium administration ($t=2.93$, $p<.05$, two-tailed). Increases in diastolic blood pressure and changes in pulse rate were not significantly different after lithium, nor were change scores on the ARCI and the 100-mm line (for the 6 patients for whom these data were available). Scores on the 100-mm line were significantly elevated after amphetamine alone but not significantly raised by amphetamine after lithium. However, the change scores from baseline were not significantly different before and after lithium.

DISCUSSION

Lithium caused significant attenuation of the amphetamine-induced increase in systolic blood pressure. Since it is generally assumed that the pressor effects of amphetamine are mediated by norepinephrine, this finding is consistent with a study by Fann and associates (4) in which lithium was found to attenuate the pressor response to norepinephrine itself in manic patients.

Lithium was also found to affect the subjective and behavioral effects of amphetamine variably in different subjects. This variability and the small number of subjects in this study in all likelihood account for the failure of the ARCI and 100-mm line to show significant changes (the stimulant subscale of the ARCI, in particular, has been shown to discriminate amphetamine effects reliably [3], but in a large number of subjects). Our individual subjects' response patterns can be summarized as follows: 3 subjects showed no clear difference in amphetamine response after lithium, 2 showed attenuation of stimulant effects after lithium, 2 showed a blockade of euphoria but persistence of dysphoric stimulation, and 1 was more euphoric after lithium and amphetamine than after amphetamine alone.

Although it may seem contradictory, the response of this last subject might also represent attenuation. Several investigators (5–7) have noted that the euphoria occurring after low doses of amphetamine or cocaine is replaced by dysphoric feelings after the dose is further increased. Thus a decreased responsivity on the basis of lithium pretreatment could lead to a euphoric response to a dose that was "too high" to cause this effect originally. If this subject's response is accepted as a type of attenuation, then 3 of the subjects could be considered to show this response pattern, while 3 showed no change in amphetamine response on the basis of lithium pretreatment and 2 experienced a selective blockade of euphoria. The patients who showed either attenuation of stimulant effects or blockade of euphoria received lithium for a mean of 8.25 days and had a mean plasma level of 1.22 mEq/liter. The patients whose amphetamine response was unaffected by lithium received lithium for 8.66 days and had a mean plasma level of 0.94 mEq/liter. The patient whose response was open to interpretation (ei-

ther increased euphoria or attenuation) received lithium for 15 days and showed a serum level of 1.24 mEq/liter. It seems unlikely that those small differences can account for the variable effects on amphetamine response observed. It is possible, however, that more consistent findings might have resulted from a longer lithium pretreatment period.

These findings are consistent with those of van Kammen and Murphy (2) in their controlled study of depressive patients. Although they stressed the highly significant attenuation of euphoria and activation for the group as a whole, their subjects' individual response patterns showed similar variability (3 of their 9 subjects showed no diminution of activation and 5 showed only slight or no decrease in euphoria). Thus the effects of lithium on the amphetamine response appear to be similar in both populations studied.

These findings are also consistent with preclinical studies in which attenuation of amphetamine effects by lithium have been noted. These include decreased effects on intracranial self-stimulation (8) and locomotor activity (9).

The mechanisms underlying this partial and inconsistent "antagonism" are unknown. Neurophysiological studies by Buchsbaum and associates (10) have shown that amphetamine-induced changes in visual average evoked responses correlate with the degree of euphoria and activation seen after amphetamine and that these changes are also attenuated by lithium, suggesting a possible common mechanism. However, both amphetamine and lithium have effects on many brain systems (11, 12) and therefore could interact in many ways. Aminergic neurotransmitter systems have been studied to some degree. Friedman and Gershon (13) have shown that treatment with lithium for 2 weeks causes a decrease in dopamine synthesis in rat brain.

Berggren and associates (14) have demonstrated that the decrease in amphetamine-induced locomotor stimulation caused by acute lithium pretreatment could be reversed by a small dose of L-dopa, which had no effect on locomotor activity per se, further suggesting a catecholaminergic mechanism, possibly involving decreased synthesis. According to a personal communication, the same group has also demonstrated decreased dopa formation (after inhibition of decarboxylase) after acute lithium, suggesting a similar mechanism. However, lithium also has complex time-dependent effects on serotonin biosynthesis, some parameters of which are directly antagonized by acute cocaine administration (15). Thus, effects on aminergic neurotransmitter systems may underlie this drug interaction, but it is difficult to specifically implicate catecholaminergic or serotonergic mechanisms. Indeed, effects other than those on aminergic neurotransmitters might also mediate this interaction.

Mandell and Knapp (15), van Kammen and Murphy (2), and Flemenbaum (1) have all suggested that lithium might prove clinically useful in the treatment of stimulant-dependent patients. The results of this study suggest that this might be the case in patients in whom lithium attenuates the effects of stimulants (perhaps 50% based on this small sample). Such patients would of course have to be highly motivated to discontinue stimulant use in order to ensure reliable lithium intake; many stimulant users are not motivated in this way. Nonetheless, lithium might prove a useful adjunct, particularly in the early phases of discontinuation when craving is most severe (i.e., the first few months). Since no specific treatment for stimulant dependence exists, lithium may be of some potential benefit to this small patient group.

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Lithium Plus Reserpine in Refractory Manic Patients

BY NORMAN M. BACHER, M.D., AND HARVEY A. LEWIS, M.D.

The authors report on six refractory manic or schizo-affective manic male outpatients who failed to respond to lithium combined with a neuroleptic. A beneficial response was noted in most patients when reserpine was substituted for their currently prescribed standard neuroleptic; in some cases lithium dosage could then be reduced. No major side effects were encountered. The use of reserpine may offer an alternative mode of therapy for manic patients who cannot tolerate large doses of lithium or do not completely respond to lithium either alone or in combination with a standard neuroleptic.

WHEN THE USE of reserpine in American psychiatry markedly diminished after 1960 (despite its usefulness in some psychotic conditions), postsynaptic dopamine-blocking neuroleptics and antidepressants became generally accepted, although lithium had not yet been introduced in this country for the treatment of manic-depressive illness. In the 1960s investigators reported on the use of antiserotonin agents in mania (1). Reserpine, which has antiserotonin properties, has been reported to be of value in manic, catatonic, and excited states (2). Although lithium has been tried alone and in combination with various neuroleptics in many refractory, cyclic, and affective states, we have found no reports of clinical studies combining lithium with reserpine (3).

In our Veterans Administration outpatient clinic we have encountered a number of agitated, excited outpatients diagnosed as manic-depressive or schizo-affective manic who do not respond to therapeutic levels of lithium alone or to lithium plus neuroleptic. This report describes six such patients; in a clinical trial we added reserpine to the patients' lithium dosage and reduced or discontinued neuroleptic after discussion with the patients concerning this mode of treatment. These patients were considered refractory (i.e., unmanageable) at the time that reserpine was added.

We told each prospective patient of reserpine's use in psychiatry and its gradual disuse after the newer major tranquilizers became established as more effective. We explained that we had used reserpine in disturbed and excited patients, that it has been approved by the Food and Drug Administration as a relatively safe psychiatric drug, and that it is still widely used in the medical treatment of hypertension. We told them that we felt reserpine plus lithium might be more effective than their current tranquilizer plus lithium, since they had not done well on that combination. Each patient was cautioned that this new combination of reserpine plus lithium had not, to our knowledge, been tried previously. If they agreed, we followed them closely, only adding reserpine gradually and watching for side effects. The patients were told to stop taking reserpine and contact us if any undesirable side effects occurred. We discussed major side effects and known contraindications of reserpine with each patient.

METHOD

All six outpatients showed the following characteristics. Diagnosis was manic-depressive, manic, or excited schizo-affective. The behavioral component of the illness was at least as marked as the thinking disorder, and the patients tended toward denial, elation, overstimulation, hyperactivity, argumentativeness, and pressure of speech with tangential, circumstantial, and irrelevant thought processes. Delusional qualities of thinking, both grandiose and paranoid, were prominent, although the former seemed more pronounced. These delusional ideas were reflected in the behavioral manifestations of the illness and persisted, although they were diminished in patients who improved, throughout the course of treatment. All patients had sexual identification problems and latent to overt acting out character defenses. A tendency toward social pathology was prominent. On a pragmatic level these patients at times were loud and boisterous, attended clinic frequently but irregularly, tended to disrupt clinic routines, and became easily involved in verbal, hostile, and sadistic exchanges with staff and other patients.

Four of the six outpatients had had several recent psychiatric hospitalizations during which they had ap-

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parently responded adequately to therapeutic levels of lithium plus neuroleptic. However, as outpatients they decompensated rapidly despite apparent continuation of the same levels of medication. Their manic symptoms may have been aggravated by alcohol or drugs or possibly by noncompliance. The other two manic outpatients, one in day care, could not be stabilized despite frequent contacts with staff and therapeutic levels of lithium plus a neuroleptic.

All six patients at times complained of their medication, especially the neuroleptic. Some complained of side effects of the lithium, such as gastrointestinal symptoms, tremors, and urinary frequency. One patient developed tardive dyskinesia while taking a neuroleptic. In most of these patients lithium levels were obtained frequently and found to be in a therapeutic range even during delusional thinking overactivity.

The issue of noncompliance is a problem with these patients, and we found that the oral lithium dose cannot accurately be correlated with blood levels from patient to patient. One patient rarely came to the laboratory to have blood drawn. Overall, the lithium-neuroleptic combination was a failure in all six cases. The literature indicates that a substantial number of patients taking lithium fail to improve or relapse and become chronic (4, 5).

RESULTS

Reserpine in dose ranges of 0.25 to 1 mg/day (up to 8 mg/day for 1 patient) was added to the lithium dosage for a short time while the neuroleptic was gradually eliminated. Response over a period of one to two weeks was marked, appeared dose-related, and affected the disruptive behavioral component of the illness more than the thinking process. The patients became calmer. Once reserpine was begun, lithium dosage in most of the patients was adjusted downward; in two patients there was remission with lithium levels below the currently accepted therapeutic range, although spontaneous remission could not be ruled out. If some dysphoria developed, reserpine dosage was adjusted or given intermittently or at night. However, there are indications that lithium may be protective against reserpine dysphoria. Experimental work shows that lithium prevents reserpine depletion of neurotransmitter amines from the presynaptic ganglion (6).

We have followed most of these six refractory patients as outpatients periodically over several years. Improvement occurred in all patients when reserpine was given with the lithium; in most patients, greater compliance and adjustment resulted. One patient's behavior improved slightly on reserpine-lithium, but he continued to experience severe thought disorder, which suggested a diagnosis of chronic schizophrenia.

Although these are observational data, two of these six refractory patients have become tractable and cooperative compared to their prereserpine state and are no longer a management problem. Since these patients are doing well, we did not selectively withdraw one or the other medication to differentiate the effect. In the two improved patients we reduced lithium dosage when lithium was combined with reserpine, and remission was maintained. This is important because lithium and neuroleptics have been increasingly reported as having serious long-term side effects.

CASE REPORTS

Case 1. This 32-year-old married unemployed male outpatient remained hyperactive, talkative, elated, loud, overstimulated, disruptive, and grandiose during frequent but irregular visits to the clinic for over 1 year. He felt he was receiving messages from the planets and stars. Although in a recurrent manic state, he had not been hospitalized. He responded poorly to various combinations of neuroleptics alone or with lithium in therapeutic dosage. Compliance and illicit drug use were unknown factors. We recorded few serum lithium levels. However, in recent months with medication changed to lithium and reserpine, this outpatient began to improve dramatically, both affectively and behaviorally. He now attends the clinic regularly, is on lithium, 900 mg/day, and reserpine, 0.75 mg/day. He remains calm and clear and intends to train for an occupation. We cannot rule out a spontaneous remission, but his improvement coincided with a lithium-reserpine combination.

Case 2. This 48-year-old married man had a stable work and marital history. However, over the previous 2 years, he has had several psychiatric hospitalizations and was diagnosed as manic-depressive each time. After some months, he would stabilize in the hospital on lithium and a neuroleptic, but as an outpatient he would quickly become hyperactive, argumentative, and elated, demonstrate some paranoid ideation, and be difficult to manage. Stresses on him as an outpatient may have resulted in an increase in alcohol consumption and noncompliance regarding lithium. After the most recent discharge, he complained of the side effects of both lithium and the neuroleptic. We stopped the neuroleptic and added reserpine up to 1 mg/day with gradual reduction in the lithium dosage to 900 mg/day. Repeated lithium levels have been at 0.3–0.4 mEq/liter. Over several months, the patient became compliant, worked on marital conflicts, and returned to his job. He remained stable for about six months but began to drink after experiencing new work stress. His mood became elevated; he discontinued work and was again seen frequently in the clinic. His reserpine dosage was adjusted to 0.75 mg/day, and he continued on lithium, 900 mg/day. A recent serum lithium level was 0.3 mEq/liter. The fact that he did well on small doses of lithium and reserpine for some 6 to 8 months suggests synergistic action and enabled us to reduce the lithium dosage. After recently becoming more disturbed, unrealistic, and noncompliant, he was rehospitalized.

Case 3. This 32-year-old single man has had repeated psychiatric hospitalizations over the past 10 years. He has received large amounts of various neuroleptics plus lithium. In recent years, his diagnosis has been more often manic or schizoaffective, with a depressive component. When seen as an outpatient, he has been loud, demanding, overstimulated, grandiose, and delusional. He insisted he was the Messiah

and could cure all mental illness and evil and prevent death. His few stable periods, during which he was out of the hospital, lasted only a few months. Some months later, he was again discharged on lithium and neuroleptic medication. His blood levels (1.0 mEq/liter) showed lithium compliance; however, he remained elated, hyperactive, grandiose, severely psychotic, and a management problem despite frequent contacts with the clinic. He complained of dysphoric effects from the neuroleptic. Reserpine was added to the 1200 mg/day of lithium, and neuroleptic medication was gradually discontinued. He was less disturbed for a time on reserpine and lithium. When reserpine was increased to 6.0–8.0 mg/day and lithium remained at therapeutic levels, his overactivity was markedly reduced within one week. He became quiet and seemed less certain of his grandiose delusions. However, because of complaints of sluggishness and dysphoria, the reserpine was gradually reduced and the patient soon became more disturbed and was rehospitalized. Large doses of reserpine combined with lithium were helpful and did not result in depression.

Case 4. This 28-year-old recently separated unemployed man came to our clinic in crisis demonstrating depression and delusions about astrology and ESP. On neuroleptic combined with antidepressant, the patient became hyperactive and required a first psychiatric hospitalization. He was diagnosed as manic-depressive and discharged on lithium and chlorpromazine. A few weeks later he returned to the clinic stating he had reduced his medicine significantly, preferring euphoric feelings. He disliked medication, especially chlorpromazine. We encouraged the use of at least some lithium plus small amounts of reserpine. The patient later reported the use of lithium over several months and reserpine on an as-needed basis. He said reserpine quieted him and lithium helped the feelings in his head. The patient has since returned to work and resumed group therapy. His current lithium dosage is 300–600 mg/day in addition to 0.25–0.5 mg/day of reserpine. It appears the low dosage of this combination helps to maintain his remission. The lithium and reserpine seem to have different antipsychotic properties in addition to being synergistic. He continues to maintain his remission.

Case 5. This 56-year-old divorced man with a long history of recurrent manic-depressive episodes had a manic attack while in our day center. He was placed on therapeutic levels of lithium plus a neuroleptic to control his hyperactivity and elation. When parkinsonism and tardive dyskinesia developed the neuroleptic was discontinued and reserpine up to 1.0 mg/day was added to the lithium. The lithium level remained in a therapeutic range. The patient soon became rational and stable, and his dyskinesia and parkinsonism improved slightly. For a few months on a regimen of lithium, 900 mg/day, and reserpine, 0.5 mg/day, he made a better dependent adjustment in the day center than at any time in the prior 10 years. Reserpine was used intermittently and then gradually withdrawn in order to control dysphoric symptoms. His lithium levels were 0.4 mEq/liter. Recently, on no reserpine and with the lithium level at 0.6 mEq/liter, the patient had another mood swing that required a brief hospitalization despite the use of a standard neuroleptic.

Case 6. This 30-year-old single male graduate student has been seen periodically over the years. He has often been emotionally labile and affected by alcohol. His diagnosis is recurrent mania, and he seems to do well on lithium when hospitalized and poorly on lithium as an outpatient. Under stress in the community, he discontinues medication, becomes delusional (feeling he is involved in an undercover plot involving murder), loses control, and is hospitalized frequently. A few months ago he returned to the outpatient clinic in remission after another brief hospitalization. He was on lithium, 1200 mg/day, with a therapeutic blood level but had discontinued chlorpromazine. He was persuaded to substitute small doses of reserpine (up to 0.75 mg/day) for the chlorpromazine and continue on lithium. He remained stable, returned to school, and tolerated the lithium well with small amounts of reserpine for a short time. However, despite therapeutic blood levels of lithium at a dosage of 1200 mg/day, he again demonstrated paranoid ideation and was manipulative. Because we could not be sure of compliance, he was rehospitalized. When he returned to the clinic, we learned that he had taken the reserpine for only a few days.

DISCUSSION

These cases demonstrate that reserpine combined with lithium may be more effective than lithium with a neuroleptic or lithium alone in certain intractable affective disorders and may provide an alternative when a patient responds poorly to lithium with a neuroleptic.

Because lithium affects brain neurotransmitter amines and is homeostatic on mood and reserpine depletes brain neurotransmitter amines presynaptically and has a tendency to produce sedation and release depression-like states, they may be synergistic in mania. Further, some reports note a relationship between mania and hypertension (7), and reserpine is a well-known antihypertensive. Recently, articles have appeared indicating that catatonic, paranoid, and affective conditions may belong to an affective spectrum (8, 9) and that lithium should be tried in refractory affective and even refractory schizophrenic states (10). In view of our observations, we suggest that reserpine can be used effectively in combination with lithium in treating certain affective conditions refractory to lithium or lithium combined with a neuroleptic.

We have noted few side effects from the reserpine-lithium combination. All our patients were normotensive, and reserpine does not usually lower blood pressures below normotensive levels. Dysphoric symptoms, such as dizziness, weakness, tiredness, and fatigue were easily reduced by adjustment or intermittent use of reserpine. A switch to depression did not occur, possibly because lithium's antidepressant effects protected against depression. We have not as yet tried this combination in similar patients, but we feel further clinical trials and controlled studies are indicated.

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BRIEF COMMUNICATIONS

DSM-III Field Trials: I. Initial Interrater Diagnostic Reliability

BY ROBERT L. SPITZER, M.D., JANET B.W. FORMAN, M.S.W., AND JOHN NEE, PH.D.

The interrater agreement for major diagnostic categories in studies using DSM-I *and* DSM-II *was usually only fair or poor. In phase one of the* DSM-III *field trials the overall kappa coefficient of agreement for axis I diagnoses of 281 adult patients was .78 for joint interviews and .66 for diagnoses made after separate interviews; for axis II—personality disorders and specific developmental disorders—the coefficients of agreement were .61 and .54. The interrater reliability of* DSM-III *is, in general, higher than that previously achieved and may be due to changes in the classification itself, the separation of axis I from axis II conditions, the systematic description of the various disorders, and the inclusion of diagnostic criteria.*

PREVIOUS STUDIES of the reliability of psychiatric diagnosis using the first and second editions of the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-I* and *DSM-II*) have indicated that for the major categories, such as schizophrenia and affective disorder, the interrater agreement was usually only fair or poor (1). An important step in the development of the third edition of the *Diagnostic and Statistical Manual* (*DSM-III*) has been a series of field trials using draft versions of the manual. One of the most important purposes of the major field trial, sponsored by the Division of Biometry and Epidemiology of the National Institute of Mental Health, has been to determine interrater diagnostic reliability. In this paper we present the interrater diagnostic reliability for adult patients from phase one of this field trial, using the Jan. 15, 1978, draft of *DSM-III*.

METHOD

Clinicians were invited to participate in the field trial through notices appearing in *Psychiatric News* and other mental health publications. All clinicians who agreed to complete the required work were accepted as participants, either as private practitioners or as groups of clinicians working within facilities. The clinicians were from all parts of the country, from Maine to Hawaii, and worked in both rural and urban settings. Over 80% identified their main professional activity as patient evaluation or care. The rest were distributed among administration (7%), teaching (7%), and research (3%). Only those clinicians who joined in groups participated in the reliability study.

Each clinician in this study was expected to participate with another clinician in at least two reliability evaluations. These two evaluations were to be done after each clinician had already used the *DSM-III* draft in evaluating at least 15 patients selected from his or her patient population as either consecutive admissions or "catch-as-catch-can" (an approximation of the ideal of random sampling). The reliability interviews, with only a few exceptions, were initial diagnostic evaluations before the beginning of treatment. Detailed instructions were given to the clinicians to avoid possible biases. For example, the clinicians were cautioned not to choose cases specifically because they presented no differential diagnostic problems and not to discuss a case before each clinician's independent completion of the diagnostic forms. Sim-

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TABLE 1
Kappa Coefficients of Agreement for *DSM-III* Diagnostic Classes for 281 Adults

Diagnostic Class	Percent of Total[a]	Interview Method	
		Joint (N=150)	Test-Retest (N=131)
Disorders of infancy, childhood, or adolescence	5.6	.66	.81
Mental retardation	2.1	.66	.85
Disorders characteristic of late adolescence	1.4	1.00	.66
Eating disorders	2.1	−0.01	.85
Organic mental disorders	12.9	.74	.83
Senile and presenile dementias	3.1	1.00	.74
Substance-induced organic brain syndromes	7.7	.48	.74
Organic brain syndrome with unknown etiology	4.5	.56	.72
Substance use disorders	22.0	.90	.74
Schizophrenic disorders	12.9	.82	.82
Paranoid disorders	0.7	1.00	1.00
Schizoaffective disorders	4.5	.56	.53
Affective disorders	44.6	.77	.59
Major affective disorders	29.6	.70	.65
Chronic minor affective disorders	19.9	.64	.29
Psychoses not elsewhere classified	7.0	.85	.43
Anxiety disorders	10.5	.74	.43
Factitious disorders	1.4	.49	1.00
Somatoform disorders	4.2	.53	.66
Dissociative disorders	0.7	1.00	−.004
Psychosexual disorders	2.1	1.00	1.00
Paraphilias	0.7	1.00	1.00
Psychosexual dysfunctions	1.7	1.00	1.00
Adjustment disorders	11.2	.74	.50
Disorders of impulsive control not elsewhere classified	1.7	−0.01	−0.01
Overall kappa for major classes, axis I		.78	.66
Overall kappa for personality disorders, axis II	60.6	.61	.54

[a]Percent of all subjects given diagnosis by at least one clinician. Because some subjects received diagnoses from two minor classes within a major class (e.g., a major affective disorder and a chronic minor affective disorder), the total percent of subjects in minor classes may exceed the percent of total subjects in the major class. Only in the case of substance use disorders was there an appreciable difference in percentages between the groups (joint=28.7%, test-retest=14.5%).

larly, participants were reminded that *DSM-III* was on trial, not them, and they should send in results even if they indicated poor agreement.

Both clinicians were to have access to the same material, such as case records, letters of referral, nursing notes, and family informants. If one clinician had such information (e.g., spoke to a family member), he or she was to inform the other clinician of the additional information, while at the same time avoiding communication of his or her diagnostic impression. Clinicians could either be present at the same evaluation interview (joint) or if this were inconvenient, separate evaluations could be done, as close together in time as possible (test-retest).

Each clinician recorded the results of his or her examination using the *DSM-III* multiaxial system. In this system personality disorders and specific developmental disorders are coded on axis II, and all other clinical psychiatric syndromes and conditions are recorded on axis I. The justification for separating the axis II disorders from the rest of the classification is to ensure that personality disorders and specific developmental disorders are not overlooked when attention is given to the axis I mental disorders, which frequently have more florid symptomatology. The use of the multiaxial system and the reliability of the nondiagnostic axes are described elsewhere (2).

RESULTS

Of the 365 clinicians participating in group settings and who see adult patients primarily, 274 participated in this reliability study. Two hundred and eighty-one adult patients (18 years and older) were evaluated. The ethnic-racial distribution of the patients (NIMH-recommended classification) was broken down as follows: white, not of hispanic origin (81%), hispanic (4%), black, not of hispanic origin (13%), and other (2%). The patients were evaluated in the following clinical settings: inpatient (33%), outpatient (34%), drug or alcohol service (10%), liaison service (6%), college mental health service (5%), and all other (12%). The inpatient evaluations were done in the following types of facilities: city or county psychiatric hospital (13%), state hospital (10%), private psychiatric hospital (47%), university-affiliated hospital (22%), and armed forces or VA hospital (9%).

Most of the clinicians evaluated 2 patients each; some evaluated 1, and a few evaluated several. Slightly more than half of the evaluations were done jointly.

The interrater reliability for the diagnostic classes that were represented in the patient sample is presented in table 1. Reliability is expressed using the kappa statistic, which indexes chance-corrected agreement (3). A high kappa (generally .7 and above) indicates

good agreement as to whether or not the patient has a disorder within that diagnostic class, even if there may be a disagreement about the specific disorder within the class. For example, diagnoses of paranoid schizophrenia and catatonic schizophrenia by two clinicians would be considered agreement on schizophrenia. The overall kappa for the major classes of axis I indicates the extent to which there is agreement across all diagnostic classes for all patients given an axis I diagnosis by at least one of the clinicians and is thus an overall index of diagnostic agreement.

DISCUSSION

For most of the classes, the reliability for both interview situations is quite good and, in general, is higher than that previously achieved using *DSM-I* and *DSM-II*. These results were so much better than we had expected that we wondered if our instructions for avoiding bias might have gone unheeded in many cases. However, the results of an anonymous questionnaire sent to all of the reliability participants indicated that there were only a few departures from the rules that might have inflated reliability; these included selection of a case because of an obvious diagnosis, changing a diagnosis after discovering the partner's diagnosis, or not sending in a case in which there was diagnostic disagreement. (It should be noted that studies of diagnostic reliability rarely consider the possibility of departure from protocol.)

It is particularly encouraging that the reliability for such categories as schizophrenia and major affective disorders is so high. Several important classes still have only fair reliability—schizoaffective disorder, chronic minor affective disorder, and personality disorder. In the final draft of *DSM-III*, schizoaffective disorder is redefined and will be included as one of the specific disorders within the class of "psychoses not elsewhere classified." The boundaries between chronic minor affective disorder and related disorders are being clarified, and we hope this will result in improved reliability. Although personality disorder as a class is more reliably judged than previously, how to further improve its reliability is not at all clear.

As expected, the reliabilities are higher when the interviews are done jointly. Although 40% of the test-retest interviews were done within one day of each other, almost half were done more than three days apart. Nevertheless, the drop in reliability for most of the classes is relatively small. Differences between reliabilities obtained under the two conditions may be due to different questions being asked by each interviewer as well as the patient's giving different responses to the same question asked at both interviews.

Several innovative features of *DSM-III* have undoubtedly contributed to the improved diagnostic reliability: changes in the classification itself (e.g., grouping all of the affective disorders together), the separation of axis I and axis II conditions, the systematic description of the various disorders, and the inclusion of diagnostic criteria. In future reports we will present the reliability of the individual diagnostic categories and the results of phase two of the field trial, which involves reliability interviews to assess the effect of changes made in the *DSM-III* classifications and in the diagnostic criteria.

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DSM-III Field Trials: II. Initial Experience with the Multiaxial System

BY ROBERT L. SPITZER, M.D., AND JANET B.W. FORMAN, M.S.W.

The multiaxial system of DSM-III *includes nondiagnostic data that are valuable in understanding possible etiological factors and in treatment planning and prognosis. The authors describe the reliability of axis IV—severity of psychosocial stressors—and axis V—highest level of adaptive functioning in the past year—for 281 adult patients interviewed in phase one of the* DSM-III *field trials. The kappa coefficient of agreement for axis IV was .62 for joint interviews and .58 for separate interviews, which the authors consider at least fair. Reliability for axis V was quite good, .80 for joint interviews and .69 for separate interviews. Eighty-one percent of the participating clinicians judged the multiaxial system to be a useful addition to traditional diagnostic evaluation, although many indicated that they had difficulty quantifying severity of psychosocial stressors.*

ONE OF THE major innovative features of the draft version of the third edition of the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-III*) is the provision of a multiaxial system for psychiatric evaluation. The multiaxial framework provides for the systematic evaluation of an individual's condition in terms of several variables, or axes, which are conceptualized and rated as quasi-independent of each other. The advantages of a multiaxial evaluation include comprehensiveness and the inclusion of nondiagnostic data that are valuable in understanding possible etiological factors and in treatment planning and prognosis. Furthermore, separating various aspects of the diagnostic information onto separate axes promotes higher interclinician diagnostic agreement.

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The basic rule in a multiaxial system is that for every case each axis is considered. For the system to be workable it must be restricted to a small number of axes, chosen on the basis of providing information of maximum clinical usefulness in the greatest number of cases. Several multiaxial systems for psychiatric evaluation have been proposed (1-3). The *DSM-III* multiaxial system was developed after reviewing these other systems and the experience of investigators using them.

In this paper we will describe the *DSM-III* multiaxial system and its use in phase one of the NIMH-sponsored field trials. Two questions will be addressed: What is the reliability of the nondiagnostic axes? and How do the users of the multiaxial system judge its usefulness?

THE MULTIAXIAL SYSTEM

Each individual is evaluated on each of the following diagnostic axes:

Axis I. Clinical psychiatric syndrome(s) and other conditions.

Axis II. Personality disorders and specific developmental disorders.

Axis III. Physical conditions.

Axes I and II comprise the entire classification of mental disorders as well as conditions not attributable to a mental disorder. The justification for separating the axis II disorders from the rest of the classification is to ensure that personality disorders and specific developmental disorders are not overlooked when attention is given to the axis I mental disorders, which frequently manifest more florid symptomatology.

Provision is made for indicating those instances in which there is no axis I diagnosis but there is an axis II disorder, and the reverse. Axis II may also be used to list prominent personality features not adequately subsumed by a personality disorder diagnosis, such as prominent compulsive traits.

Axis III permits the clinician to indicate any current physical disorder or condition outside of the mental disorders section of *ICD-9-CM* (4) that is potentially relevant to the understanding or management of the individual.

For special clinical, research, or teaching purposes, the following two nondiagnostic axes may be used:

Axis IV. Severity of psychosocial stressors.

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Axis V. Highest level of adaptive functioning in past year.

Axis IV permits the clinician to indicate 1) the specific psychosocial stressors that are judged to be significant contributors to the development or exacerbation of the current disorder and 2) a rating of the overall severity of stress that an "average" person with similar socioeconomic and cultural circumstances would experience. This judgment involves consideration of the amount of change in the individual's life due to the stressor, the degree to which the event is desired and under the individual's control, and the number of stressors. The individual's idiosyncratic vulnerability or reaction to the stressor should not influence the severity rating. The severity rating should reflect the summed effect of all the psychosocial stressors that are listed. A seven-point severity scale ranging from "none" to "catastrophic" is provided, with examples for both adults and children and adolescents. The specific psychosocial stressors are not coded.

Axis V permits the clinician to indicate his or her judgment of an individual's highest level of adaptive functioning during the past year. Adaptive functioning is a composite of three major areas: social relations, occupational functioning, and the use of leisure time. The clinician indicates the highest overall level of adaptive functioning that was characteristic of the individual for at least a few months during the past year, giving greater weight to the level of social relations because of its particularly high prognostic significance. A six-point scale ranging from "superior" to "grossly impaired" is provided, with examples for both adults and children and adolescents.

METHOD

Clinicians were invited to participate in the NIMH-sponsored field trial through notices appearing in *Psychiatric News* and other mental health publications. All of the clinicians who agreed to complete the required work were accepted as participants, either as private practitioners or as groups of clinicians working within facilities. The clinicians were from all parts of the country, from Maine to Hawaii, and worked in both rural and urban settings.

We have described elsewhere (5) the methodology used in the reliability study to ensure a representative sample of patients and to avoid bias, the distribution of patients among the clinical settings, and the ethnic-racial distribution.

Each participant in the field trial was expected to use the Jan. 15, 1978, draft of *DSM-III* to evaluate at least 20 patients selected as either consecutive admissions or "catch-as-catch-can," after which they were sent a lengthy questionnaire that included several questions about the multiaxial system. To insure candor in the replies, the questionnaire was anonymous. After each participant had completed at least 15 cases he or she was expected to complete 2 reliability evaluations with a colleague, either during a joint interview or separately, within no more than a few days. (Most of the clinicians evaluated 2 each, some only 1, and a stalwart few evaluated several patients.)

RESULTS

Of the 365 clinicians participating in group settings and who see adult patients primarily, 274 participated in this reliability study. Two hundred and eighty-one adult patients (18 years and older) were evaluated. Slightly more than half of the evaluations were done jointly.

The intraclass reliability coefficient for axis IV was .62 for cases evaluated in joint interviews (N=150) and .58 for evaluations made after separate interviews (N=131). For axis V the coefficients were .80 for joint interviews and .69 for separate interviews. The reliability of the judgment of the severity of psychosocial stressors is at least fair. The reliability of the judgment of the highest level of adaptive functioning is quite good. As expected, in both instances the reliability is better when the interviews are done jointly than when done separately.

Questionnaires were received from 271 field trial participants. The first question relevant to the use of the multiaxial system asked whether it was useful to divide the entire classification of mental disorders into axis I and axis II conditions. Sixty-eight percent responded yes and 10% responded no. (The proportion of clinicians responding "not sure" will not be reported for any item.)

Seventy-four percent reported that it was "useful to be able to note personality traits (as distinct from personality disorders) on axis II." Ten percent reported that it was not.

Since many field trial participants indicated in their critiques of *DSM-III* that they were dissatisfied with some aspect of axis IV, several mutually exclusive options were presented. Thirty percent favored changing the instructions so that the rating is purely descriptive of stressors that occurred before the current disorder, whether or not the clinician judges them to be significant contributors to the onset or exacerbation of the disorder. Ten percent favored eliminating axis IV on the grounds that "we do not yet know enough to measure stress in a way that would be useful clinically" and in actual practice this axis would not be used. Thirty-eight percent favored leaving axis IV as is and indicated that they found it "clinically useful in deciding a treatment approach and in evaluating prognosis" and that they would use it regularly in their diagnostic evaluations. Fourteen percent favored leaving axis IV as is even though they doubted that they would use it themselves but that "in some settings it would be useful information (e.g., for research)."

Only one question seemed necessary to assess axis V. Sixty-three percent judged it useful, and 10% did not. One question assessed the overall attitude toward

the multiaxial system: 81% agreed with the statement, "The *DSM-III* multiaxial system will be a useful addition to traditional psychiatric diagnosis," and 4% disagreed.

DISCUSSION

The limited reliability of the severity of psychosocial stressors aspect reflects the problems involved in quantifying this dimension. Many of the participants noted that they had difficulty in using the concept of the hypothetically "normal" individual as the baseline for judging the severity of the stressor. They often objected to the instruction to ignore the patient's idiosyncratic vulnerability in assessing severity of stressors. The solution to this problem is not at all clear; if one were to include the individual's vulnerability, the ratings would be more a reflection of individual psychopathology than of psychosocial stressors.

Other field trial participants took the opposite approach and objected to any etiological attribution to the stressors. They argued that the rating should merely be of stressors that occurred before the development of the illness, whether or not the clinician judged them to have any relationship to the onset or exacerbation of the illness. Although a large portion of the questionnaire respondents favored this approach, the majority seemed to favor leaving the instructions as they were. We are reluctant to change the instructions in the direction of pure description, despite its apparent advantages in objectivity. If the clinician were asked to consider events that he or she believed had no relationship to the onset or exacerbation of the disorder, the clinical usefulness of the information would seem to be extremely limited.

Because of the large number of negative comments about axis IV, we were surprised that so few wished to eliminate it from the multiaxial system.

Axis V was not only reliably judged but met with considerable approval. Several participants made valuable suggestions for modifying the examples in the scale to include more examples of high levels of adaptation in members of low social class.

Despite the greater demand placed on the clinician by the multiaxial system and the difficulties involved in the use of axis IV, the vast majority of the field trial participants apparently believe that the *DSM-III* multiaxial system is a useful addition to the traditional diagnostic evaluation. Future studies are necessary to determine the extent to which the multiaxial system and, in particular, axes IV and V will actually be used in clinical practice and their impact on patient care.

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Massive Benzodiazepine Requirements During Acute Alcohol Withdrawal

BY ELAINE WOO, M.D., AND DAVID J. GREENBLATT, M.D.

Severe alcohol withdrawal developed in an abstinent chronic alcoholic man. Massive doses of benzodiazepines (2,335 mg of diazepam intravenously, 21,225 mg of oxazepam orally) achieved only marginal control of delirium and agitation. Analysis of multiple blood samples drawn during and after the withdrawal episode indicated, as expected, very high concentrations of diazepam and metabolites and of oxazepam. There was no evidence of an abnormal pharmacokinetic profile. Benzodiazepine resistance in withdrawing alcoholics probably reflects a receptor-site phenomenon rather than an abnormal drug disposition.

BENZODIAZEPINE DERIVATIVES, in particular chlordiazepoxide and diazepam, are extensively used in the treatment of the acute alcohol withdrawal syndrome (1, 2). The safety and effectiveness of benzodiazepines in acute alcohol withdrawal are well established, but doses greatly exceeding the usual therapeutic range are often required to produce an adequate clinical response. The reasons for these increased drug requirements are not known. The present report describes a case of acute alcohol withdrawal in which very large doses of oxazepam and diazepam were needed to control clinical manifestations.

CASE REPORT

A 33-year-old man was hospitalized following a grand mal seizure. The patient had a history of at least 3–4 years of heavy ethanol ingestion. He had been hospitalized two previous times for the alcohol withdrawal syndrome. In prior admissions hepatomegaly and ascites were noted, and micronodular cirrhosis was documented by percutaneous liver biopsy. Liver function tests were normal during periods of abstinence.

During the first day of hospitalization, the patient developed tremulousness and sinus tachycardia, but mental status remained normal. High-dose oxazepam therapy was begun, and the patient received 780 mg during the first 24 hours. Despite this, he rapidly developed severe agitation, hallucinations, and disorientation, consistent with the alcohol withdrawal syndrome.

Massive doses of oxazepam and diazepam were given over the next 7 days. A total of 2,335 mg of diazepam was given intravenously over 4 days. Bolus doses ranged from 10 to 100 mg, with a maximum of 270 mg given in three doses over 45 minutes. The maximum 24-hour dose of diazepam was 875 mg on the third hospital day. Oxazepam was given concurrently whenever oral intake was possible. Over 9 days, a total of 21,255 mg of oxazepam was given. Maximum single doses were 150 mg/hour, and a maximum 24-hour dose of 3,500 mg was administered with only minimal control of the withdrawal syndrome. There was no evidence of respiratory or cardiovascular depression, and only minimal sedation, attributable to any dose. Diazepam was discontinued on the fifth hospital day (point A, figure 1), and oxazepam was tapered and discontinued on the ninth day (point B, figure 1). Normal mental status had returned by the seventh day of hospitalization. The patient was subsequently discharged to a long-term treatment and rehabilitation facility.

METHOD

A venous blood sample was obtained prior to the initiation of benzodiazepine therapy and then at multiple points in time over the next 35 days. Plasma was separated and stored at −20°C until the time of assay. The patient's mental status was assessed at the time of blood sampling by observers having no knowledge of the drug concentrations. Plasma concentrations of diazepam and its three major metabolites (desmethyldiazepam, temazepam, and oxazepam) were determined by electron-capture gas-liquid chromatography (3–5).

RESULTS

Figure 1 shows plasma concentrations of the four active compounds over a 38-day period. High levels of oxazepam were rapidly achieved within the first 12 hours. The onset of delirium tremens coincided with a decrease in oxazepam levels due to a premature a-

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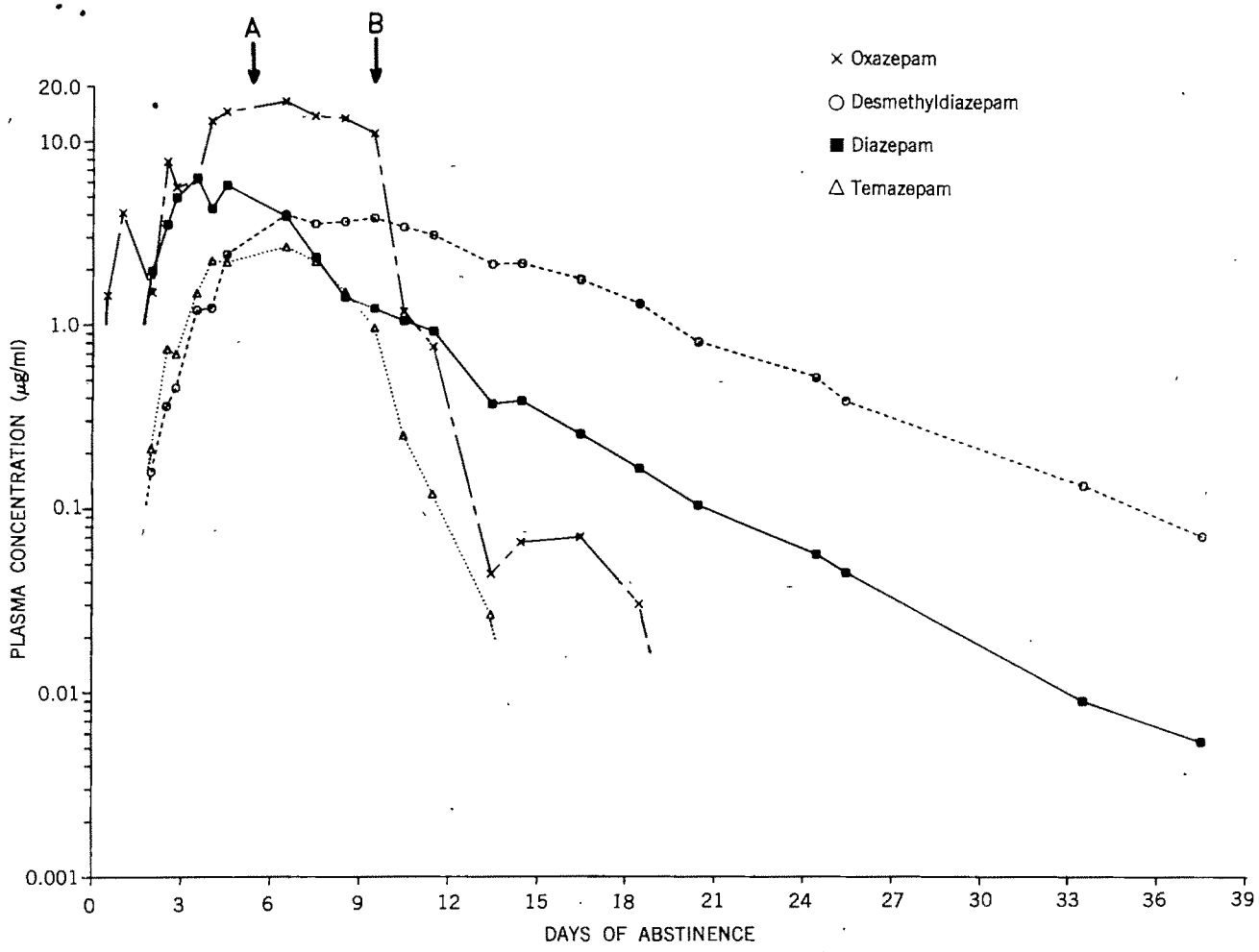
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FIGURE 1
Plasma Concentrations of Diazepam, Desmethyldiazepam, Oxazepam, and Temazepam During and After Benzodiazepine Therapy for Alcohol Withdrawal[a]



[a]Point A, termination of intravenous diazepam therapy (total dose: 2,335 g); point B, termination of oral oxazepam therapy (total dose: 21,255 g).

tempt to wean the patient off sedative medication. The maximum plasma concentration of oxazepam was 14.8 μg/ml on day 6, at which time the patient was severely disoriented and agitated.

High concentrations of diazepam were obtained within 12 hours of beginning therapy. The maximum level was 6.4 μg/ml achieved on day 3, but even these concentrations produced only transient improvement of the patient's severe agitation.

Desmethyldiazepam and temazepam, the two active diazepam metabolites, rapidly appeared in plasma, reaching maximum concentrations during the 6th day of hospitalization. Temazepam concentrations thereafter fell to undetectable levels by the 14th day; its apparent half-life of disappearance was 21 hours. Disappearance of diazepam and desmethyldiazepam was much less rapid. Both compounds were detectable 32 days after the last dose of diazepam. The apparent elimination half-life values for diazepam and desmethyldiazepam were 68 and 120 hours, respectively. Oxazepam levels fell sharply after termination of therapy on day 9, with an apparent disappearance half-life of 13 hours. Thereafter, relatively low concentrations of oxazepam remained present in plasma, probably consistent with that produced from endogenous metabolism of diazepam.

DISCUSSION

The present report describes a patient with severe alcohol withdrawal syndrome requiring massive doses of benzodiazepine derivatives. Despite the very large doses, adequate clinical control was barely achieved. Plasma concentrations of diazepam and oxazepam were 10- to 100-fold greater than those usually observed clinically, consistent with the large doses administered. Furthermore, biotransformation of diazepam to its pharmacologically active metabolites, as well as elimination of these compounds, appeared to

be relatively normal. Thus the patient's resistance to drug effects cannot be attributed to failure of drugs to reach the systemic circulation or to abnormalities of drug biotransformation or elimination. We cannot exclude the possibility that the compounds failed to reach their sites of action in the brain. This, however, seems unlikely, since benzodiazepines are highly lipophilic and readily penetrate the blood-brain barrier (6). During a previous admission, the patient experienced a similar clinical course and was treated with high doses of chlordiazepoxide. Analysis of cerebrospinal fluid (CSF) concentrations of chlordiazepoxide and metabolites at that time clearly indicated the presence of adequate levels of drug and metabolites in CSF.

A similar type of drug resistance or adaptation occurs following massive overdosage of diazepam in physically healthy individuals (5). The resistance of such patients to the action of benzodiazepine derivatives probably occurs at the cellular sites of drug action (7). The alcohol withdrawal syndrome itself, by poorly understood mechanisms, may also contribute to rendering such individuals relatively insensitive to the effects of central depressant compounds.

Further complicating the clinical problem of drug resistance during the early phase of treatment is the persistence of diazepam and its active metabolites for weeks after cessation of therapy. High residual levels of active compounds did not lead to untoward sequelae in our patient. However, prolonged obtundation has been reported following treatment of tetanus with high-dose diazepam (8, 9). The use of shorter-acting benzodiazepine derivatives having no active metabolites, such as oxazepam or lorazepam, might obviate such potential problems (10) and deserves further evaluation in the therapy of alcohol withdrawal.

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Implications of Increased Dosage of Neuroleptic Medications During Psychotherapy

BY JOSEPH N. DIGIACOMO, M.D., AND RICHARD CORNFIELD, M.D.

Unrecognized emotional reactions on the part of the psychiatric trainee may result in the inappropriate use of medications. To illustrate the possibility that increases in medication dosages may be related to the psychiatric clinician's lack of control, the authors present three clinical examples and discuss the concept of countertransference. A model of supervision is described in which the supervisor, trainee, and patient meet to correct these therapeutic distortions and reduce the amount of medication required.

WITH INCREASED experience in assessing the effectiveness of neuroleptic medications, psychiatrists have gradually evolved general guidelines for a range of acceptable dosages in clinical practice. Some psychiatrists, however, continue to use neuroleptics in dosages substantially higher than those recommended in the clinical literature (1). The employment of high doses of neuroleptic medication merits careful evaluation. Although such practice is often therapeutically justifiable, it may also reflect patterns of lack of psychotherapeutic control in the doctor-patient interaction.

With the exception of a 1975 Group for the Advancement of Psychiatry study (2), psychiatry seems to have been curiously unattuned to subtle issues in the doctor-patient relationship that are obscured by such prescribing policies. Specifically, we are suggesting that excessive use of neuroleptic medication may indicate countertransference distortion on the part of the unwary psychiatrist and masquerade as effective rational treatment.

Marked changes in drug dosages should alert the supervisor of psychiatric residents and medical students to possible unrecognized emotional reactions on the part of the trainee to his or her patient. One pattern consists of gradual or rapid increase in medication, designed to control the patient's symptoms or behavior. The observable worsening of the disorder is initially assumed to be caused by the psychotic process for which the patient is being treated. If a therapeutic impasse is then reached, the trainee may label it "refractoriness" and increase the medication dosage, accentuating untoward side effects without concomitant control of the symptoms. If this sequence coincides with increasing distance in the therapeutic relationship, the pattern of medication usage may become a fertile area for the trainee to explore his unrecognized emotional response, or countertransference, to his patient. With a beginning therapist, the lack of countertransference control often seems most pronounced in those areas in which intense feelings or unpleasurable affects are generated. We will present examples that involve sexual feelings, hostility, and fear.

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CLINICAL EXAMPLES

Sexual-Erotic Feelings

A 25-year-old schizophrenic man was admitted to the hospital because of the acute onset of unruly behavior and bizarre ideation. He had been maintained on antipsychotic drugs and had remained out of the hospital for 2 years. His wife had become exhausted and resentful from caring for him. The patient responded dramatically to a female therapist, who used supportive psychotherapy and prescribed 600 mg/day of chlorpromazine. In 6 weeks the patient was participating in the ward community. However, at the end of the second month, his medication was abruptly increased to 1200 mg/day of chlorpromazine and 30 mg/day of trifluoperazine, and he required periods of restraint and seclusion.

Because of the high dosage of medications, a three-way interview was suggested with the trainee, patient, and supervisor. On entering the patient's seclusion room the supervisor observed that the patient was partially undressed and had many *Playboy* posters on his wall. A display at the foot of his bed depicted a stuffed turkey on a plate with the caption "I would like to stuff your turkey" (related to the Thanksgiving holiday). He appeared agitated yet somewhat jocular, with a rapid-fire, non-goal-oriented speech pattern. The supervisor inquired about the nature of the patient's relationship with his doctor and he replied that he was

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very attached to and dependent on her. It was soon evident from his associations that the patient was having intense sexual fantasies about his therapist. He talked about the problem of dealing with sexual feelings in the hospital. He felt secure with and close to his therapist at first but later noted a strong sexual attraction developing, with no means of letting off the sexual tension.

Later in supervision, the therapist said she had not noticed the posters. She revealed feelings of extreme protectiveness and solicitousness toward this patient and most of her patients. She said that about 2 weeks before this interview she began to feel mounting tension in the therapeutic relationship. It was at this time that she increased the dose of neuroleptics. After several discussions, both in individual and group supervision, the therapist was able to talk about her difficulty in dealing with a patient who made a strong sexual overture to her and saw that this problem was related to personal conflicts. Within a week she decreased the dosage of chlorpromazine to 600 mg/day, and the patient was again out of seclusion and participating in ward activities. In this situation the therapist, who was made anxious by the mobilization of her own sexual conflicts, retreated from the proper therapeutic approach and substituted psychoactive drugs in high dosages.

Hostile Feelings

A 50-year-old schizophrenic man was admitted to the hospital because of his progressive inability to cope with his family and to remain in the community. He spoke of various aches and pains in his body. Initially the patient had responded positively to 300 mg of thioridazine. His therapist was changed, and 4 days later the new therapist prescribed 800 mg/day of thioridazine and recommended an additional drug. Because of the apparent deterioration in the patient's condition, a supervisory three-way interview was arranged and the patient was asked about his feelings regarding his relationship with his new therapist. The patient was somewhat hesitant at first but with little encouragement began to speak in a very controlled way, periodically glancing at the student. The patient expressed concern that his current therapist had little regard or respect for him, that he was feeling "always put down," and that he was tense after their meetings. He said, "We always seem to end up disagreeing."

In group supervision following the interview, the student expressed surprise at what the patient had said. He felt, in retrospect, that he was "struggling with the patient for some reason or other." It was then noted that in the past his interpretations regarding this patient seemed "overheated," and he was asked if this case reminded him of something in his own life. The therapist admitted with annoyance that he could not tolerate the patient's constant complaints about his chest pain. He sensed that he was controlling the patient's verbal productions through frequent verbal interventions of his own in the therapy sessions. When asked why the complaints of chest pain produced this reaction, he responded that it was just something he felt strongly about.

Several days later he shared enthusiastically with the staff his insight that his father had had a myocardial infarction and had gone to the hospital, been treated, and returned home without ever showing a single sign of discomfort. He felt a deep sense of pride about his father's response to this painful, life-threatening situation and realized that it predisposed him to be critical of his patient's behavior. He was quick to point out that he still thought that his father reacted better to his heart attack than the patient did, but at least he now felt less antagonistic toward his patient. In a group discussion later, the therapist became aware that not only was he creating a very high standard for his patient but that he was trying to lead his own life by the same standards. He returned to his patient with considerably more comfort and enthusiasm. After the patient complained of being too sedated on 800 mg of thioridazine the dose was rapidly reduced to 200 mg/day.

Fearful Feelings

A 21-year-old acute schizophrenic man was admitted to the hospital in an extremely disorganized and hostile state, requiring both restraints and seclusion. The therapist prescribed 1200 mg/day of chlorpromazine and then added 40 mg/day of trifluoperazine, but little improvement in the patient's condition was noted. After 3 weeks, the staff was somewhat concerned about the escalating drug dosage and the continued use of leather restraints and spoke to the supervisor, who scheduled a three-way session.

During the interview, the therapist sat at a considerable distance from his patient. The patient became increasingly unruly and agitated as the therapist remained "frozen" in the background. The supervisor approached the patient, made physical contact with him through a handshake, and moved a chair close to the bedside. He initiated a friendly discussion, alluding to the fear that had been generated in the staff and patients by this patient's hostile demeanor. Although he was surprised to hear about the reaction, the patient admitted to being quite frightened himself. The supervisor attempted a demeanor of warmth and interest, which had clearly been lacking in the resident's interaction with the patient. A frank discussion of the nature of the relationship between the therapist and the patient ensued, and it became clear to the therapist that he had been unaware of the paralyzing fear that the patient generated in him. He realized that he tried to assuage his own fears by greater reliance on psychotropic medication as a chemical "restraint" of the patient.

In supervision, the therapist told about restrictions in his own life and fears of loss of control. The therapist gradually sensed that he had overcontrolled the patient's responses both with medication and with dis-

tancing maneuvers during the psychotherapy sessions. However, his subsequent responses appeared overcompensatory; he began to spend a great deal of time in close contact with the patient, frequently accompanying him on walks around the hospital grounds. He was never able to establish optimum distance and objectivity with this patient.

Peer group supervisory sessions in which the therapist became aware of his fears were not sufficient to help him deal with this patient or other intimidating patients. Attempts to desensitize his anxiety and increase his tolerance of aggressive behavior through psychodrama, observation of other trainees' work with similar patients, and direct observation of a senior staff member's management of such a patient were unsuccessful.

DISCUSSION

The interrelationship between pharmacotherapy and psychotherapy has raised many complicated issues (3–6). Recently, Docherty and associates (7) discussed the problem of "bimodal relatedness," involving the therapist's relationship to the patient as a diseased organ or as a disturbed person; they stress the need to continually resolve the tendency toward such polarization. The Group for the Advancement of Psychiatry in its 1975 publication (2) attempted to summarize studies of both treatment modalities in the major psychiatric disorders. The report discusses the possible beneficial effects of drugs on psychotherapy, such as facilitating the patient's accessibility to therapy by relief of the patient's symptoms, which enhances the ego functioning that is essential for participation in the therapy process. However, there is no documentation of the harmful effects of countertransference problems on proper clinical pharmacotherapy.

Sabshin and Eisen (8), several years after the introduction of neuroleptics in this country, showed that fluctuations in ward tension had a significant effect on the doses of tranquilizers used and suggested the need for careful studies related to prescribing practices and shifting levels of staff anxiety and discomfort.

Sarwer-Foner (5, 9, 10) emphasized the psychodynamics of prescribing practices in various settings. He stressed not only specific pharmacologic drug effects on the mental apparatus, such as dampening intensity of impulses, but also such factors in the total physician-patient relationship as the social setting, the patient's expectations of a drug's effect, the patient's transference to the therapist, and the physician's countertransference reactions (e.g., medication choice based on fear of interpersonal relatedness and a need for distance from a particular patient) (9).

In general, there is little documentation of the effects of stresses in psychotherapy on prescribing practices, particularly as manifested by sharp increments in medication dosage. The examples cited involve countertransference attitudes of the therapist, defined as emotional reactions to the patient resulting from unresolved unconscious conflicts in the therapist that impede effective therapy and lead to inappropriate use of neuroleptic medications.

Greenson (11) has discussed a variety of emotional reactions that may suggest lack of control. Among them are attitudes of love, hatred, pugnaciousness, and one-upmanship, as well as some subtle forms of countertransference such as rescue fantasies, unyielding good-naturedness and benevolence, protectiveness and motherliness, and feelings of boredom, coldness, and indifference. He mentioned the effect of "extraordinary experiences in the [therapist's] life [that] make him temporarily excessively susceptible to countertransference reactions as, for example, personal miseries, illness or death in his family, a pregnancy, the loss or suicide of a patient, etc."

There appears to be general knowledge of countertransference difficulties revealing themselves in either the failure to medicate the patient or the underutilization of medication (6). The dynamics of such situations may relate to the therapist's need to preserve an image of himself as a messianic healer who can fulfill all of the patient's needs and desires, thus assuring himself of his own ability to handle unresolved infantile fears and needs.

It seems equally important to consider the increased use of medication as an indication of a countertransference reaction. Such lack of control should always be suspected when a therapist is struggling to be helpful to the patient. This behavior results in a therapeutic step that often betrays the hidden conflict and usually proves to be nontherapeutic. To deal with such situations, particularly at a time of growing concern in the field of psychiatry over the morbidity associated with high doses of neuroleptics (12), we suggest that trainees and staff become sensitive to the issue of fluctuating doses of medication, particularly in those patients who were treated initially in an effective manner with modest doses.

The presence of delayed escalating doses can be an indication for an interview with the therapist, patient, and attending psychiatrist. This model of supervision requires careful thought and intervention, as it involves more than the usual problems inherent in a supervisor-trainee relationship. Particularly, countertransference needs on the part of the supervisor must be guarded against and the triadic interview must be delicately handled to avoid frightening the already struggling therapist. During the interview, the supervisor may take the initiative to clarify the interaction between the patient and therapist and identify affects that have developed which the patient and therapist have been unable to approach. The therapists are frequently able to use these encounters to assimilate areas of selective inattention through further direct and/or peer group supervision. Students and trainees, after overcoming defensive tendencies during the sometimes stressful interviews, often refer to these sessions as high points in their learning experience.

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Relations Between Academic Departments of Psychiatry and Pharmaceutical Companies

BY MICHAEL JELLINEK, M.D., AND AARON LAZARE, M.D.

The financial pressures placed on academic departments of psychiatry make the offers of assistance from pharmaceutical companies very attractive. The authors provide a sequential three-step decision-making approach to help the academic physician and the psychiatry department address the ethical issues involved in any given interaction with a pharmaceutical company. They also provide examples of the application of these guidelines.

RECENT FINANCIAL CONSTRAINTS on departments of psychiatry require even more vigorous efforts to maintain high academic and service standards. Hospitals, reflecting institutional budget concerns, are developing administrative systems that emphasize cost containment and budgets based almost solely on income from patient services. Federal training grants have virtually disappeared, and third-party carriers have limited the definition of reimbursable services. As a result, funds for many departmental activities and resources, such as resident training, library facilities, guest lecturers, and research, have been severely restricted or eliminated. Departments have responded to these financial pressures by acquiring such new sources of revenue as contracting with community facilities and the Veterans Administration for resident time, sponsoring postgraduate courses, and engaging in various collaborative relationships with pharmaceutical companies.

The current extent of the interaction of academic physicians and psychiatry departments with pharmaceutical companies is remarkable in both number and complexity (see appendix 1). In one day the academic physician may use a pen embossed with a pharmaceutical company's name, write on note paper that has company advertising printed on it, carry company gifts of medical instruments in a company-donated bag, and

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participate in company-sponsored research or teaching. The academic department may accept funds from a company for a guest speaker, use reference books donated by a company, carry out research funded by a company, or provide consultation or teaching time to a company.

The patient and the physician are often unaware of the interaction between department and pharmaceutical company because of the organizational complexity of the academic institution. For instance, a patient may not realize that a research project in which he or she has agreed to participate is serving, in part, the interests of a pharmaceutical company. A staff member may attend a lecture without knowing how much of the subject matter has been determined by a sponsoring drug company. The private practitioner, on the other hand, is in direct contact with the drug representative and invariably knows the nature of the interaction. Further, the private practitioner is not involved in research and teaching in his or her office.

At first glance it may appear that the variety of interactions between department and company simply enhance the position of both parties. In return for its financial investment the drug company may gain goodwill, the department's gratitude, an opportunity to educate and influence physicians as to the efficacy of its product, and a form of quasi-affiliation with the department, all with the understandable goals of enhancing its prestige, employee morale, and its competitive position. In return for its services the academic department is able to gain support for a wide range of patient care, education, and research programs.

A closer examination reveals that although the drug company's sales-oriented goals are straightforward and understandable, the psychiatry department's goals are more complex. For example, a department is entrusted by its larger institution (hospital and/or medical school) and by the public to discover truth (research) and to disseminate truth (teach). When a department appears to promote a particular drug by using company-printed stationery, by selecting company-designated research, or by sponsoring drug displays at lectures, the department faces the ethical dilemma of achieving its goals only by risking its public trust and academic purpose.

Academic departments are increasingly reliant on pharmaceutical companies through a wide variety of conceptually different interactions. It is important, therefore, that departments study the effects of these interactions on their broader goals and develop a system of ethical decision making. Traditional medical oaths or the more modern contractual models define basic ethical principles, but they provide no guidelines that are readily applicable for dealing with pharmaceutical companies or the complex ethical dilemmas in large medical institutions. We propose a sequential three-step decision-making approach that we hope will help the academic physician and the psychiatry department address the significant issues involved in any given interaction with a pharmaceutical company.

GUIDELINES FOR DECISION MAKING

Step 1. Review the specific details of the proposed activity in terms of basic ethical principles. In practice, these basic ethical principles would not permit unauthorized review of a patient's medical record (right of confidentiality), would not allow physicians to be diverted from the immediate and direct care of patients (physician's duty to the patient and pledge to do no harm), and would mandate that the patient be given the right to be fully informed and exercise free will in giving consent.

Step 2. Review the proposal in terms of its ultimate benefit to patient care. This guideline requires that there be significant benefit to the patient, to the department, to the physician, or to society in terms of increased knowledge (research or training) and/or increased quality and amount of patient care.

Step 3. If the proposal does not violate basic ethical principles and is a significant benefit, then consider potential risks. Risks include 1) diverting departmental or individual resources of time and energy to company interests, 2) raising patient concerns or even mistrust based on the patient's perception that the physician's judgment may be influenced by pharmaceutical companies' advertising or gifts, 3) the companies' use of their "affiliation" with the department to suggest an endorsement of their sales representative and their products in subsequent interactions with community physicians, and 4) contributing to the higher cost of medications without ultimate benefit to the patient.

EXAMPLES

1. Donations of funds for books. The anonymous donation of funds for books does not violate basic ethical principles (step 1), is of potential benefit to the department and indirectly to patient care (step 2), and has little risk of departmental or social corruption (step 3). The donation of specially designated books would carry certain risks if these books represented an unjustifiably large percentage of the available reading material in a small departmental library.

2. Company support for guest speakers. Support for speakers is a more complex problem. In general, funding speakers violates no basic ethical principles (step 1). However, the benefits are limited if the speaker or topic is circumscribed or biased on the basis of such company guidelines as a speaker's bureau or support limited to specific topics (step 2). The risks of diversion of departmental interest as well as departmental endorsement are variable as well, depending on the setting of the lecture (special lecture versus grand rounds, for example) and whether there is publicized sponsorship or active sales promotion associated with the lecture (step 3).

3. Stationery or memo paper with company advertising. In this example the benefit to the department is purely financial, i.e., a relatively minor administrative

saving (step 2). The risks include subliminally influencing physician prescribing patterns (choice of drug and use of trade name) and arousing patient concern or mistrust because the physician is identified on an advertising handout rather than on departmental or individual letterhead, memo sheets, or blank paper (step 3).

4. Outright gifts. Personal gifts such as lunches are without direct or indirect benefit to patient care (step 2). There are several risks in gifts, including influencing physicians by considerations other than what is best for the patient, blurring the boundaries between company and physician (step 3), and increasing the price of medicine without ultimate benefit to patient care.

5. Training pharmaceutical personnel. The training of pharmaceutical personnel by direct patient contact violates the basic ethical principle of confidentiality (step 1). Even when teaching is conducted without patient contact, the academic department risks violating the public trust when the company personnel attempt to use their "teaching affiliation" to enhance sales promotion (step 3).

CONCLUSIONS

The significant financial pressures on academic departments make pharmaceutical company offers very attractive and may easily divert a department's or a physician's attention away from making judgments about the most valuable use of time and the broader ethical implications of eroding the integrity of a department or teaching position. Conceptually, our guidelines on relations with pharmaceutical companies suggest that the academic department is generally bound by the same basic ethical principles as are its individual physician members and, further, that departmental activities be guided by ethical principles that acknowledge the department's societal position, role, and concomitant obligations.

APPENDIX 1
Examples of Pharmaceutical Company Support to Academic Departments of Psychiatry

GIFTS
- Pens (with or without advertising)
- Memo pads and stationery (with or without advertising)
- Medical instruments (with or without advertising)
- Reprints (general reviews or sales promotional)
- Textbooks
- Monographs (general reviews or sales promotional)
- Meals
- Drug samples for the physician's personal use, use for patients, or charitable use

SPEAKERS
- Academic conference of general topics (with or without acknowledging sponsorship)
- Company-approved speakers for specific topics
- Directly promotional speakers for specific drugs

RESEARCH
- Anonymous general support
- Support of drug evaluation research
- Research design based on sales promotional criteria

CONSULTATION TO THE COMPANY
- Company executive training without patient contact
- Company executive training with patient contact
- Sales promotion (with or without funds)
- Product improvement

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A Clinical Note on Hysterical Psychosis

BY JESSE O. CAVENAR, JR., M.D., JOHN L. SULLIVAN, M.D., AND ALLAN A. MALTBIE, M.D.

The authors review the concept of hysterical psychosis and its reported psychodynamics. They suggest a psychodynamic issue that apparently has not been previously reported, namely, emotions engendered by an overt sexual advance or rage and disappointment over a lack of a sexual advance. This finding can be most helpful in distinguishing a case of hysterical psychosis from an acute schizophrenic episode.

MOST CLINICIANS AGREE that there is an entity best described as a hysterical psychosis that is distinctly different from an acute schizophrenic episode. Hysterical psychosis usually has a sudden and dramatic onset and may be manifested by hallucinations, delusions, depersonalization, derealization, and bizarre behavior. The hallucinations may be visual instead of auditory. The affect is not flat or blunted as may be seen in schizophrenic psychosis; instead, the affect may be effervescent and expansive. If a thought disorder is present, it does not resemble the looseness of associations seen in schizophrenia. Brill (1) stated, "The thought disorders in hysterical psychosis, even though characterized at times by delusions and hallucinations, are more like the simple distortions of reality seen in the very angry or fearful child, which disappear when emotional control is achieved" (p. 1066).

The acute psychosis may last from a matter of hours to several weeks. Once the psychosis has run its course, there are no residual effects; no chronic disability exists.

Hysterical psychosis occurs most commonly in persons (usually women) who are best described as having hysterical personalities. This personality type is suggested by a person who is self-centered, vain, dramatic, seductive, suggestible, emotionally labile and excitable, attention-seeking, and dependent. The person's moods tend to be shallow and superficial, and the thinking pattern is directed and controlled by the prevailing mood. These people tend to react to an affect-laden situation by becoming distraught; as the anxiety mounts, the person becomes overwhelmed and unable to mobilize an integrated pattern of behavior. At the extreme, this breakdown in functioning leads to a hysterical psychosis.

The dynamic events leading to decompensation in psychosis are more obscure. Hirsch and Hollender (2) have suggested only that a profoundly upsetting event or circumstance leads to decompensation. Mayer-Gross (3) felt that the psychosis develops with an emotional loss, an affective trauma, or a deprivation of support and home. Astrup and associates (4) suggested that hysterical psychosis is precipitated by fairly easily recognized exogenous factors. However, they did not suggest what these factors are.

Langness (5) has described the dynamics of hysterical psychosis in the Bena Bena tribe of New Guinea. In the examples given, it appears that the psychosis served as a discharge for aggressive impulses that could not be released in a socially acceptable manner without the psychosis.

Martin (6) has described the dynamics of women who have developed hysterical psychosis as a result of marital difficulty. He believed that these women have symbiotic attachments to their husbands and that their marriages are founded on hate instead of love. Yet these women have marked unresolved separation anxiety that ties them to their husbands, who are hated need-satisfying objects. Martin suggested that the women show oral, narcissistic character structures with primitive symbiotic object relationships and that they become psychotic when their life situation becomes intolerable. A short-lived hysterical psychosis provides the needed escape from the intolerable situation.

In the meager literature on hysterical psychosis, various dynamics have been proposed as leading to psychotic decompensation. The purpose of this report is to suggest that, in our contemporary society, there is a common dynamic factor leading to hysterical psychosis which has not to our knowledge been previously reported. This factor has been present in every case of hysterical psychosis that we have seen. We believe it has direct clinical importance because of the occasional difficulty in distinguishing a hysterical psychosis from an acute schizophrenic episode. If this dynamic issue can be ascertained, it clearly directs the physician's thinking toward a hysterical psychosis. Since the treatment and prognosis of a hysterical psychosis and a schizophrenic disorder may be quite dif-

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ferent, the presence of this dynamic issue has paramount clinical importance.

The following case reports are illustrative of our experience.

CASE REPORTS

Case 1. A 21-year-old single woman was seen for emergency psychiatric evaluation because of the acute onset of auditory and visual hallucinations, bizarre behavior, and a dazed state. These symptoms had been present for 5 days and were worsening.

The mental status examination revealed a young woman who appeared dazed and bewildered. There was a rhythmic movement disorder of her right hand, and her eyes were fixed in the upward field of gaze. She did not appear depressed and denied depressive symptoms. She admitted to auditory hallucinations of her father's voice and had visions of her father reading the Bible to her. All of her conversation seemed to concern the father.

The patient was admitted to the inpatient unit because of the obvious psychosis and extreme regression. After a short period of observation, a psychiatrist asked the patient to tell him about her sexual feelings concerning her father. The rhythmic motion of the right hand immediately stopped, the upward gaze ceased and eye contact was established, and she began to talk in a coherent and logical manner. She revealed that her father had seemed to have "unusual" feelings about her since she was an adolescent. She had gone into military service to get away from home and particularly her father. Upon her discharge from military service and return home, her father commented on how much she had grown and casually asked her to remove her clothes. She complied; the psychosis began soon afterward.

After the patient openly discussed her sexual feelings for her father and her father's sexual feelings for her, the psychiatrist reminded her that people have many different feelings about many different things and that just because feelings may be present, it is not necessary to act on the feelings. After the one psychotherapy session, the patient's hysterical psychosis cleared completely in 24 hours; she was then transferred to an open ward.

Case 2. A 23-year-old single woman was admitted to a psychiatric facility on an emergency basis because of the sudden onset of psychotic behavior. She had been with a date in a nightclub when she suddenly stood up and screamed that she was being raped and began to throw various objects at patrons in the club. The police were called, and she was brought in this violent state for admission.

The mental status examination revealed an attractive young woman who talked about being raped in view of everybody at the nightclub. She commented that she would probably need to be hospitalized for 9 months and reminded the psychiatrist that this was the length of a pregnancy. She was seductive and coy throughout the interview; her affect was volatile and ranged from rage to flirtatiousness.

The patient improved rapidly after hospitalization. Subsequent interviews revealed that her date was a man whom she had dated for several months; she was aware of strong sexual feelings for him and was angry that he was not making sexual advances toward her.

Case 3. A 24-year-old married woman was found unconscious in her home and was taken to a community hospital where she was believed to have had a cerebral hemorrhage. She was transferred via helicopter to a large medical center for neurosurgical intervention; prior to transfer a brachial cutdown had been done without anesthesia.

Neurological examination at the medical center revealed no localizing signs or nuchal rigidity. The patient appeared to be comatose but did respond to deep pain. Psychiatric consultation was requested.

The mental status examination revealed a very attractive young woman who was comatose and dressed in "baby doll" attire. The psychiatrist sat by her examining table and, after calling her by name, began to talk to her about how frightened and anxious she must be. After a few minutes, the patient opened her eyes and looked around in a dazed manner; slowly she began to ask where she was and how she arrived there. The intravenous tubing was removed and the cutdown site sutured.

As the patient became more alert and responsive, she revealed that her husband was a military officer who was stationed overseas. She had entertained sexual fantasies about another man for several months; she had gone unaccompanied to the officers' club the previous evening and "accidentally" met this man. He had made an overt sexual proposition; she had no memory of the events from the time of the proposition until she awoke in the emergency room. Subsequent inquiry revealed that she had been acting in an irrational manner and talking strangely before leaving the officers' club the previous evening.

Case 4. A 30-year-old married woman was brought for psychiatric evaluation because of bizarre behavior of 48 hours' duration. During the mental status examination she looked about the room in a dazed, bewildered manner and responded to questions by darting her tongue in and out of her mouth without making a sound.

Family members who accompanied her said that she had attempted to wrap herself around a utility pole the previous evening. They suggested that she had been "hypnotized by a snake." A salesman known to the patient had called at the home she shared with another woman. The salesman and the other woman had sexual intercourse in a nearby room, and the patient could hear various noises. Later, the woman inquired about the travel bag the salesman had, and he told them that it contained his pet snake. At the women's insistence, he unzipped the bag and the snake crawled out. The patient commented that it was "the biggest, most beautiful snake" she had ever seen; she was immediately "hypnotized" and had displayed the bizarre behavior ever since.

The psychiatrist, who believed the woman had a hysterical psychosis, explained to her clearly that she was hypnotized by the snake but that he had medication more powerful than the snake's hypnotic power. With great inspiration and ceremony, he explained that she would be well before the medication reached her stomach. He gave her a colorful pill, and she suddenly recovered. The family members felt she had returned to her usual level of functioning.

The patient was seen for two follow-up visits and remained well. She was aware of intense sexual feelings for the salesman and was able to verbalize the conflict about her feelings.

DISCUSSION

In each of the cases reported, either a sexual advance was made to the patient or the patient was angry

because a sexual advance had not been made. Hysterical persons tend to sexualize all encounters and human relationships but are frequently in conflict about their own sexuality, as manifested by the fact that many hysterical women are nonorgasmic. The internal conflicts are tremendous. On the one hand, the woman wants to accept the sexual suggestion; on the other, she feels intense anxiety and guilt because of the desire to accept. One escape for the conflicted person overwhelmed by intense feelings is to regress and experience a psychosis.

Other women, such as the one in case 2, will become angry when a desired person does not make a sexual advance. To repair the narcissistic injury of not being sexually pursued, they may resort to pseudologia fantastica, or a wish-fulfilling delusion. This may reach such intensity that regression and psychosis rapidly follow.

We have found that in suspected cases of hysterical psychosis, it is important to inquire whether a sexual advance was made shortly before the onset of the psychosis. If no sexual advance was made, it is important to try to establish whether the woman felt that an advance should have been made. This information can be most useful in establishing a correct diagnosis of hysterical psychosis.

It should be recognized that hysterical psychosis is not limited to persons with hysterical personality. In patients with other personality types, the precipitant of a hysterical psychosis may be emotions of a nonsexual nature. We believe that our findings apply to those persons with hysterical personality organization.

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Organic Personality Disturbance: A Case of Apparent Atypical Cyclic Affective Disorder

BY CARLA HELLEKSON, M.D., ROBERT BUCKLAND, M.D., AND TREVOR PRICE, M.D.

The authors present the case report of a 19-year-old woman that contains several important points in the diagnosis and management of organic personality disturbance associated with temporal lobe epilepsy. The behavioral changes may antedate the onset of the clinical seizure. The diagnosis of complex partial seizures is a clinical diagnosis in which surface EEGs may repeatedly not demonstrate focal activity. Carbamazepine can have important psychotropic effects, in addition to its well-established anticonvulsant effects, and it may be particularly indicated if a lithium-resistant bipolar affective disorder is a differential diagnostic possibility.

A RENEWED INTEREST in the psychiatric aspects of temporal lobe epilepsy, with Waxman and Geschwind's description of the interictal personality syndrome (1) and work on the characteristics of interictal behavior in temporal lobe epilepsy by Bear and Fedio (2) and Blumer and Benson (3, 4), has led to the inclusion of organic personality disturbance in the proposed *DSM-III* classification of organic mental disorders. The following case presents several unusual features that are necessary to bear in mind in order to correctly diagnose and effectively manage this disorder.

CASE REPORT

Ms. A, a 19-year-old college student, had apparently been well adjusted until August 1976, when she rather suddenly became irritable, preoccupied, slept less, and by her own report, "wasn't aware . . . like in a fog or in a dream." In addition, she began talking about being evil and worthless, and on one occasion while at a wedding she blurted out the words, "I almost committed suicide." She said these words "popped into my head." She said of the wedding, "It was like a religious experience."

The patient's parents described her as alternating between "highs and lows." For several days she would be friendly and talkative and then for several days she was irritable, withdrawn, self-deprecatory, and preoccupied with religion and the devil. Treatment for a presumed depression was begun with desipramine hydrochloride, 50 mg three times a day, without significant improvement. Four months after her behavioral changes had first been noted, she experienced her first generalized tonic-clonic seizure. Subsequently she was admitted to our medical center from a local hospital with a chief complaint of, "I've been mean." She had gained several pounds and was occasionally bothered by insomnia. There was no hypergraphia, and her sexual interests were limited. She had no history of head trauma, febrile seizures, or prior psychiatric disturbances. Family history revealed the suicide of her brother when she was 5 years old and suicide of a maternal aunt.

Ms. A was a tall, athletic woman who was hyperactive, pacing, and combative when approached. Her speech was pressured but coherent. Her mood was irritable, and her affect was detached and suspicious. Her thought content centered on self-deprecatory comments such as, "I need to be locked up because I did something wrong—I lost my judgment." No hallucinations were noted, but she made delusional comments such as, "I'm clinically dead now; no brain waves, no blood pressure, no heart." Illusions were not uncommon, i.e., in speaking of the nearby church bells, she said, "I remember when I heard bells ringing in the middle of the night; I thought it was Judgment Day." Her orientation was intact. The findings from a physical examination, including a detailed neurological examination, neuropsychological testing, laboratory work with metabolic screen, skull films, computerized tomographic scan, and lumbar puncture were within normal limits. Two EEGs done on admission, the second with nasopharyngeal leads, were slightly abnormal due to some slowing of the background rhythm with diffuse excess of fast and slow frequencies; no consistent focal features were observed.

Over the next 8 months spanning several admissions, the patient's cyclic pattern of "highs and lows" continued with the periodicity progressively shortening from 6 to 2 weeks. During her "lows" she was withdrawn, preoccupied, and occasionally angry, with a driven verbal stream of guilty self-condemnation and religious concern. In retrospect, she described these times as, "I didn't know where I was; it didn't seem real. I woke up and thought I was in Hell or something." Once, while at home, she attempted suicide by wrist slashing and drowning. During her "high" periods she was friendly, talkative, even socially intrusive, with many changes of clothing in a day and flamboyant use of makeup. For short periods in between these extremes, she was a warm and cooperative young woman interested in athletics and school. Extended trials of lithium carbonate in dosages up to 1800 mg/day, with blood levels of 1.2 to 1.4 mEq/liter, and amitriptyline in dosages up to 300 mg at bedtime

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led to no improvement. Imipramine in dosages up to 200 mg at bedtime was also ineffective. A trial of perphenazine, 64 mg/day, did not help.

In June 1977 the patient experienced a second generalized seizure, and treatment with phenytoin, 300 mg daily, was started. The seizure was thought to have been related to high-dose tricyclic antidepressants. No further seizure activity was noted until the evening of July 22, 1977, when she walked up to the nursing station, appeared detached, and automatically stuffed several cellophane-wrapped sour balls into her mouth. She was led back to her room, where a generalized tonic-clonic seizure ensued. She denied premonitory sensations or olfactory/gustatory aura. The level of phenytoin was 15 mg/liter. An EEG with nasopharyngeal leads, the fifth since the onset of her symptoms, was slightly abnormal, with no lateralization of spike activity. The above episode was considered to be a complex partial seizure that generalized. One week after the institution of carbamazepine, 400 mg daily, her behavior stabilized dramatically. She was discharged in September 1977 on 300 mg of phenytoin and 400 mg of carbamazepine daily.

In the 16 months since discharge she has continued to report stabilized mood and has returned to her excellent level of premorbid functioning. This remission of symptoms has continued with carbamazepine alone since the discontinuation of phenytoin 5 months ago. No further seizure activity has been noted.

DISCUSSION

This case illustrates three important points in the diagnosis and management of *DSM-III*'s organic personality disturbance secondary to temporal lobe epilepsy. First, the behavior changes may antedate the onset of the clinical seizure disorder. Second, the diagnosis of complex partial seizures is a clinical diagnosis in which surface EEGs may repeatedly not demonstrate focal activity. Third, carbamazepine can have important psychotropic effects in addition to its well-established anticonvulsant properties when used in the management of patients with behavioral abnormalities associated with complex partial seizures.

The behavior changes described above began abruptly 4 months before the onset of generalized seizures, with periodic fluctuations between highs and lows. Although behavior changes antedating seizures have been reported (4), most accounts, including Slater and Beard's study (5), have described the slow development of the characteristic traits in the years after the onset of seizures. In our patient only three seizures occurred during the 12-month period of behavioral disturbance. This is in keeping with the trend suggested in Flor-Henry's retrospective study (6), in which the periodic or manic-depressive manifestations of temporal lobe epilepsy were more likely to be associated with infrequent generalized seizures than the schizophrenic-like psychoses.

Although organic personality disturbance was entertained in the differential diagnosis from the outset, the patient's disordered behavior was prematurely diagnosed as either schizo-affective disease or bipolar affective disorder with atypical features because of its superficial similarity to these functional disorders. The diagnosis was further delayed by the appearance of generalized seizures, which were thought to represent idiopathic epilepsy iatrogenically unmasked by high doses of tricyclic antidepressants and neuroleptics. During the patient's 12-month clinical course, she underwent extensive medical and neuropsychological evaluations, including five nonspecific EEGs (two with nasopharyngeal leads). Multiple adequate but unsuccessful clinical trials of lithium carbonate, antipsychotics, and tricyclic antidepressants, alone and in various combinations, and phenytoin were unsuccessful.

The probable diagnosis of temporal lobe epilepsy with interictal behavioral disturbances became apparent only with the nurses' observation of automatic, stereotyped behavior preceding the third generalized seizure. Based on these observations, carbamazepine was started, with dramatic and immediate improvement in the behavioral disorder that had continued over the subsequent 16 months of follow-up. This case emphasizes that the diagnosis of temporal lobe epilepsy and its related organic personality disturbance is a clinical diagnosis based on history and course. Multiple EEGs may fail to reveal specific abnormalities. Blumer (3) has outlined excellent guidelines for diagnosis of this disease.

The role of carbamazepine as an effective anticonvulsant in complex partial seizures is well accepted, while controversy continues concerning its possible behavioral effects (7). This case demonstrates a striking behavioral response, with stabilization of affective swings, modulation of maladaptive behaviors, and a return to the excellent premorbid level of functioning. It cannot be definitively determined whether this represents a response to carbamazepine of an atypical bipolar mood disorder that is lithium resistant (8, 9) or of temporal lobe epilepsy with an associated organic personality disturbance; however, the latter seems more likely.

What appears to be most important clinically is the fact that an earlier trial with carbamazepine was not considered. In retrospect, clinical features that might have suggested this are worth emphasizing. They include 1) the sudden onset of episodic and fluctuating behavioral symptoms, particularly if associated with a seizure disorder and a good premorbid history; 2) the nonresponse of a presumed cyclic mood disorder to lithium carbonate; and 3) symptoms specifically suggestive of temporal lobe epilepsy, such as feelings of unreality, alternating periods of appropriate interpersonal relating and social withdrawal, driven speech, forced thoughts, and ideational viscosity in the form of persevering religious and self-deprecatory preoccupations.

In conclusion, a clinical trial of carbamazepine may well be indicated when the symptoms of an organic personality disturbance are present and temporal lobe epilepsy is a diagnostic consideration, even in the absence of definite complex partial seizures or a diagnos-

tic EEG, particularly if lithium-resistant bipolar affective disorder is a differential diagnostic possibility.

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CLINICAL AND RESEARCH REPORTS

This section contains 1) new research findings, including preliminary data from pilot studies, either clinical or laboratory; 2) worthwhile replication studies; 3) case reports that describe a truly new syndrome or cast new light on established ones; and 4) case reports that indicate a new therapeutic procedure of potential value or call attention to adverse effects of drugs or previously unreported complications of therapeutic interventions. Program descriptions and literature reviews cannot be printed in this section. Criteria for format are listed in "Information for Contributors" in each issue; papers that do not adhere to these criteria will be returned to the author.

Effects of Lithium Therapy on MAO in Manic-Depressive Illness

BY WADE H. BERRETTINI, M.D., PH.D., WOLFGANG H. VOGEL, PH.D., AND ROBIN K. LADMAN

Several investigators have evaluated platelet monoamine oxidase (MAO) in bipolar affective illness (1–5). Three of these reports indicated that platelet MAO activity is significantly lower in patients with bipolar illness than in control subjects (1–3). However, two other reports indicated increased platelet MAO activity in manic-depression (4, 5). A study of electrophoretic patterns of platelet MAO revealed no differences between manic-depressive and control subjects (6). The origins of these discrepant results are unclear. Investigators on both sides of this issue have used very similar radioenzymatic assays with the same substrate (tryptamine) and have based the specific activity on platelet protein (1–3, 5). Of course, differences in control and subject populations, which could result from the use of different diagnostic criteria, may account for the conflicting results. Another possibility is that bipolar affective illness may be a heterogeneous disorder with no common genetic factor by which abnormal platelet MAO activity might be determined.

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The authors thank Robert Stolberg-Acosta, M.D., Robert Jones, M.D., and Jeffrey J. Dekret, M.D., Department of Psychiatry, Jefferson Medical College; Ms. Rachael Clark, R.N., and the staff of 14 Thompson; and Ms. Linda McComas, R.N., and the staff of 12 Thompson, Thomas Jefferson University Hospital, for their assistance. They also thank the patients and volunteers who made this study possible.

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All of the existing studies of platelet MAO in bipolar affective illness have used substrates of questionable physiologic importance, such as benzylamine (4, 5) and tryptamine (1–5). These studies are limited because platelet MAO activity was measured at one substrate concentration, which provides little kinetic information.

Previous work in our laboratory indicates that platelet MAO is kinetically different in chronic schizophrenics and controls (7). This kinetic difference is characterized by a decreased maximal velocity (V_{max}) and Michaelis constant (K_m). This result is consistent with uncompetitive inhibition. We decided to determine whether a kinetic study of platelet MAO in manic-depressive illness would yield similar kinetic results, i.e., decreased V_{max} and decreased K_m. If similar kinetic results are obtained in manic-depressive illness, it would be less likely that the finding in schizophrenia is related specifically to that disease in a genetic or causal manner, particularly in view of the apparent lack of common genetic factors in schizophrenia and affective disorders (3).

We decided also to determine the kinetic effect of lithium treatment on platelet MAO among individuals with manic-depressive illness. Our interest in this study stems from two sources. It has been reported that lithium treatment increases platelet MAO activity in vivo but that addition of lithium carbonate to platelet preparations in vitro had no effect (8). Unfortunately, this study provided no control platelet MAO values with which one might compare the values from the manic-depressive subjects. In addition, Sullivan and associates (2) have reported that normal platelet MAO activity in a bipolar patient is predictive of good response to lithium treatment (2). They found that lithium responders as a group had normal MAO activity,

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TABLE 1
Platelet MAO Activity in Manic-Depressive and Control Subjects

Group and Patient Number	Age (years)	Sex	Duration of Illness (years)	Kinetic Values: Baseline V_{max}[a]	Baseline $K_m \times 10^{-4}$	During Lithium Therapy: V_{max}[a] N	V_{max}[a] Mean	V_{max}[a] SD	$K_m \times 10^{-4}$ Mean	$K_m \times 10^{-4}$ SD
Subjects										
1	56	F	31	21	5.9	6	25	9	6.1	2
2	29	F	7	16	4.8	4	12	2	3.2	.6
3	31	M	4	10	2.4					
4	26	M	5	12	4.2	6	11	1	3.7	.7
5	21	M	2	18	4.0					
6	36	F	6	10	3.6	4	11	1	2.4	1
7	33	F	2	16	4.3	3	22	2	5.5	.3
8	62	F	22	33	5.3	—	—	—	—	—
9	58	F	16	31	4.4	—	—	—	—	—
10	62	F	20	36	5.0	4	35	4	4.8	.7
11	46	F	17	33	3.2	—	—	—	—	—
12	21	M	2	14	2.3	—	—	—	—	—
13	67	F	29	25	5.8	—	—	—	—	—
Mean	42			21	4.2		19		4.3	
SD				9.4	1.2		10		1.4	
Controls										
1	36	F		18	7.0					
2	54	F		11	5.9					
3	49	M		11	3.6					
4	24	M		20	4.3					
5	28	M		16	6.2					
6	29	F		18	6.9					
7	34	M		15	5.0					
8	36	F		18	3.7					
9	63	F		20	3.3					
10	41	F		15	2.4					
11	68	F		20	3.3					
12	22	M		19	4.2					
13	30	F		9	3.0					
Mean	39			16	4.4					
SD				3.8	1.5					

[a] V_{max} is expressed in nanomoles of dopamine per milligram of protein per hour.

while nonresponders had significantly less MAO activity than either the responders or the control group. This study indicates some degree of heterogeneity among individuals with bipolar affective disorders.

Method

Patients selected for this study were drawn from two acute care inpatient units at Thomas Jefferson University Hospital. All patients except numbers 4 and 8 were admitted for a manic exacerbation (see table 1), and all had been hospitalized at least once for depression and once for mania. None of the patients had taken lithium for at least 1 month before hospitalization. All patients except one (number 4) were treated with antipsychotic medication throughout the first 2 weeks of hospitalization. The duration of illness, defined as the number of years since the initial hospitalization, ranged from 2 to 32 years. All patients were free of other major illnesses and had normal complete blood counts, including platelet estimates. Informed consent was obtained from controls and patients. Baseline blood samples were obtained from all patients before they started lithium therapy. Thereafter blood samples were obtained periodically (at the same time as blood lithium samples were drawn) at least 10 hours after the last lithium dose. All patients satisfied the Research Diagnostic Criteria (9) for bipolar affective disorder.

The details of platelet MAO enzyme preparation and assay are described in full elsewhere (10). Controls were unit staff and laboratory personnel who were drug-free and had no personal or family history of psychiatric illness.

Results

The results (table 1) show that V_{max} and K_m did not differ significantly between manic-depressive patients and controls. Further, treatment with lithium did not significantly alter V_{max} or K_m values among these patients.

Discussion

In terms of platelet MAO activity, manic-depressive patients appear to be a very heterogeneous group. In this small series, several patients had high activity and 2 had rather low activity; the rest fell within 1.5 SD of

the mean activity for the control group. It seems clear that there is a subgroup of patients with very high activity. In our series, such patients were older women, but it is unlikely that this fact alone can account for this result. Several older women in our control group had normal platelet MAO activity. We were unable to determine any clinical measure that separates the high-MAO subgroup from the other subjects.

Our finding that lithium therapy does not significantly affect platelet MAO contrasts with the report of Bockar and associates, who reported increased platelet MAO activity during lithium treatment (8). There appeared to be very little variation in V_{max} values over a wide range of lithium levels, with the exception of patient 1, who showed a rather large fluctuation in platelet MAO activity. The patients, regardless of pretreatment platelet MAO activity, retained essentially the same activity during several weeks when therapeutic lithium plasma levels were maintained.

The previous report of increased platelet MAO activity during lithium treatment (8) is provocative because it provides an explanation of the efficacy of lithium in mania that agrees with the catecholamine hypothesis of affective disorders. An accumulation of catecholamines, causing mania, would be reversed by lithium via its effect on MAO. Unfortunately, we have been unable to duplicate these results.

These results do offer partial support to reports of both increased (4, 5) and decreased (1–3) platelet MAO activity in manic-depressive illness. In our series, there are patients with both high and low platelet MAO activity. Certainly, differences in patient populations, diagnostic criteria and enzymatic procedures could account for these results. It does seem that our group of patients may be more heterogeneous, at least biochemically, than other groups.

Perhaps the most interesting finding in this study is the subgroup of patients with high platelet MAO activity. The identification of biochemically (and/or genetically) distinct subgroups in major psychiatric illnesses would almost certainly lead to a more comprehensive, complex, and accurate conception of these diagnostic categories.

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Micrographia and Akinesia

BY DANIEL R. SHACKMAN, M.D., THEODORE VAN PUTTEN, M.D., AND PHILIP R.A. MAY, M.D.

Part of the art of antipsychotic drug therapy is distinguishing side effects from the vicissitudes of schizophrenia. In particular, mild akinesia is evident primarily as a behavioral state of diminished spontaneity characterized by few gestures, unspontaneous speech, apathy, and difficulty initiating usual activities (1). Akinesia is insidious and, particularly when superimposed on chronic schizophrenia, can worsen such negative symptoms as blunting of affect and emotional withdrawal (2). The problem is that akinesia is easily confused with depression, demoralization, or the negative symptoms of schizophrenia, and even experts at times cannot distinguish between a depressive mood shift and an antipsychotic-induced akinesia (3). What is needed is some reliable and readily observable clinical correlate of mild akinesia.

Constriction of handwriting (micrographia) has been reported to be a sensitive indicator of extrapyramidal involvement (4–6). According to Haase and Janssen (4) and Angus and Simpson (6), constriction of handwriting precedes observable drug-induced parkinsonism. This notion has gained some measure of acceptance but has seldom been rigorously examined. We conducted the study reported in this paper to determine whether constriction of handwriting is a reliable correlate of mild akinesia.

Method

Twenty-four newly admitted patients who met Feighner and associates' (7) criteria for schizophrenia received treatment with an antipsychotic drug (most commonly thiothixene) after a drug-free observation period of 1–21 days. The average duration of drug treatment was 43 days (SD=16.5 days).

The control group consisted of 7 nurses and secretaries who had no knowledge of the protocol.

Handwriting samples were collected at 9:00 a.m. before treatment started, and weekly thereafter, by a technician who was blind to the akinesia ratings. All subjects were asked to copy the nursery rhyme "Mary Had a Little Lamb" from a typed card three times, on unlined translucent paper, with the same ballpoint pen. (This procedure follows the technique used by Haase and Janssen [4].)

Three measures of handwriting were chosen for study: average word length (total length of lines divided by number of words), average area per word (total verse area divided by number of words), and representative letter height. The three measures were found to parallel each other closely within each verse and between the three verses in each sample.

Average word length varies slightly more than the other measurements, is easy to measure and calculate, and has been studied more often than the other measures, so we chose this as the final criterion of micrographia.

In 3 cases, the baseline handwriting was so large, unruly, and bizarre ("psychotic handwriting") that it was not measurable. For this reason, as well as the possibility that reduction in size of these handwriting samples would more likely be due to the antipsychotic effect of the drug than to an extrapyramidal micrographic effect, these patients were excluded from the study.

Akinesia ratings were made independently by one of the authors on an extrapyramidal symptom rating scale (8). The akinesia scale was modified as follows: 0=none; 1=mild (subjectively the patient feels "slowed up" *and* objectively there is a subtle motoric slowing, patients walk less briskly, arm swing is somewhat reduced, face conveys less expression, spontaneous gestures and movements are reduced); 2=moderate; and 3=severe (loss of facial expression, arm swing markedly reduced, and patient moves and walks very slowly).

Results

During the course of treatment with antipsychotic medication, 10 of the 21 patients developed akinesia (9 mild, 1 moderate).

Before drug therapy there was no significant difference in handwriting size between those patients who eventually developed akinesia (mean word length=2.13 mm, SD=.36), those who did not develop akinesia (mean=2.27 mm, SD=.52), and the control group (mean=2.16 mm, SD=.30). When the akinetic patients developed this symptom, their handwriting constricted to a statistically significant degree (mean=1.93 mm, SD=.31, $t=2.457$, $p<.05$). However, the nonakinetic patients' handwriting constricted just as much at some time during their course of treatment ($2.05\pm.56$, $t=4.115$, $p<.01$), as did that of the control group ($1.95\pm.25$, $t=6.8754$, $p<.001$).

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The handwriting of 7 of the 10 patients who became akinetic showed constriction. In 1 case handwriting size remained unchanged, and in 2 cases the handwriting actually expanded. However, 10 of the 11 nonakinetic patients' handwriting constricted at some time during the course of drug treatment. Further, a group of control subjects demonstrated the same degree of handwriting constriction at some point during an equivalent period of study.

Discussion

Our study demonstrated that the handwriting of a group of medicated schizophrenics tended to constrict when these patients became mildly akinetic. However, when examined in a rigorous prospective design, handwriting constricted just as much at some time in a group of nonakinetic medicated schizophrenics and in a group of normal controls. This observation is in accord with those of Rifkin and associates (3), who found that change in size of the area of a copied paragraph did not distinguish between those patients with mild akinesia and those without. We conclude that micrographia is not a reliable correlate of mild akinesia.

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Use of Propranolol in the Treatment of Postencephalitic Psychosis

BY HERBERT A. SCHREIER, M.D.

Propranolol, a β-adrenergic blocking agent, has been used extensively in medicine, particularly in cardiology. Recently, reports have appeared of this drug's efficacy in such diverse conditions as anxiety (1, 2), migraine (3, 4), and chronic drug-refractory schizophrenia (5). Elliott (6) reported on its effectiveness in controlling irritability and/or "episodic belligerence" that occur after brain injury from trauma or anoxia. The following is a case report of the successful treatment with propranolol of a 12-year-old boy who developed a psychosis during an acute encephalopathy of unknown etiology.

Case Report

John is a 12-year-old boy who was admitted to Children's Hospital Medical Center (CHMC) in a fluctuating state of consciousness. Eleven days before his admission he had started having severe headaches that were not relieved by aspirin. Three days before his admission he had a grand mal seizure and was admitted to a local hospital, where he was described as alert and afebrile postictally. Results of general physical and neurologic examinations were normal. A lumbar puncture at that time showed protein of 36 mg/100 ml, glucose of 56 mg/100 ml, a white count of 180 (mostly lymphocytes), and a normal blood count and urinalysis. He also had a normal skull series and cinetomography brain scan. He was treated with diphenylhydantoin, 200 mg/day, and phenobarbital, 120 mg/day, and was transferred to CHMC on the 4th hospital day when he became disoriented to time and place.

On admission, John was afebrile and had normal vital signs. He was alert, watched people, and was able to follow simple commands after only a few seconds' delay, but he would not speak or answer questions. A neurologic exam revealed asymmetric deep-tendon reflexes at the knees, the right being brisker than the left, a few beats of clonus at both ankles, and bilateral Babinski reflexes. He continued to be disoriented and had occasional episodes of lethargy, alternating with episodes of extreme agitation. During the latter episodes he used obscene language with marked sexual overtones, which was described as atypical behavior for him. On his 2nd day at CHMC he had a second grand mal seizure. A repeat lumbar puncture revealed 370 white cells, 100% lymphocytes, 60 red blood cells, a total protein of 18 mg/100 ml, and a glucose of 79 mg/100 ml. CSF analysis, including fungal, tuberculosis, viral, bacterial, and cytological studies, was negative. John continued to have episodes of lethargy alternating with delirious agitation, and on the 3rd hospital day he exhibited clinical signs of increased intracranial pressure, requiring the use of mannitol. He was also started on dexamethasone sodium phosphate, 4 mg I.V. every 6 hours.

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An EEG performed on the 2nd day of hospitalization showed an excess of slow activity, with somewhat higher amplitudes on the left side. His agitation was not controlled by chloralhydrate and/or chlorpromazine.

On the 17th hospital day John suddenly seemed alert, with an appropriate mental status, but 3 days later he again exhibited periods of disorientation, agitation, and lethargy. Steroids were discontinued, with no benefit. On the 20th day the patient became alert and oriented but started hearing voices and reported seeing bugs in his bed. At times he was verbally abusive (in marked contrast to his premorbid functioning), and he attempted to flee the hospital several times. Propranolol, 20 mg b.i.d., was begun and increased over the next 2 days to 100 mg/day. At that dose John had no further episodes of agitated behavior, was alert and oriented, without hallucinations. He was described by his grandfather as being regressed in his behavior, acting more like a sibling who was 8 years old. He was discharged on the 25th day on diphenylhydantoin, 300 mg/day, propranolol, 100 mg/day, and chlorpromazine, 100 mg at bedtime as needed.

At home, John continued to exhibit regressed behavior (e.g., asking for toys of the type he had not played with in years) and exhibited clinging behavior toward his grandfather and some increased sibling rivalry. Over the next 2 weeks there were no aggressive incidents or indications of psychosis. The dosage of propranolol was decreased over several days. Chlorpromazine had not been given for over a week. The day after his last dose of propranolol, John became irrational and belligerent while shopping with his grandfather and had to be taken home. At home, he became increasingly aggressive, wrecking his room, scattering and tearing clothes, and breaking pictures. Propranolol, 100 mg/day, was resumed and no further incidents of this nature occurred. One month later, John was showing less regressive behavior and no evidence of the former belligerence or psychosis so propranolol was again tapered, without further incident. A year later he continues to do well and has regained his former level of functioning at school and in his social relations. The diagnosis was encephalitis, probably of viral origin, with an organic psychosis.

Discussion

Propranolol has been shown to be useful in treating atypically belligerent behavior in some patients after concussions and anoxia. This case probably represents the successful treatment of a psychosis with belligerent behavior that developed in a 12-year-old boy after an encephalopathy of infectious origin. In Elliott's report (6) the patients with belligerent behavior did not respond to diphenylhydantoin or a variety of sedatives, benzodiazepines, or, in one case, phenothiazines. In some of his cases, the belligerence recurred when propranolol therapy was discontinued after several weeks. In that group of patients, symptoms were relieved after administration of propranolol was resumed.

Propranolol's mechanism of action on the central nervous system is poorly understood. It appears to have effects on mood and behavior that are not related to its β-adrenergic blocking potential. Caution should be observed in patients with cardiac problems, asthma, pulmonary disease, or diabetes (7).

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A Double-Blind Evaluation of Nomifensine and Imipramine in Depressed Outpatients

BY AUSTIN MCCAWLEY, M.D.

Nomifensine maleate has been effective as an antidepressant in a number of clinical studies conducted in this country and in Europe (1, 2). The drug has properties similar to the tricyclic antidepressants but is not identical to them; its chemical structure appears to be intermediate between imipramine and methylphenidate. It has an early onset of action, little anticholinergic activity, and, in the animal studies, no addictive potential. Nomifensine maleate is 8-amino-1,2,3,4-tetrahydro-2-methyl-4-phenylisoquinoline maleate. This study compared nomifensine maleate with imipramine HCl in 20 depressed outpatients.

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Method

Patients were selected for the study on the basis of the following criteria: 1) evidence of primary affective disorder (depression) in accord with a predetermined checklist, 2) age between 18 and 65, 3) a minimum baseline total score of 20 on the Hamilton Depression Scale with at least a score of 2 for depressed mood, 4)

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a total rating of 14 on the Beck Depression Inventory, 5) onset of symptoms at least 4 weeks before entry into the study, and 6) illness duration less than 6 months. Patients were excluded for a number of reasons, including medical illness, pregnancy, the need to take other medication, and chronic abuse of alcohol or drugs.

Two groups of patients, 13 of whom received nomifensine and 7 imipramine, were studied in a double-blind parallel group fashion established by random assignment. Patients who were already taking psychotropic medication were given a placebo washout for 7 days before starting the study medication. The dosage regimen for both drugs was 50 mg b.i.d. for 1 week, 50 mg t.i.d. for 1 week, and then 200 mg/day if the symptoms had not remitted. The duration of the study was 28 days, but some patients were continued for periods up to 6 months. Statistical calculations for this report are made on the response after 28 days. In the interest of brevity, the extensive evaluations done at baseline will not be detailed. The instruments used for evaluation in this report are the Hamilton Depression Scale, done at weekly intervals, and the Clinical Global Impression Scale, done at baseline, day 14, and day 28. At each weekly evaluation, the patient completed the Hopkins Symptom Check List.

All patients received a physical examination and extensive laboratory tests, including SMA/12, CBC, urinalysis, and ECG at baseline and day 28. Vital signs were recorded weekly.

Results

Nineteen patients completed the study. The mean Hamilton Scale scores of the two groups before treatment were almost identical (25.92 and 26 for nomifensine and imipramine, respectively), as were the mean difference scores of the group (14.85 and 14.5).

When the samples were combined to determine whether the group as a whole had improved significantly, the mean difference score was found to be 14.74 (SE=1.41); this was significant ($t=10.469$, $p<.001$).

Patients were rated by the physician as very much improved, much improved, minimally improved, or unchanged. Chi-square analysis of these ratings agreed with the Hamilton Depression Scale scores in indicating that imipramine and nomifensine were equally effective.

At the onset of the study no attempt was made to distinguish between endogenous and reactive depressions. In some cases, this would have been difficult. However, there was a group of 11 patients (3 on imipramine and 8 on nomifensine) with unusually difficult situational or interpersonal problems in whom the depression appeared to be reactive. These two small groups were compared, with interesting results. It appeared that the initial mean scores of the groups did not differ from each other or from the mean score of the larger group. Following treatment, however, the scores of the two groups differed significantly in favor of the nomifensine group ($t=3.88$, $df=9$, $p<.05$). A Wilcoxon test did not reach this significance level but did tend to confirm that nomifensine was superior to imipramine in the treatment of these reactive depressions. The small size of the groups and the fact that this analysis was retrospective do not allow one to draw conclusions, but this observation would appear to merit further study.

Side Effects

One patient was withdrawn from the study after she developed a syndrome of inappropriate antidiuretic hormone secretion. She had been a very unreliable subject, had taken only 50 mg/day of nomifensine, and was about to be withdrawn from the study for this reason. However, there were a number of capsules unaccounted for in the medication she returned, which raised the (unresolved) question of whether she took an overdose. This syndrome has been reported in psychiatric patients as a side effect of several drugs and also in unmedicated patients (3). Therefore, there is some question whether this was in fact a side effect of nomifensine; it has not been reported in any of the considerable number of other studies now completed.

All other side effects were mild, transient, and easily managed. Nausea, headaches, rapid pulse, and flushing were each reported by two patients. Paresthesia in the legs, palpitations, faintness, a metallic taste, and a slight high were each reported by one patient. Four patients on nomifensine had no side effects whatever, whereas all of the patients on imipramine had at least one side effect. Insomnia was not reported by the patients on nomifensine. A number of slightly elevated laboratory tests were observed, but they had no clinical significance and followed no significant pattern.

Conclusions

The results of this study indicate that nomifensine is as effective as imipramine in treating depression. There were some indications that it may be more effective in reactive depressions.

Many of these patients were followed beyond the 28-day study period. They sustained their improvement, and one of the patients on nomifensine who was reported to be unimproved on day 28 showed a dramatic improvement after 6 weeks. For several reasons this patient had reached the maximum dose of 200 mg later in the course of treatment. In nearly all of the patients who were followed, the treatment evolved from pharmacotherapy to a more psychotherapeutic form of treatment. There were numerous psychological issues that had to be dealt with in psychotherapy, and in this study, as in others, it became clear that depression is not simply a biochemical process.

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Lithium Treatment in Older Patients

BY STEVEN P. ROOSE, M.D., STANLEY BONE, M.D., CATHERINE HAIDORFER, R.N., DAVID L. DUNNER, M.D., AND RONALD R. FIEVE, M.D.

The purpose of this paper is to describe our experience with maintenance lithium therapy in patients over the age of 60. As the indications for the use of maintenance lithium therapy broaden, clinicians will encounter a greater number of older patients who are taking lithium or beginning lithium treatment. There is a paucity of clinical data specifically focused on medical problems related to lithium treatment in patients over 60 (1, 2). We will report assessments of the medical status of 31 patients over the age of 60 who are in active treatment in the Lithium Clinic of the New York State Psychiatric Institute.

Method

All patients over the age of 60 in the Lithium Clinic filled out a questionnaire that surveyed side effects, medical history (before and after lithium treatment), and current indications of acute or chronic medical problems. In addition, each patient was interviewed individually to supplement or clarify responses to the questionnaire. All patients met the diagnostic criteria of Feighner and associates (3) for primary affective disorder and gave written informed consent to participate in the study. In addition to the questionnaire, all patients had had a complete physical examination, complete blood count, blood chemistries, and electrolyte studies within the past year. A 12-level ECG was performed on all patients in the study and read by an attending cardiologist.

Results

Patients ranged in age from 60 to 79 years, with a mean age of 67 years. They had been treated with lithium for 1 to 10 years.

The most unexpected result of this survey was that 9 out of 31 patients had no regular medical contact other than the Lithium Clinic. Thus, our subspecialty clinic was in fact serving a primary care function for about 30% of these patients.

The most frequent chronic medical problem was hypertension, which was reported in 20% of the patients. In all cases the hypertension was satisfactorily controlled with α-methyldopa, with no increase in depressive episodes. Arthritis and hypothyroidism were each reported by 10% of the patients, and in 1 case the hypothyroidism had developed shortly after lithium treatment was initiated.

There were 4 episodes of lithium toxicity in these 31 patients during an 18-month period. In the same time period there were only 2 cases of toxicity in 164 lithium-treated patients under 60. This difference is statistically significant ($\chi^2=8.32$, $p<.01$, Yates's correction applied). As reported elsewhere (4) two patients developed cardiac sinus node dysfunction that could be directly attributed to lithium therapy as demonstrated by Holter monitoring of cardiac rhythm both when the patients were taking lithium and when they were lithium-free.

Only 13 of the 31 patients had completely normal ECG tracings. Five patients had arrythmias—3 cases of sinus tachycardia (rate>100), 1 case of sinus bradycardia (rate<55), and 1 case with frequent atrial premature contractions. Fourteen patients had evidence of conduction defects—10 cases of incomplete atrial-ventricular block (PR interval>.20 seconds), 2 cases of right bundle branch block, and 2 cases of incomplete ventricular conduction defect.

Discussion

The patient over the age of 60 is prone to develop lithium toxicity, and it appears that these episodes can fulminate more rapidly and at lower serum lithium levels than those seen in younger lithium-treated patients. Consequently, we have tended to keep this elderly patient population at a somewhat lower maintenance lithium level (.6–.7 mEq/liter), and to date we have not noted an increased number of affective episodes, which would indicate decreased prophylactic efficiency. Whether this lithium toxicity diathesis is a reflection of an increased sensitivity to lithium or of decreased renal clearance is not yet determined. In general, it appears that older patients can be safely

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maintained on lithium despite various medical problems and a proneness to develop toxicity. Such patients' lithium levels should be closely monitored, and they should be followed by a primary care physician, with routine physical examinations, ECGs, blood chemistries, and evaluations of kidney and thyroid function. The fact that 30% of our patients had no primary care physician is distressing.

The ECG results initially indicate no consistent pattern of abnormalities. However, the limitations of a routine ECG and the small number of subjects make it inadvisable to draw conclusions about the cardiac effects of lithium from these data. Sinus node dysfunction is one proven side effect of lithium treatment, but whether there are others remains to be seen. It is clear that larger, prospective studies of the effects of lithium on cardiac function are necessary.

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Behavioral Treatment for Raynaud's Disease: A Comparative Study with Long-Term Follow-Up

BY ALAN M. JACOBSON, M.D., THEO C. MANSCHRECK, M.D., AND ELIZABETH SILVERBERG, PH.D.

Biofeedback and relaxation techniques are being evaluated as possible treatments for many psychosomatic conditions, including Raynaud's disease, which causes considerable pain and discomfort in the fingers and toes after exposure to even mildly cool temperatures. Behavioral treatment for Raynaud's disease has focused on teaching patients to warm digits to abort or prevent painful episodes. Previous case reports (1, 2) and uncontrolled outcome studies (3) have suggested that an approach which combines biofeedback and relaxation in an individually tailored program can be clinically beneficial in the treatment of this disorder.

Recent studies of hypertension (4) and migraine headaches (5) have suggested that relaxation alone may be as effective as a combined biofeedback/relaxation approach. Because there have been no comparisons of these two approaches in Raynaud's disease, we decided to examine the benefit of relaxation and finger temperature biofeedback in patients with clinically significant Raynaud's disease.

Method

We formulated 3 specific hypotheses:

1. Finger warming would be greater in patients using biofeedback and relaxation than those using relaxation only.
2. There would be more clinical improvement in the combined-treatment patients than the relaxation-only patients.
3. Clinical improvement would be associated with finger warming demonstrated in laboratory sessions.

The subjects (N=12) were selected on the basis of the following criteria: 1) a diagnosis of currently symptomatic Raynaud's disease, 2) absence of concurrent collagen vascular disease, 3) at least a two-year history of symptoms, 4) no prior sympathectomy or gangrenous changes in the digits. Informed consent was obtained from all patients after the procedures had been fully explained.

After an initial history-taking session, patients were randomly assigned to one of two treatment conditions, biofeedback and relaxation or relaxation only. These groups were not significantly different in age or duration of illness. All patients were first taught a simple relaxation technique (6) that required systematic muscle relaxation and focused attention in a single half-hour session. They were asked to practice this technique twice daily during the treatment period. The biofeedback group was also taught to use a temperature trainer with auditory and visual feedback.

The sessions, which consisted of two 5-minute trials, were given twice weekly for 6 weeks. During the trials the relaxation group practiced relaxation and the biofeedback group used the temperature trainer to warm the right forefinger.

Laboratory outcome was measured by the change in finger temperature during each trial. Forehead temperatures were recorded at the same time to control for general body warming. Clinical results were based on patients' ratings of symptom severity 1 month and 2

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TABLE 1
Patient Characteristics and Responses to Treatment

Subjects and Group	Sex	Age (years)	Duration of Illness (years)	Temperature Increases per Trial	Self-Rated Improvement[a]	
					One Month	Two Years
Biofeedback and relaxation						
1	F	42	15	0.4°C	3	3
2	F	25	3	1.0°C	2	3
3	F	47	10	0.7°C	3	2
4	F	27	3	0.3°C	1	1
5	M	47	2	0.1°C	3	3
6	F	40	6	0.3°C	3	2
Group mean		38	6.5	0.5°C	2.5	2.3
Relaxation alone						
7	M	29	3	2.0°C	3	3
8	F	25	7	0.9°C	2	3
9	F	30	2	0.3°C	3	3
10	M	31	14	1.6°C	3	3
11	F	32	4	0.6°C	1	1
12	F	30	15	3.2°C	3	—[b]
Group mean		29.5	7.5	1.4°C	2.5	2.6

[a]Ratings are on a 5-point scale, where 0=symptoms worse, 1=no change, 2=slight improvement, 3=marked improvement, and 4=asymptomatic.
[b]No follow-up.

years after treatment. Because all patients expected to receive biofeedback training, those in the relaxation-only group were offered biofeedback one month after the study period. Four of the 6 patients in this group eventually underwent biofeedback treatment.

Results

Both treatment groups demonstrated consistent finger temperature increases during biofeedback or relaxation trials (see table 1). Temperature increases were noted in 53 of 72 trials for the biofeedback group and in 62 of 72 trials for the relaxation group. However, the hypothesis that finger warming would be greater in the biofeedback group than the relaxation group was not supported. The relaxation group averaged a 1.4°C increase per trial while the biofeedback group demonstrated a 0.5°C increase per trial ($t=1.97$, $df=10$, $.05<p<.10$).

Our second hypothesis, that clinical improvement would be greater in the biofeedback group, was not supported. Five patients from each group reported a decrease in symptom severity at 1-month follow-up. Typically, patients tolerated cold better than before treatment and remarked that emotional stress was less likely to trigger symptoms. To our surprise, clinical benefit was not positively correlated with ability to warm fingers in the laboratory setting. For example, patient 1 was among the best responders clinically but showed below average finger-warming ability in the experimental sessions compared with others in the biofeedback group.

Long-term comparisons between the two groups were not possible because 4 of the 6 relaxation patients eventually received biofeedback training. At 2-year follow-up, 7 of the 11 patients contacted noted continued clinical benefit, characterized by less severe and less frequent episodes. This improvement was maintained regardless of the initial treatment regimen. Six of the 7 long-term responders noted that continued benefit was dependent on regular practice of relaxation exercises. For example, patient 7 found that symptom severity increased after only 2 days without practice. Interestingly, two other patients (patients 3 and 6), who reported little improvement at 2-year follow-up, said they had stopped regular relaxation practice because of "laziness" but found that relaxation on even an occasional basis lessened the severity of symptoms.

Comment

The most interesting finding of this study is that combined relaxation and biofeedback treatment appears to have not only short-term but long-term clinical benefit for patients with Raynaud's disease. This continued benefit seems to be dependent on regular practice of relaxation. Previous reports of behavioral approaches for treating Raynaud's disease provided only limited evidence for long-term benefit (1–3). In fact, few studies of biofeedback and relaxation treatments for other conditions have reported on the long-term benefit of these approaches.

Also of interest is the finding that treatment with relaxation alone matches the short-term benefit of biofeedback treatment. This is consistent with the finding of recent reports on the treatment of migraine headaches (5) and hypertension (4) but requires corroboration because the relative superiority of biofeedback may not be demonstrable on measures of laboratory change or short-term response. Rather, the value of biofeedback may be in the specific learning of a new response to stressful life events, which can only be assessed with long-term follow-up.

This study could be faulted for the lack of a no-treatment control group; placebo factors rather than the specific effects of biofeedback or relaxation might ac-

count for the reported benefit. However, there are two findings which suggest that the effects go beyond placebo factors. First, patients noted a regular relationship between relaxation practice and clinical benefit and when the practice stopped, clinical benefit ceased. Second, these practice-related benefits persisted long after the original treatment period. Certainly, further studies with both a control population and blind observers will be an important next step in these investigations. It does appear that a behavioral approach will provide an additional method for treating Raynaud's disease. This gives the psychiatrist new opportunities for treating patients who have conditions beyond the scope of traditional psychotherapeutic techniques.

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Appearance of Manic Psychosis Following Rapid Normalization of Thyroid Status

BY ALLAN M. JOSEPHSON, M.D., AND THOMAS B. MACKENZIE, M.D.

The appearance or exacerbation of psychiatric symptomatology early in the treatment of hypothyroidism is well documented (1). The possibility that the appearance and severity of these symptoms may depend on the speed with which thyroid status is normalized has been mentioned (2). However, a clinical course compatible with a manic psychosis has not been well described. We will describe a case in which mania was observed in a hypothyroid woman treated with thyroxine.

Case Report

Ms. A, a 34-year-old woman, was admitted to the medical service of the University Hospitals on April 10 after her family brought her to the emergency room because of progressive insomnia and agitation of 5 days' duration.[1] A psychiatric consultation was sought on the day of admission.

Four years earlier the patient had been admitted to a general hospital because of "agitation, insomnia, mild euphoria, and distractibility." She was discharged after 6 days with a diagnosis of "emotional lability and probable manic-depressive state." Thyroid indices and psychiatric consultation were not obtained. Three years before her admission, a diagnosis of Grave's disease was made after thyromegaly was noted; anxiety was her only symptom. The patient was given 44 mCi of ^{131}I four months before her admission.

On March 30, her tendon reflexes were noted to be moderately prolonged and thyroid studies revealed a hypothyroid state (T_4=1.6 μg/dl, T_3 uptake=0.79, thyroid index=1.3 μg/dl, TSH=133 μIu/ml). No psychiatric abnormality was described at that time. On April 1, her family noticed an increased interest in religious themes. As directed by her physicians, she took 150 μg of thyroxine daily on April 3, 4, 5, and 6. On April 5, she was described as alternately tearful and euphoric. She was ordinarily a "good listener," but friends now found her to be garrulous and unable to focus her thoughts. She discontinued the thyroxine on April 7 because she was "getting hyper." Over the next 3 days increasing preoccupation with religious concerns and progressive agitation and insomnia were noted.

Ms. A was taking no medications on admission and denied a history of drug use or excessive alcohol intake. There was no family history of endocrine or psychiatric disorder.

On admission positive physical findings included brisk reflexes, diaphoresis, and a pulse of 110. Ms. A was exophthalmic and complained of sleeplessness. Her motor activity was mildly retarded, with no unusual posturing or grimacing. Her affect fluctuated between tearfulness and elation and she demonstrated thought blocking and delusional thinking of a religious nature. She was oriented to time, place, and person.

Ms. A was started on 2 mg of haloperidol p.o. h.s. Thyroid indices at admission were normal (T_4=6.5 μg/dl, T_3 uptake=0.86, thyroid index=5.6 μg/dl) with TSH remaining elevated at 30 μIu/ml. Laboratory studies on admission, including plasma cortisol and urinary hydroxycorticosteroids, were within normal limits. An EEG obtained on April 17 revealed excessive and diffuse bitemporal slowing. A sleep-deprived EEG obtained on April 24 with NP leads showed no seizure activity.

She continued to show marked affective lability and on her third hospital day was transferred to the psychiatry service. She also continued to have insomnia and grandiose delusions, such as believing that "everyone on this ward is a doctor because I am a special patient." Concomitant cognitive examination showed no impairment. She was placed on loxapine succinate, 75 mg/day p.o., and simultaneously 150 μg of thyroxine was resumed at the recommendation of the

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[1]Dates have been changed to protect the patient's anonymity.

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endocrinology consultant. Within 3 days marked improvement of her insomnia and delusional thinking was noted. In view of the hypomanic quality of Ms. A's behavior, lithium carbonate, 1800 mg/day, was started 4 days before her discharge.

Ms. A was discharged 2 weeks after admission on a regimen of loxapine, 25 mg p.o. h.s., lithium carbonate, 1800 mg/day p.o., and thyroxine, 150 μg 4 days a week and 75 μg 3 days a week. Her lithium and loxapine were subsequently discontinued, and there was no return of psychotic symptomatology.

Discussion

This patient's insomnia, mood lability, grandiosity, garrulousness, and psychomotor agitation satisfied Research Diagnostic Criteria for mania (3). The appearance of these symptoms followed rapid normalization of her thyroid status (600 μg of thyroxine in a 4-day period). The progressive appearance of her manic symptomatology over a 7-day period is consistent with observations that peak physiologic activity of thyroxine occurs 7–10 days after administration (4). Her symptoms were readily controlled by neuroleptics and her clinical symptoms abated within 2 weeks. Withdrawal of psychotropic medication was accomplished uneventfully.

Although her mania occurred in the context of a rapid normalization of her thyroid status, the underlying neurobiologic mechanisms are unclear. Thyroid hormone is known to play a role in modulating catechol receptor sensitivity (5). Animal research has shown a decreased receptor sensitivity to CNS catecholamines in hypothyroidism, accompanied by a compensatory increase in catechol concentration (6, 7). If such an adjustment occurs in human subjects, then rapid administration of thyroxine, abruptly augmenting catechol receptor sensitivity, could precipitate a hypercatecholaminergic state. The catecholamine theory of affective disorder proposes that in susceptible individuals, such an excess of catechol activity is the neurochemical basis for mania (8). This patient's susceptibility to affective disturbance was suggested by her history of hospitalization for "probable manic-depression." We speculate that the rapid administration of thyroxine precipitated a manic episode in a person disposed to affective instability.

Should this patient's clinical course be regarded as an instance of secondary mania? In their recent review of this syndrome, Krauthammer and Klerman (9) did not mention thyroxine administration as an etiology. According to their criteria, this patient's history of affective disorder would militate against the diagnosis of secondary mania. However, her previous affective disturbance appeared within a year of the discovery of hyperthyroidism and may have been an organic affective syndrome. In support of the diagnosis of secondary mania are the late age of onset, close temporal proximity to administration of thyroxine, negative family history of psychiatric disorder, rapid resolution of the psychosis, and the diffusely abnormal EEG at the height of the symptoms. We suggest that alteration in thyroid status merits consideration as a possible antecedent to secondary mania.

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The "Wish Book" in Child Psychotherapy

BY JOEL A. MOSKOWITZ, M.D.

Mail order catalogs can serve as a unique window into the inner fantasy world of children in therapy. Slavson (1) stated that play therapy for young children was developed because of the inadequacy of language as a medium of expression. When the child is supplied with appropriate materials, he conveys symbolically his fantasies and preoccupations. Slavson refers to the use of toy guns, soldiers, darts, clay, dolls, and water. A more structured but highly evocative medium is the use of the mail order catalog to involve, stimulate, and benefit troubled children, including adolescents. Even in pioneer days it was well known that the Sears catalog, for example, opened for isolated settlers wide vistas of needed and wished for clothing, utensils, and building materials. A collection of catalogs in the therapist's office provides a new way to penetrate the initial silent barriers that often prevail and to elicit useful therapeutic data. In a sense, the world is a projective test. Opinions, attitudes, and feelings may be elicited by asking the patient to comment about anything he or she observes. Inquiring about the child's ideas relevant to something he or she finds interesting in a catalog is less threatening than posing more direct questions.

The catalog the child picks tells the therapist what the child likes. The therapist must actively focus the child's attention on the mail order catalog and will find it easy to engage the child in conversation about such items as model airplanes or the latest stereo equipment. There should be a wide selection of readily accessible catalogs. Catalogs from the major mail order department stores are obvious choices, but many specialty catalogs dealing with sports equipment, new homes, games, scientific instruments, radio equipment, books, magic, travel, schools, records, etc., are also available on written request.

The use of the mail order catalog is not a separate form of therapy. It is a clinical tool designed to serve as a medium of conversation between therapist and child and to help the therapist assess the child's mood, attitude, temperament, and even perceptive and cognitive abilities. Depending on his or her age, the child may merely look at the pictures or may study actively the relative costs of items he or she has long desired. How children envision the way they would obtain the sought-after prize, and what they think it would do for them, are significant questions. In a way, the mail order catalog makes concrete the classical question, "Supposing you had five wishes, what would you wish?" In fact, Sears, Roebuck Co. calls their Christmas catalog "The Wish Book."

With more retiring children, the therapist may have to be quite active and thumb through the pages of a catalog commenting about the contents from his own point of view. This requires divulging information about oneself, and therefore is not in accord with customary psychoanalytic approaches. However, many children respond when their attention is focused on a relatively neutral book or magazine. The therapist, armed with the knowledge of the child's presenting problem, can skillfully select particular catalogs or certain sections about which he might comment in order to trigger some response from the child. A few case vignettes will help illustrate this approach.

Case Vignettes

Vignette 1. Ian, a 7-year-old boy, had become extremely unmanageable in class; he was hostile, uncooperative, inattentive, and negativistic and was being considered for expulsion. An obvious precipitant about which he refused to speak was the birth of a brother. In the opening session of therapy I allowed him to rummage in the catalog box. He selected a Montgomery Ward catalog and leafed through it carelessly. Upon reaching the section dealing with cribs and baby supplies, he became irritable and tore the page, although he had previously commented that there was nothing troubling him and he was pleased that he had a new baby brother. This behavior gave us an opportunity to discuss the situation in further detail, which led to a reduction in his tension and anger toward the new rival in the family.

Vignette 2. Lynn, an 11-year-old girl, after initially expressing disdain for psychiatry, her parents, and people in general, seemed pleased to examine a catalog from I. Magnin. The luxurious clothes were something she admired but had been told she could never hope to obtain. Her mother called her ugly, fat, and stupid. In talking about the color, styles, and the kinds of people who would purchase such expensive garments, Lynn displayed a remarkable sophistication and sensitivity in the areas of self-image, sexuality, economics, and oedipal competition. She spoke of her mother's fear of Lynn's growing sexuality and noted that her father seemed to like her better than her mother in many ways.

Vignette 3. Sharon had been in a class for the educationally handicapped. At age 9 she was 2 years below grade level and had almost no reading skills. Her referral for psychiatric evaluation was prompted by an episode of petty theft. She came from an affluent middle-class family. The catalog experience revealed that although she was dyslexic, she could readily identify graphic symbols and understand their function. Her sense of deprivation seemed rooted in an emotional distance from her parents, who considered her retarded. Her mother had a degree in geology but rarely shared this interest with her daughter. Sharon selected a rock hunter's catalog. She spoke about the methods of distinguishing rocks by

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hardness, color, and other physical properties. This opened a level of communication between mother and daughter that was immensely therapeutic.

Vignette 4. Jerry, a precocious 6½-year-old boy, was described as hyperkinetic by school authorities. He made a whirlwind entry into my office. He was extremely distractible and often apologized for his clumsiness. To the surprise of his mother, who accompanied Jerry to the office, his restlessness diminished abruptly when he focused his attention on a catalog of scientific instruments. He told me of his interest in science fiction and building things. He then turned to a real estate catalog and shared his ambition of becoming an architect. A seldom perused catalog of orthopedic appliances seemed to be of particular interest to Jerry. He explained that another little boy he knew had been hit by a car and was wearing a brace. He had stopped playing with the boy because he was afraid to look at the brace. He was able to use the orthopedic catalog to desensitize himself to such threatening objects and to explore his feeling about injury and loss of bodily function. He and his parents then were able to cooperate with a trial of methylphenidate, which was successful.

Discussion

Like my experience with using magic (2) in the psychotherapy of children, the mail order catalog provides an interesting alternative to conventional therapeutic devices in child psychotherapy. In addition to being a vehicle for communication, it is a potential source of projective and cognitive assessment.

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Decreased Serum Prolactin in Opiate Withdrawal and Dopaminergic Hyperactivity

BY MARK S. GOLD, M.D., DONALD R. SWEENEY, M.D., PH.D., A.L.C. POTTASH, M.D., AND HERBERT D. KLEBER, M.D.

We have recently proposed a noradrenergic hyperactivity hypothesis to explain the signs and symptoms of opiate withdrawal (1). However, a dopaminergic hyperactivity hypothesis has also been proposed; it is supported by a large number of animal studies (2, 3). Serum prolactin is predominantly controlled in vivo and in vitro by an inhibitory dopaminergic mechanism (4). Consistent with this control mechanism, augmentation of dopaminergic function results in decreases in serum prolactin, and inhibition of dopamine function results in increases (4, 5).

To begin evaluating these hypotheses we measured serum prolactin in five opiate addicts during significant opiate withdrawal and after suppression of symptoms by clonidine. In addition, to provide a drug-free baseline we measured serum prolactin in these addicts 2 weeks after they had been discharged from the hospital and were free of clonidine and opiate.

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Method

The subjects were five opiate addicts who had been maintained on 15–60 mg of methadone for 2–10 years. All gave informed consent to participate in a study that involved abrupt discontinuation of methadone and administration of 5 μg/kg of clonidine after at least 36 hours with no opiate administration. A research nurse clinician observed the presence or absence of 21 withdrawal signs and symptoms every 30 minutes, as described previously (1), with the subjects at bed rest after an indwelling venous catheter was in place. Ratings were done simultaneously with blood samples taken 60 minutes before clonidine administration, at the time of administration, and 60 and 120 minutes after clonidine administration. While the subjects were taking methadone, prolactin levels were not measured due to the stimulatory effects of methadone on serum prolactin. Opiate-free samples were taken by venipuncture 2 weeks after hospital discharge. All samples were placed on ice and centrifuged within 1 hour. Serum was frozen at −20°C until radioimmunoassays were performed in duplicate (6).

Results

As reported previously (1), clonidine caused a rapid and significant decrease in the signs and symptoms of opiate withdrawal. Opiate withdrawal symptoms decreased from a mean (±SE) just before clonidine ad-

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ministration of 15.2±0.7 to 3.6±0.8 at 60 minutes after clonidine administration (paired t=10.3) and to 0.6±0.4 120 minutes after clonidine administration (paired t=16.7, p<.001). Serum prolactin was significantly decreased from a mean (±SE) just before clonidine administration of 10.8 ng/ml±1.0 to 8.2±0.8 at 60 minutes and to 7.0±0.8 at 120 minutes after clonidine administration (t=3.92, p<.02). Serum prolactin was significantly reduced during the peak of opiate withdrawal to a mean of 10.2±0.7 ng/ml compared to a mean of 13.6±0.9 ng/ml for the assay done when patients had been free of opiate and clonidine for 14 days (t=3.50, p<.02).

Discussion

Significant decreases in serum prolactin are normally attributable to the administration of medications that stimulate dopamine receptors or augment dopamine release or neurotransmission (4, 5). The decreased serum prolactin reported here for patients in opiate withdrawal supports a hypothesis of dopamine hyperactivity in opiate withdrawal (2, 3). However, as the symptoms of opiate withdrawal were reversed and virtually eliminated by clonidine, the serum prolactin did not return to preclonidine or postaddiction levels. This suggests that dopaminergic hyperactivity accompanies opiate withdrawal but is not directly related to the symptoms or signs of withdrawal. Clonidine administration has not been reported to produce any changes in serum prolactin in humans (7). Although clonidine neither augments dopamine neurotransmission nor stimulates dopamine receptors, it is possible that the further decreases in serum prolactin represent continued dopaminergic hyperactivity, which is present in patients with symptomatic or asymptomatic opiate withdrawal. Clonidine may produce the additional decrease in serum prolaction through nondopaminergic mechanisms (6).

We have suggested that endogenous and exogenous opiates increase serum prolactin by inhibition of dopamine neurotransmission (8). Withdrawal from chronic opiate administration might be expected to release dopamine neurons from inhibition and reduce serum prolactin. Animal studies (2, 3) have found dopaminergic hyperactivity in opiate withdrawal and there has been speculation on the relationship of this hyperactivity to the pathophysiology of withdrawal. However, the failure of clonidine to reverse both the opiate withdrawal symptoms and the decreased serum prolactin suggests the postulated dopamine hyperactivity is present but not intimately related to generation or relief of symptoms.

Clonidine and the opiates reverse the behavioral effects of electrical or chemical activation of noradrenergic nuclei in primates (9, 10). On the basis of these and other studies we have suggested that opiate withdrawal results from increased neuronal activity in noradrenergic brain areas, which are regulated by both α-2-adrenergic and opiate receptors (1). Additional studies that concurrently measure withdrawal symptoms and the major brain metabolite of norepinephrine, 3-methoxy-4-hydroxyphenylglycol (MHPG), during spontaneous opiate withdrawal and after clonidine administration are necessary to determine whether the postulated noradrenergic hyperactivity (1) for opiate withdrawal is present (i.e., increased MHPG) and related to symptom relief (i.e., return to nonwithdrawal baseline levels after clonidine administration).

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Saliva Lithium and Lithium Therapy

BY ROY J. MATHEW, M.D., JAMES L. CLAGHORN, M.D., DAVID FENIMORE, PH.D., CHESTER DAVIS, PH.D., AND MAXINE WEINMAN, PH.D.

The substitution of saliva lithium measurements for the accepted serum assays in the monitoring of lithium therapy has been a topic of recent interest (1–3). Despite the consistently high correlation between saliva and plasma lithium levels, it has been indicated that individual variability is too high to permit the clinical use of saliva assessments as a valid measure of lithium levels (2). In addition, the reliability of plasma lithium values as an index of lithium activity has also been questioned, and it has been suggested that red blood cell (RBC) lithium levels may be a better index of lithium activity (4–7).

This study evaluated the relationships among plasma, RBC, and saliva lithium levels using atomic absorption spectrophotometry.

Method

Thirty synchronous samples of blood and saliva were taken from nine subjects at varying stages of lithium therapy after the procedure was explained and their informed consent obtained. All samples were collected in the morning before the initial daily dose of lithium was taken. The patients rinsed their mouths four times with tap water and chewed a piece of paraffin wax for 3–4 minutes; they were instructed to swallow the saliva produced during this period. They were then instructed to spit into a container several times until 2–3 ml of froth-free saliva was collected. If the sample was not clear, colorless, and froth-free, the procedure was repeated until an acceptable sample was obtained.

Assay procedures. Lithium levels were determined in saliva, plasma, and erythrocytes on an atomic absorption spectrophotometer. The intracellular level of lithium was determined by the method of Hisayasu and associates (8).

The lithium concentration within the erythrocyte was calculated from values in whole blood, plasma, and the hematocrit. In order to verify results, all samples were assayed in duplicate alongside commercial control samples.

For the lithium measurements, plasma, whole blood, saliva, and standard were diluted 10-fold with deionized water; this mixture was aspirated directly into the flame of the spectrophotometer.

Results

Pearson product-moment correlations were calculated between saliva lithium and plasma lithium levels ($r=.569$, $p<.001$), saliva lithium and RBC lithium levels ($r=.501$, $p<.01$), and RBC lithium and plasma lithium levels ($r=.597$, $p<.001$). A high degree of individual variability was noticed when saliva lithium levels were plotted against plasma lithium and RBC lithium. Intraindividual variations in the ratios of saliva and RBC, saliva and plasma, and plasma and RBC lithium levels were also found to be high. No significant correlations were found among these three ratios.

Discussion

Our finding confirms that of Sims and associates (2), i.e., saliva lithium levels have limited usefulness in monitoring lithium therapy. We found a high correlation between the plasma and saliva lithium values, but there was an unacceptably high interindividual and intraindividual variation. Saliva lithium showed a stronger correlation with plasma lithium than RBC lithium; a high degree of scatter was seen when RBC lithium was plotted against salivary lithium. Even if one accepted the viewpoint that RBC lithium levels are a better index than plasma levels for the monitoring of lithium therapy, saliva lithium assessments would still be of limited use.

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IN MEMORIAM

H. Houston Merritt
1902–1979

H. HOUSTON MERRITT, M.D., Dean Emeritus and Vice President Emeritus in charge of Medical Affairs and Emeritus Professor of Neurology, College of Physicians and Surgeons, Columbia University, died on January 9, 1979, three days before his 77th birthday. Despite an incapacitating gait disorder, he was active to the end, having just completed the sixth edition of his famous *Textbook of Neurology*, which is regarded as a standard text throughout the neurologic world.

Dr. Merritt graduated from Johns Hopkins Medical School in 1926. After training in medicine at New Haven Hospital, he went to Boston City Hospital in 1928 for training in neurology. There he joined a brilliant group of clinical researchers and by 1938 his name had appeared on 55 papers, some in collaboration with men of the caliber of Solomon, Frank Fremont-Smith, Moore, and Lenox. In 1939 he collaborated with Tracy Putnam in the discovery of phenytoin (Dilantin) as an anticonvulsant—a godsend to sufferers of convulsive attacks. At the same time Dr. Merritt became known as an authority on cerebrovascular diseases and on cerebrospinal fluid. Ironically, his final illness included disorders in both of these areas.

Meanwhile, Dr. Merritt had risen to the rank of Associate Professor in Neurology at Harvard Medical School. In 1944 he was called to New York. First he was Clinical Professor and Chief of the Montefiore Service, Columbia University, and then in 1948 became Professor and Chairman of the Department of Neurology at Columbia and head of the New York Neurological Institute. Ten years later he was elected Dean and Vice President of Medical Affairs at Columbia while he still retained his position as Director of the Neurological Institute.

Under his direction, the Neurological Institute became a major training center for clinical neurology and neurologic research. The William Black Research Building was erected in 1960 and a short time later the medical school affiliated with the Harlem Medical Center—both events under Merritt's aegis—and thus his university demonstrated its commitment to research and to the care of the not-so-fortunate.

In recognition of his many achievements the Merritt Chair of Neurology was dedicated in his honor, as was the Merritt Clinical Research Center for Muscular Dystrophy and Related Diseases. Honors too numerous to mention accrued to him through the years. When he entered neurology it was languishing as a discipline and there was danger that it would be absorbed by medicine and neurosurgery. But, fortunately, he lived to see it attain its present major status—indeed, he is regarded by many as the principal architect in the rise of modern neurology. Always well disposed toward psychiatry and helpful to its cause, Dr. Merritt was a Life Member of the American Psychiatric Association.

Dr. Merritt was a modest man who wore his honors lightly. Many of the honors he received were associated with great responsibilities, yet he carried them out quietly, skillfully, and with éclat. He was called into consultation regarding a President of the United States, a famous Russian official, and the dictator of Portugal. Portugal conferred upon him its highest honor, the title of Grand Officer of the Order of Santiago. Withal he treated the high and the lowly with equal skill and devotion.

An astute and popular clinician and teacher, Dr. Merritt had an intelligence and a remarkable memory that were often commented upon, as was his fabled reputation as a master diagnostician. Indeed he was a bit of a genius, and fortunately his lamp of genius remained lit until near the end of his life. He was a warm, sensitive man—soft-spoken and sparing of speech to the point of being laconic—and people from all walks of life were attracted to him. He cared for people and people in turn cared for him. He could relieve tight situations with a witty remark or an appropriate comment taken from his fund of humorous stories.

He received a number of distinguished service awards from colleges and professional societies. On the occasion of the golden anniversary of the American Neurologic Association (which he helped to bring to full flower) the president of that association, while presenting the award, unwittingly furnished an epitaph for him. He said, "For H. Houston Merritt, Neurologist to the World." This designation is indeed a fitting epitaph for a great man.

F.J.B.

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LETTERS TO THE EDITOR

This section contains reactions to Journal *articles, statements of opinion, comments on Association activities, requests for information, etc. Letters should be submitted in duplicate to the Editor, who makes all decisions regarding publication. Letters must be typed double spaced throughout and should not contain more than 500 words and 5 pertinent references. Criticisms of published articles will automatically be sent to the author(s) for response. Letters will be edited for clarity and conformance with* Journal *style. We regret that we cannot inform writers of the disposition of letters to return those which are not printed.*

Further Perspectives on Primary Prevention

SIR: The article "Primary Prevention in Perspective" (January 1979 issue) by Richard Lamb, M.D., and Jack Zusman, M.D., takes the seemingly paradoxical position which advocates treatment of mental disorders as a first priority and prevention as a separate and secondary activity. The authors argue not that primary prevention is less important than treatment, but rather that it is not scientifically based and virtually impossible to carry out. Although I recognize that there are difficult methodologic problems in evaluating prevention activities, I strongly disagree with the authors' implicit antiresearch position. Although this is by no means a new debate, as one who sees greater possibilities—both theoretically and clinically—for individual and population-based strategies of prevention, I wish to address a few observations to the authors.

First, the studies they cited on the relationship of social factors to mental disorders are indeed largely based on statistical associations, and each has its methodologic limitations. Like all such studies, including research demonstrating relationships between cigarette smoking and lung disease, these studies alone do not prove causation, nor could they be expected to do so. On the other hand, the evidence for the so-called "proven" forms of prevention described in the article, which I would not dispute, is also largely based on statistical observations (e.g., "seldom seen today," "lowered incidence"). Despite complex methodologic differences among studies, the saw cuts both ways. The list of proven preventions can be made much longer than that offered by the authors, as a review of any volume of a good public health journal will reveal. The potential impact of these prevention programs in terms of numbers of persons affected can be very large indeed.

Second, the authors seem to imply that their scientific imagination does not encompass future developments in basic clinical research. Are they certain that the next 10, 20, or 50 years does not hold the promise of effective medical or biological prophylaxis of some psychiatric disorders?

Finally, while I would not disagree with the need to devote a preponderance of resources to the treatment of persons with severe and chronic psychiatric disorders, it does seem unscientific to argue against research on prevention or evaluation of treatment. Research is usually expensive, but the potential payoff is more proven preventions. In a field where treatment produces few "cures," prevention is a valuable source of hope, not a "dangerously foolish gamble."

ROBERT A. DORWART, M.D.
Cambridge, Mass.

SIR: Drs. Lamb and Zusman have correctly identified many of the conceptual and practical problems anent prevention. Prevention programs tend to be popular, cosmetic, and inconsequential (1). In one community mental health program, I observed that growth groups with adequate but unactualized clients receive top priority for a therapist's time, while more intransigent cases are placed on a waiting list or referred elsewhere.

Of course, this is not news. Therapists' proclivities for the YAVIS (young, attractive, verbal, intelligent, and successful) were identified 15 years ago by Schofield (2).

The full irony of the analysis presented by Drs. Lamb and Zusman is that many of their arguments against prevention can be applied equally well to psychotherapy. London (3) quoted the classic definition: "Psychotherapy is an ill defined technique applied to an unknown population with indeterminate results. For this technique, long and arduous training is required." It has held up well.

In psychotherapy, as well as in prevention, there are many theories with little validation. There have been few unequivocal demonstrations of the efficacy of psychotherapy. Perhaps most ironic of all is the authors' insistence that in allocating funds to prevention "we need to distinguish carefully among those programs which are proved, those which are not yet proved but seem well on the way to attain a status, and those which are at best experimental." It seems to me that there are remarkably few mental health programs that meet even the minimal standards of experimental design in the sense of collecting evaluative data and using them for something more than a head-count to justify continued funding.

The authors performed a valuable service by documenting areas where prevention efforts have worked. Those excited by "doing prevention" often overlook these areas and sometimes consider them irrelevant. I think that the dramatic reduction in drug poisonings in children following legislation mandating "child-proof" packaging serves as a good example of prevention, as does the reduction of barbiturate-related suicides that was seen after prescription practices were tightened (4).

I would encourage would-be mental health "preventors" to examine these cases of successful prevention more closely. In fact, I would contend that the most effective and far-reaching prevention will come from increased attention to the role of policy and legislation.

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MARK WORDEN, M.D.
Roseburg, Ore.

SIR: I want to comment on Drs. Lamb and Zusman's paper because what is valid and important in it may be obscured by the surprising number of assertions that are simply incorrect or controversial editorializing. I have long admired the work of these two scientist-practitioners and am saddened to see them associated with a point of view that is so parochial and unconstructively negative.

The authors' position is that "there is little evidence that primary prevention has been effective," that "research. . . in prevention is sorely needed but should be funded separately and with descretion," and that "scarce mental health funds should not be diverted from direct treatment for this purpose" (p. 12).

The authors are correct that the need for mental health services now far outstrips the availability of mental health professionals and will probably continue to do so for the foreseeable future. They are correct that the renewed hopes for effective primary preventions have come about, in part, from the increasing realization of this melancholy mismatch between the supply and demand for mental health services. However, I have never heard or read a proposal by any responsible person reflecting "an enthusiastic acceptance of prevention as an alternative to direct treatment" (p. 12). Such an assertion on the authors' part is frank hyperbole and a bit irresponsible. I can assure Drs. Lamb and Zusman that the Task Panel on Prevention of the President's Commission on Mental Health never entertained such a notion.

The authors focus on psychosocial aspects of prevention. They urge the reader to be cautious because the assumption behind much of contemporary thinking about primary prevention—that "difficult life circumstances lead to mental illness," or that "later psychopathology is to be understood in terms of earlier life experiences"—is as yet unproved. It is not clear how the authors have chosen to use the phrase "mental illness" or what they would accept as proof, but I would point out that most of the mental health clinical enterprise rests on that same assumption.

The authors incorrectly stated that "without knowledge of cause, primary prevention programs can only be shots in the dark" (p. 13). The great triumphs of prevention in the field of public health have occurred in the absence of knowledge of cause. Most successful primary prevention programs have been implemented on the basis of suspected associations, not known causes. If Drs. Lamb and Zusman want to argue that suspecting associations between events is the same as knowing cause, then we are in the same position in the field of emotional disorders that we were in with regard to the infectious and nutritional disorders of the past century. We have enough empirically derived associations to undertake a whole series of modest, well-designed experimental intervention programs.

"Recent research has increasingly suggested the operation of genetic and biochemical factors in the production of mental illness, and these factors are not targets of typical primary prevention programs" (p 13). The authors are uninformed in this case. Interest in learning more about genetic and biochemical factors pertinent to primary prevention of emotional disorders is intense and central in the thinking of virtually all persons professionally concerned with primary prevention.

"Recent research . . . indicates that major mental illness is probably in large part genetically determined and is probably therefore not preventable" (p. 13). This assertion would make a geneticist cringe. Huntington's chorea, Tay-Sach's disease, and phenylketonuria are all genetically determined and preventable.

Drs. Lamb and Zusman urge that "prevention's appeal must be resisted by mental health professionals lest it become a glamorous rationalization for avoiding treatment of difficult, mentally ill persons" (p. 16). I share their concern for the chronic difficult patient but, regrettably, mental health professionals have not had to wait for the glamor of prevention to avoid treating such patients. Our professions have a long history of spending most of our clinical hours with the most mildly disturbed individuals.

The recommendations of the President's Commission on Mental Health on the subject of prevention are, in my judgment, prudent, appropriate, and timely. These recommendations—for the establishment of a Center for Prevention within the National Institute of Mental Health, for special programs directed toward children at risk, and for the development of services and programs oriented around times of significant life stress—deserve the active support of all mental health professionals and professions. To insist on waiting until all direct treatment needs are paid for before resources are devoted to prevention efforts is to doom our professions to unending continuation of the hopeless spiral we are in now. Of course, some money may have to be diverted from direct treatment in order to undertake a significant effort in extending our knowledge in the field of primary prevention. In terms of the total cost of mental illness to our society, that resource diversion is not only trivial but, more importantly, represents one of the few hopes we have for the ultimate control of one of our major sources of human suffering.

BERNARD L. BLOOM
Boulder, Colo.

SIR: Drs. Lamb and Zusman should be applauded for their article on primary prevention, which should have been subtitled, "The King Is Naked." They expose the fact that in our current state of the art, no one can deliver on the promise of prevention of psychiatric illness.

In the political marketplace, hollow promises often prevail over hard, and unpleasant, facts. The mental health field is riddled with politically favored programs that pursue laudable goals with unproven and often patently ineffectual means. We need more articles debunking popular myths. Efforts will be better spent on serious research that will someday provide answers than on present-day equivalents of snake oil. The article by Drs. Lamb and Zusman is a step in the right direction.

JOSEPH MORE, M.D.
Wilson, N.C.

Drs. Lamb and Zusman Reply

SIR: The purpose of our article "Primary Prevention in Perspective" was to direct attention to the fuzziness of many

of the concepts and definitions involved in primary prevention and to focus on some of the complex issues that are often glossed over in looking for the elusive "ounce of prevention."

Dr. Dorwart has misperceived our position on prevention as "antiresearch." No one feels more strongly than we about the need for research in this area. We would only reiterate that we think this research should be funded separately, with a full understanding of the issues, that it consist of carefully controlled studies, and that it not be at the expense of reducing direct services. Further, we feel that wide-scale "prevention" programs should not be set up until there is good research evidence that they are effective.

Unlike Dr. Bloom, we are well aware of responsible persons who reflect "an enthusiastic acceptance of prevention as an alternative to direct treatment." This was especially true in the '60s and early '70s, when it was often implied (and sometimes promised) that the techniques of prevention would result in a significant reduction of mental illness and would eventually drastically reduce the need for conventional treatment because there would be so few patients (1, 2). More recently, advocates of prevention have been just as enthusiastic but more careful in their wording.

We hope that Dr. Bloom is not correct when he states that the entire mental health clinical enterprise rests on the assumptions that difficult life circumstances and social conditions lead to mental illness (when we use that term we refer to diagnosable mental illness) or that later psychopathology, including the psychoses, is to be understood in terms of earlier life experiences. The convincing evidence of the past decade that there are genetic factors in the etiology of schizophrenia, the primary affective disorders, alcoholism, and antisocial personality should cause us to consider modifying some of these assumptions and take these genetic factors into full consideration (3–5).

We stand by our statement, "Without knowledge of cause, primary prevention programs can only be shots in the dark." Of course, even shots in the dark will occasionally hit the mark, and there are notable instances of this in the history of public health. But with all the pressing demands for scarce mental health funds and the tremendous complexities and intangibles in the area of mental health, how many millions of dollars can we afford for such long shots?

We are pleased to hear from Dr. Bloom that "interest in learning more about genetic and biochemical factors pertinent to primary prevention of emotional disorders is intense and central in the thinking of virtually all persons professionally concerned with primary prevention." We hope this attitude becomes more prevalent, for to date such persons are in the minority of those advocates of prevention who make their views known publicly. For example, the lack of discussion of such factors was notable in the report of the Task Panel on Prevention of the President's Commission on Mental Health, in which Dr. Bloom was involved. Further, genetic counseling that has the potential for preventing mental illness seems to be only of peripheral interest to most of those involved in prevention efforts.

We want to thank Dr. Worden for pointing out the need for meaningful evaluation of all mental health services and Dr. More for his encouragement of our efforts.

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H. RICHARD LAMB, M.D.
JACK ZUSMAN, M.D.
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Defending the Military Family

SIR: "The Military Family Syndrome" (September 1978 issue) by Don M. LaGrone, M.D., which painted an unfavorable picture of the military family, was based on a number of false and/or misleading analyses of statistics. Since the damage done by this article has been magnified considerably by its quotation in *Time* magazine (Dec. 11, 1978) and in *Air Force Times* (Dec. 25, 1978), I feel it incumbent on me and the Air Force to correct Dr. LaGrone's analysis.

Dr. LaGrone stated (p. 1043) that "the incidence of child abuse is five times higher than the national rate." In fact, the USAF *Medical Service Digest* article he used as a source said, "Some studies indicate that our mortality rate could be as much as five times the national average figures." This comment was based on a study done before the Air Force started collecting comprehensive data on child abuse; since the implementation of the Air Force Child Advocacy Program in 1975, the actual figures collected indicate that the overall incidence rate of child abuse in the Air Force is about the same as in the nation at large, and the mortality rate from child abuse is lower in the Air Force. In 1976 the incidence of child abuse/neglect cases among Air Force families was reported to be 392 per million, with a mortality rate of 1.8%. In 1977 the number of cases dropped to 336 per million, but with a mortality rate of 2.5%. In 1974 among U.S. civilian families the incidence of child abuse/neglect cases was reported as 380 per million, with a mortality rate of 5%.

Dr. LaGrone said that "the incidence of alcoholism is high" (p. 1043). Contradicting this assertion, a 1978 Rand Corporation study (available in draft) states that "alcohol-related behavior in the Air Force is similar to that among civilians."

Dr. LaGrone miscalculated and misinterpreted the figures on psychiatric treatment of children of military members (p. 1040). If we accept Dr. LaGrone's premise that there was an entirely new group of 3,200 families on base each of the 2 years, then the parallel figures for children would be 14,800 in 2 years, not 12,800. Of the 14,800 children, 5.35% (N=792) were seen for evaluation. Since 6% of these (N=48) had no psychiatric diagnosis, the number potentially needing help was 744, or about 5% of those on base. If we accept Dr. LaGrone's estimate that "10% to 12% of the general population of children in the United States have emotional problems and need treatment," it appears that the military children have much less need—5% versus 10%-12%. Finally, the catchment population for the military clinic that Dr. LaGrone used as a basis for his statistics was understated since the hospital and its clinic drew from a regional population.

This would further lower the incidence of mental illness in military children.

Contrary to Dr. LaGrone's statement that "when confronted with such information (alcohol and child abuse incidence) the military becomes defensive and moves slowly, if at all" (p. 1043), the Air Force is among the leaders in establishing human service programs to help individuals and families deal with alcohol and child abuse problems. The first Air Force alcohol treatment center was established in 1966 at Wright-Patterson Air Force Base, Ohio. The Air Force Child Advocacy Program was launched in April 1975 and has been highly effective.

GARTH B. DETTINGER, MAJ. GEN., USAF, MC
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Dr. LaGrone Replies

SIR: Gen. Dettinger's letter is difficult to address specifically, since he entirely missed the point of my paper. He chose to attack two side issues I raised in my closing comments and on that basis claimed my conclusions were erroneous. In my opinion, he wrote his letter for a political purpose rather than in the spirit of scientific debate.

I am struck by the drastic drop in the mortality rate of military child abuse he reports since 1975. I cannot help but be skeptical and invite Gen. Dettinger to quote the source of his statistics. A recent study summarized by the *Houston Post* (January 22, 1979, p.9a) stated that the incidence of military child abuse is two to three times higher than the civilian rate. A study mentioned in that review compared the incidence of child abuse at Fort Carson, Colo., with the local civilian population and found it twice as high. The Rand Corporation study quote Gen. Dettinger used says nothing about the significance of drug and alcohol abuse in the military. The "behavior" probably is the same. While in the military I attended a lecture given by an Air Force social actions instructor who said that 80% of all students and new recruits were abusing drugs.

Regarding the clinical statistics in the paper, there was a mistake in the number given for children living on the base. It should have been 6400 instead of 7400. I based this on the personnel office estimates of 2 children per family. I believe the rest of the numbers given in the paper are correct. I think the fact that we evaluated almost 12% of the children over 2 years of age is notable, especially in view of the resistance in officers' families to mental health involvement. The fact that our base was regional has no bearing on our study since, almost without exception, the families resided on the base.

The purpose of my paper was twofold: to explain the higher incidence of behavior disorders in children evaluated in our clinic as opposed to civilian clinics and to help the nonmilitary therapist understand the dynamics of the military family and the military. To effect positive change, the therapist must also consider the identified patient as part of a very complex "system."

Two other studies, those of Bloom (1) and White (2), were similar to mine. White, in fact, reported that 59% of his diagnoses were behavior disorders and attributed the cause to similar dynamics.

In my response to Dr. Jon Shaw's letter (3), I explained that I first tried to publish my paper while in the military. It was rejected in "official channels" eight times. Finally I was told that it would not be cleared because "it would hurt the recruiting program." How valid is research derived from a system that represses "unofficial" points of view? When considering the dynamics of such a system, I would suggest that Gen. Dettinger's use of the word "damage" in reference to the impact made by clinical research could be viewed as pathognomonic.

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Reserpine, Serotonin, and Schizophrenia

SIR: The improvement of some chronic schizophrenic patients with the addition of reserpine to their neuroleptic regimen as reported in "Addition of Reserpine to Antipsychotic Medications in Refractory Chronic Schizophrenic Outpatients" by Norman M. Bacher, M.D., and Harvey A. Lewis, M.D., (April 1978 issue) is a clinical finding that we have observed repeatedly over the past two years.

Our clinical (uncontrolled) trials with reserpine produced a tangible and sometimes marked improvement in certain chronic schizophrenic patients who for years had not responded fully to various neuroleptics of the dopamine (DA) receptor-blocking type. We also observed that chronic schizophrenic patients with prominent psychomotor agitation, auditory hallucinations, and paranoid schizophrenia (chronic paranoid schizophrenia or schizo-affective schizophrenia, excited type) tended to respond better to reserpine than chronic schizophrenic patients with negative symptoms such as withdrawal-retardation, flat affect, and anhedonia.

Because reserpine depletes the stores of serotonin as well as dopamine, norepinephrine, and other catecholamines, the authors raised the question of whether there is an increased activity of serotonin in chronic schizophrenia. The question has of course been raised repeatedly in the literature of the past two decades, and researchers still do not agree (1) on whether serotonin activity is increased or decreased in schizophrenia; there is evidence for both possibilities.

One of our severely psychotic patients who achieved his best remission in over 12 years with the addition of reserpine (10 mg/day) to his neuroleptic (chlorpromazine, 1600 mg/day) had previously become worse with the addition of the serotonin precursor 5-hydroxytryptophan (5-HTP) (up to 2700 mg/day with peripheral decarboxylase inhibition) (2). He had also shown no improvement with the addition of the dopamine synthesis inhibitor α-methyl-*p*-tyrosine (AMPT) (up to 3000 mg/day) (3). His response profile is consistent with an increase in serotoninergic activity in schizophrenia. The patient also had a low platelet MAO activity, which is compatible with an increase in serotonin (4). Platelet MAO activity has been reported to be lower in chronic paranoid schizophrenics with auditory hallucinations (the type of schizophrenia we noted to be reserpine-responsive) than in other chronic schizophrenic patients (5).

It is possible that a certain subtype of chronic schizophre-

nia characterized by increased psychomotor agitation, paranoid delusions, and prominent auditory hallucinations, with partial or no response to dopamine-receptor blocking neuroleptics, and showing clinical improvement with the addition of reserpine, could have increased CNS serotoninergic activity with or without increased dopaminergic activity. Such a subtype would 1) have low platelet MAO activity, 2) worsen with serotonin agonists, and 3) (theoretically) benefit from a trial of serotonin antagonists like para-chlorphenylalanine (PCPA). An opposite schizophrenic subtype could exist, characterized by negative symptoms like autism, flat affect, and minimal agitation or auditory hallucinations. Patients in this group could have low CNS serotoninergic activity (with or without dopaminergic hyperactivity), high platelet MAO activity, partial or no response to dopamine receptor blocking neuroleptics, worsening with reserpine or PCPA, and improvement with serotonin precursors such as 5-HTP (2).

Such hypothetical subtypes of chronic "neuroleptic-resistant" schizophrenia would not only explain the response to reserpine but reconcile the confusing literature on serotonin and schizophrenia.

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Decision-Making Approaches in Medicine

SIR: We were disappointed in the uncritical approach to decision tables taken by Bruce Roberts, M.D., in "A Look at Psychiatric Decision Making" (November 1978 issue). Dr. Roberts correctly noted that decision tables have existed for a long time and that a great deal is known about their properties (1). We believe that these properties make decision tables unsuitable as a means of representing algorithms for all but a small group of medical decisions. There are three major sources of difficulty:

1. There is neither an optimum nor a systematic way to order decisions (e.g., in figure 2, p. 1386, of the article one might argue that ruling out organicity should take precedence over determining the presence of a normal grief reaction, since neither precludes the other), and the order selected has a significant impact on the amount of effort required to use decision tables (1). Depending on the sequencing of items, there can be an order of magnitude variation in the number of questions that a user must answer to reach a decision. A constructor of decision tables is often faced with the unacceptable alternatives of building tables that are either medically optimum but contain too many items or are mathematically optimum but medically less sensible.

2. There are some simple decision rules that are easy to state but difficult to express in a decision table. An example is the rule used in the current draft of *DSM-III* to conclude whether a patient is suffering from a depressive episode. The rule requires that, among other things, 4 out of 8 symptoms be present. In a decision tree there is no way to directly express this "4 out of 8" consideration before taking an action.

3. There is no meaningful way to handle uncertain or imprecise information. In Dr. Roberts' example of the ancient Persian poem (p. 1384–1385), what is one to do when one is uncertain whether one knows? If one cannot give a yes or no answer after exhausting all sources of information in a clinical situation, what is the procedure to take when one reaches a point that allows only a yes or no answer?

For these reasons, we feel that, even as pedagogic devices, decision tables are unattractive. There are other approaches, such as production rules (2) or decision sets (3), that have all of the power of decision tables and fewer drawbacks.

We share Dr. Roberts' opinion that formalizing medical decision making is an important task. It is also a complex and difficult task. Why attempt it with inadequate tools?

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Dr. Roberts Replies

SIR: Drs. Brooks and Heiser state, "There is neither an optimum nor a systematic way to order decisions. . . ." I disagree. There are times when one decision has a higher priority than another decision. Whether a depressed patient is an immediate danger to self or others should be assessed before a decision is made to use antidepressant medication. There are times in the decision-making process when there is no optimum next decision. This uncertainty can be incorporated into a systematic approach. An example of this is whether to first investigate for organic causes a depression with vegetative symptoms or to first treat this depression with an antidepressant. There are no consistent data on the percentage of positive findings of organic illness by age as a cause of depression with vegetative symptoms. In light of this uncertainty, many approaches are acceptable. One could empirically treat the depression with antidepressants and then look for organic causes if the antidepressant does not totally improve the symptoms. Another approach would be to look first for organic causes of the depression. If no organic causes are found or treating the organic causes only partially improves the symptoms, then antidepressants can be tried. Multiple combinations of looking for organic causes and treating with antidepressants can be tried. This flexibili-

ty can be incorporated into a systematic approach and is being done by Weed (1) and Schultz in the Problem-Oriented Medical Information System (PROMIS) Laboratory.[1] The PROMIS system allows for multiple choices on one display. In the above example, the computerized problem-oriented record would allow the user the freedom to investigate one or more organic causes and/or empirically treat the patient with antidepressants. There are many times in psychiatry, even if all the knowledge is available on a given subject, when there is no clear direction to take. A decision must be made in the face of uncertainty. This uncertainty can and has been incorporated in the computerized problem-oriented record.

Drs. Brooks and Heiser state that "in a decision tree there is no way to directly express this '4 out of 8' consideration before taking an action." In my original manuscript there was a decision requiring "5 out of 8" to be present to help decide if medication was indicated, and I have sent Drs. Brooks and Heiser a copy of the manuscript. There is no difficulty incorporating this type of decision in a decision table. There is also no difficulty providing a "not sure" option along with the yes or no option.

I think it is crucial to understand clearly what decisions are being made and how we are making them. I think Drs. Heiser and Brooks share my enthusiasm for objectifying the decision-making process. The underlying question is how this work will be computerized and how it will assist psychiatrists. My bias is that the goal should be to develop a system to organize information and provide a logical structure for making clinical decisions—not to replace the psychiatrist's ability to think. This system would relieve the psychiatrist from memory-oriented tasks and allow him or her to focus on the more difficult tasks of incorporating empathy and understanding into the decision-making process.

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BRUCE ROBERTS, M.D.
Chicago, Ill.

[1]Schultz JR: PROMIS: Problem-Oriented Medical Information System: a medical and technical solution to four problems in medicine (unpublished manuscript). Copies are available from the PROMIS Laboratory, University of Vermont College of Medicine, Burlington, Vt. 05401.

A Plea for Consistent Terminology

SIR: In "Psychotherapy of the Medically Ill" (January 1979 issue) Toksoz B. Karasu, M.D., used inconsistent terminology and limited conceptualization. Although he was dealing with the status of psychotherapy in general, he restricted his scope to psychosomatic conditions with the exception of a report on group therapy for postmyocardial patients.

Dr. Karasu omitted several important groups of patients. For many years liaison psychiatry has been involved in the diagnosis and treatment of psychiatric conditions that do not have psychosomatic etiologies. It has also dealt with sequelae of medical and surgical interventions and their impact on the psyche. Psychiatric support and psychotherapy for cancer patients are relatively new and important areas of psychiatric intervention that are missing from Dr. Karasu's review. Another important area excluded from the review is psychological management of the chronically ill and physically handicapped patient. Dr. Karasu also left out treatment of the child with congenital and chronic illness.

Psychotherapy results are generally more encouraging for medically ill patients who do not suffer from psychosomatic conditions than Dr. Karasu indicated. Examining both groups as controls might shed light on the different defense mechanisms these patients use.

Making "medically ill" synonymous with "psychosomatic" adds to confusion in a field where conversion symptoms, imaginary ailments, malingering, and psychosomatic conditions are often poorly defined and used interchangeably by many practitioners.

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Dr. Karasu Replies

SIR: I share Dr. Nir's concern about the terminology and scope of my paper "Psychotherapy of the Medically Ill." I intentionally selected the rubric "medically ill" because the term "psychosomatic" and the range of conditions within its purview are either restrictive or unclear at this time, an issue which reaches the heart of the current psychosomatic matter. A broader or less anachronistic terminology tends to promise more than it can deliver, since the controlled investigations I focused on still dwell disproportionately on the more classical psychosomatic disorders. My article was not nearly as confined to psychosomatic conditions as Dr. Nir claims; it included studies, albeit limited, of patients with chronic pain, migraine headaches, those who had undergone ileostomy surgery, menopausal women, patients with anorexia nervosa, and problems of obesity, all of which do represent active trends away from the most orthodox disorders.

Dr. Nir faulted the term "medically ill" for being too broad for its anticipated content. Ironically, he endowed the term "psychotherapy" with a breadth never intended. The introduction of some of the fine work that has been accomplished in the field of liaison psychiatry was an overextension, I feel, of the more structured practice of psychotherapy. By psychotherapy I meant certain specified forms of psychological intervention practiced today, on which the paper clearly pivoted. I never hoped to encompass all forms of psychological care (which would certainly make another article in itself). However, this delimitation was in no way meant to undermine the advances that this growing branch of psychiatry continues to make.

Dr. Nir placed in bold relief the fact that definitions of such complex domains as "psychosomatic" and "psychotherapy" need clarification and are, fortunately, still open to discussion and debate. I am indeed interested in locating any systematic studies of the efficacy of psychotherapy for illnesses other than those mentioned, especially if, as Dr. Nir claims, outcomes for such populations have been more encouraging than the ones I presented.

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Nutmeg and *Epená* Snuff: Differing Hallucinogens

SIR: In "Spice Cabinet Intoxication" (July 1978 issue) Robert A. Faguet, M.D., and Kay F. Rowland, Ph.D., de-

scribed the symptoms of a 19-year-old student who experienced self-intoxication with nutmeg.

These writers made two erroneous statements in their attempt to elucidate the cause for the near-hallucinatory condition of the patient. First, they stated that "myristicine" (myristicin) is the psychoactive ingredient of nutmeg (*Myristica fragrans*). No evidence in the scientific literature supports this statement. Indeed, a number of workers have attempted to identify the active principle, all without success.

The second error was a direct quote from Margaret Kreig's book *Green Medicine* (1, pp. 99–100) which described *epená* snuff intoxication as experienced by Professor Richard Evans Schultes. While the quote is correct, Faguet and Rowland indicated that these were symptoms of "myristicine intoxication via snuffing." In fact, there was no myristicin in the *epená* snuff that Schultes used. The snuff is prepared in South America from any one of several *Virola* species; *Virola* is classified in the plant family *Myristicaceae*. Schultes often refers to these materials as "Myristicaceous" snuffs, which apparently confused Faguet and Rowland. The active hallucinogens in *Virola* species (and *epená*) are derivatives of 5-hydroxytryptamine, which under normal circumstances would have no effects when used orally. How the natives discovered that the hallucinogenic effects of *epená* snuff could only be derived through nasal inhalation and not by taking the material orally remains a mystery.

Thus any resemblance between nutmeg intoxication and the effects described by Schultes and others following *epená* inhalation must be considered purely coincidental, and surely not caused by the same chemical compounds.

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NORMAN R. FARNSWORTH, PH.D.
Chicago, Ill.

Drs. Faguet and Rowland Reply

SIR: Professor Richard Evans Schultes (1) was our source for the disputed statement that myristicine is the psychoactive ingredient in nutmeg and *yakee* snuff (*epená*, *Virola*). He wrote: "The active principle is undoubtedly the same essential oil—*myristicine*—that is common throughout the family and makes our household nutmeg a dangerous narcotic when used in the appropriate amounts" (p. 107).

Apparently Schultes (2) later emended this view with respect to *yakee*. His later belief supports Dr. Farnsworth's comment that a tryptamine derivative causes the snuff's hallucinogenic effects. But tryptamines alone are ineffective in the human body. To produce a psychotomimetic effect, they must be taken with a monoamine oxidase inhibitor. According to Schultes (2), the B-carbolines, added to *yakee* in the preparation process (the *yakee* resin is mixed with the ashes of any of several MAOI-containing plants), act as monoamine oxidase inhibitors and allow tryptamine to produce its psychoactive effects.

While Dr. Farnsworth wonders how natives discovered that the hallucinogenic effects of *yakee* could be derived only through nasal inhalation, Dr. Schultes asks how "peoples in primitive societies, with no knowledge of chemistry or physiology, ever hit upon a solution to the activation of an alkaloid by a monoamine oxidase inhibitor" (2, p. 38).

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Dangerously High Doses of Haloperidol

SIR: The case report "Sudden Death in a Patient Taking Haloperidol" by Richard Ketai, M.D., and associates (January 1979 issue) was of special interest to me. I am not surprised that haloperidol was recently implicated in the sudden death of a 35-year-old mother of three. Increasing numbers of patients are being treated by rapid tranquilization with neuroleptic dosages that are much higher than generally found necessary. Indeed, the relative absence of α-adrenergic blockade and anticholinergic properties renders haloperidol a uniquely safe and effective antipsychotic agent for the acute patient. However, during the past year I have been concerned about the number of psychiatrists who administer 200–300 mg of haloperidol in 24 hours to acutely agitated and psychotic patients, viewed as "resistant" to much smaller dosages.

In our pilot study using haloperidol in the treatment of acute psychosis, we observed two distinct groups of patients: those whose psychosis and agitation responded to 40–60 mg of haloperidol given during the first 24 hours, and those occasional patients who clearly did not respond (1). For this latter group, the addition of amobarbital sodium, 500 mg I.M., to the haloperidol regimen proved consistently effective in relieving both psychosis and agitation while limiting the need for additional neuroleptic medication.

Since that time my clinical experience has repeatedly demonstrated that the combination of haloperidol and amobarbital sodium is dramatically effective and safe for acute psychotic patients. Further, this combination limits the dosage of the antipsychotic and avoids dystonic reactions. The merits of the rapid tranquilization technique are well documented. Modifying this technique in the context of safety remains a worthy objective for us all.

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Dr. Ketai Replies

SIR: I share Dr. Thornton's concern about very high doses of haloperidol. For the majority of acutely psychotic patients dosages of more than 60 mg of intramuscular haloperidol in 24 hours provide diminishing returns in respect to the likelihood of response. Occasionally, a patient will respond for the first time at doses greater than 100 mg/day, but potential

risks at these high doses suggest consideration of alternative approaches. For example, three patients on our ward recently complained of sudden respiratory distress and gagging while being treated acutely with standard doses of haloperidol. We noted these cases in light of the current report by Flaherty and Lahmeyer (1) about haloperidol-induced laryngeal-pharyngeal dystonia. We do not know whether higher doses of haloperidol would make this potentially dangerous side effect more likely to occur, but it would be wise to exercise caution for now. Our patients who had this side effect, like those previously reported, responded immediately to intravenous diphenhydramine.

Dr. Thornton's report of an enhanced antipsychotic response to 500 mg of intramuscular amobarbital sodium added to haloperidol in refractory patients is intriguing. We have used secobarbital and amobarbital in doses of 100–300 mg I.M. every 6 hours for several haloperidol nonresponders but have not been impressed by an increased antipsychotic response with this combination. If Dr. Thornton is correct this could be explained by an inadequate barbiturate dose. We do have better results by switching to a different neuroleptic, usually chlorpromazine, if the acutely psychotic patient has not responded to haloperidol in 3–5 days. Sometimes we observe that the addition of lithium is necessary before an adequate response results. This procedure should definitely be considered if mania has not been ruled out.

A study focusing on treatment options for acutely psychotic haloperidol nonresponders (at the 60 mg/day level) would be helpful. A blind comparison should at least be made between a switch to chlorpromazine versus addition of intramuscular amobarbital to the haloperidol regimen.

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Efficacy of Consultation-Liaison Settings

SIR: We wish to underscore the importance of educational research such as that reported by William W. Weddington, Jr., M.D., and associates in "Consultation-Liaison Versus Other Psychiatry Clerkships: A Comparison of Learning Outcomes and Student Reactions" (December 1978 issue). Reifler and Eaton (1) have pointed out the lack of outcome studies in this area.

The Weddington and associates article demonstrates that the consultation/liaison setting is at least as effective as more traditional psychiatric settings when performance on standardized testing is used to measure learning outcome. In our own health care center, during the June 1977–July 1978 academic year 19 students rotated through the consultation/liaison service, while 113 were assigned to one of six inpatient services or an emergency room service. All students then viewed a videotaped clinical interview, and their clinical knowledge was individually rated by oral examination as fail, low pass, high pass, or honors. Consultation/liaison students were more frequently rated as achieving high pass or honors status than students on other services ($\chi^2=8.88$, $df=3$, $p<.05$). There were no significant differences in the low pass and pass categories. No students failed. Since students were not randomly assigned to their clinical rotations, these results are necessarily biased. Nevertheless, they are suggestive.

The Report of the President's Commission on Mental Health, which indicated that over 50% of psychiatric care is delivered by nonpsychiatrists, suggests that most psychiatric care may continue to be delivered in traditional medical settings (2). The consultation/liaison setting may, therefore, be a particularly meaningful one for educating students who do not plan to specialize in psychiatry, since it emphasizes medical-psychiatric integration.

Long-term outcome studies are needed to assess the effectiveness of various psychiatric settings in educating medical students, especially those who do not specialize in psychiatry.

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Meta-Janusian Thinking

SIR: In his interesting paper, "Einstein's Creative Thinking and the General Theory of Relativity: A Documented Report" (January 1979 issue), Albert Rothenberg, M.D., helps clarify the nature of scientific creativity, which he views as the product of active secondary process thinking, during which "Janusian thinking" takes place. Here, the formulation of simultaneous opposite ideas results in a creative discovery, an example of which is the theory of relativity, in which Einstein reconciled an apparent contradiction—that an object standing still could be moving at the same time. Dr. Rothenberg emphatically states that Janusian thinking, which also occurs in literary and artistic creativity, is not prelogical, illogical, or "schizophrenic" thinking.

Nevertheless, it is striking how remarkably similar Janusian thinking is to one of Bleuler's primary symptoms of schizophrenia, ambivalence. This poorly understood concept is described as the simultaneous expression of opposite ideas or feelings (1). In addition, illusory feelings of creativity and inspiration are often reported by individuals with functional psychoses and users of hallucinogens, who can become preoccupied with the contradictions of the universe. In such circumstances, however, the lack of an intact ego seems to diminish creativity (2, 3), as can be seen in the works of Van Gogh, whose talent deteriorated during his psychotic disorganization (2).

Is there really no connection whatsoever between simultaneous opposite thoughts that result in creativity and those which result in an illusion of creativity? Perhaps Janusian thinking itself is Janusian in nature. If so, then this very creative, sophisticated, and logical type of thinking would at the same time be regressive, childlike, and illogical.

To reconcile this contradiction of paradoxical thinking, a hypothesis that incorporates cognitive development, such as the following, might be helpful: The retention of certain preoperational (4) thought patterns into adulthood enables them to be activated during both psychosis and Janusian thinking. In psychosis they are fragmented and appear bizarre and pseudo-creative. In Janusian thinking, they are harnessed in the service of secondary process and result in creativity and sophisticated cognition. (Such thinking could have evolutionary implications.)

In summary, the inherent magical thinking of the child, who quite easily tolerates contradiction of logic, might be the common denominator of both Janusian thinking and Bleuler's ambivalence.

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IRA BRENNER, M.D.
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Dr. Rothenberg Replies

SIR: In my forthcoming book (1), I discuss the relationship between Bleuler's ambivalence and Janusian thinking. Briefly, Bleuler described three types of ambivalence in schizophrenia: affective ambivalence, ambivalence of will, and intellectual ambivalence (2). It is difficult, as Bleuler himself pointed out, to draw hard and fast distinctions among these types. By and large, the term "ambivalence" is now used only to refer to the types involving affect or will, which are virtually indistinguishable. Although we seldom refer to intellectual ambivalence, it does appear in schizophrenia and, although superficially resembling Janusian thinking, it is markedly dissimilar.

When the schizophrenic patient displaying intellectual ambivalence says, "I am a human being like yourself, but I am not a human being" (Bleuler's example), he is not using Janusian thinking because he is not aware of the impossibility of the statement. He neither recognizes the contradiction nor intends the remark to be taken figuratively; he believes it to be true literally. As with primary process thinking, opposites are considered equivalent. One of the reasons for this is that schizophrenic persons seem to have difficulty in forming conceptual categories (3). As opposites are similar to each other in many respects, despite their essential sharp difference and contradiction, the schizophrenic person tends to categorize them together on the basis of the immediately perceived superficial similarity. As for the affective or will type of ambivalence, it differs from Janusian thinking in the lack of conceptual simultaneity of opposition. One does not consciously experience love and hate simultaneously—it must be inferred by him, and by others, from his behavior.

Nevertheless, I think Dr. Brenner's suggestion that there is a developmental connection between the cognition of creativity and that of psychotic or regressive thinking, the "illusion of creativity," is valid. In my research with highly creative writers, artists, and scientists, I have collected a great many data indicating a particular similarity between the backgrounds of creative and psychotic people. Both tend to come from family circumstances in which there is seriously distorted parent-child communication. Both are required to cope with verbal and nonverbal incongruities, and both learn to attend more carefully than other children to subtle and overt aspects of parental communication. The incidence of frank parental psychosis is high in both groups. An important difference is that there is seldom frank psychosis or severe psychiatric illness in both parents of the creative person. In a large number of the cases I have studied, psychosis or severe disturbance in one of the parents is rather strongly balanced by a relatively healthy mate. The prototype for this family structure has, in fact, been immortalized in an extremely creative playwright's well-known drama about his own life, "Long Day's Journey Into Night," in which O'Neill poignantly documented his experience with severe family communication difficulties and a seriously disturbed parent.

Psychotic and creative persons seem to be exposed to similar forces that require them to question and even to ignore the fundamental tenet of ordinary logic that something cannot be both true and not-true at the same time. The creative person, however, is able to espouse seemingly illogical Janusian constructs with clear rationality because of developmental experience with the healthy logic of an intact parent. Thus, psychotic and creative thinking are related developmentally and at times appear similar on the surface. I must, however, correct Dr. Brenner on some particular points. Einstein did not say that "an object standing still could be moving at the same time," but that an observer in motion would be at rest at the same time. In this case, the opposites involved are not symmetrically reciprocal—stationary objects do not, in Einstein's formulation, move. Also, Janusian thinking does not involve the "harnessing" of regressed, primitive thought. That would be similar to the idea Ernst Kris (4) proposed in his famous concept of creativity as "regression in the service of the ego." Janusian thinking instead is a form of high-level cognition at the very moment of its activation, radically and dramatically different from—although superficially resembling—primitive and regressive conceptualizing.

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ALBERT ROTHENBERG, M.D.
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A New Form of Polypharmacy?

SIR: Daniel S.P. Schubert, M.D., Ph.D., has presented the ultimate case of polypharmacy: the use of bethanechol chloride to counteract anticholinergic effects (blurred vision and dry mouth) in a patient treated with the combination of flu-

phenazine and benztropine mesylate ("Use of Bethanechol Chloride with Phenothiazine: A Case Report," January 1979 issue). He stated that the patient "was given 20 mg of fluphenazine and 8 mg of benztropine mesylate p.o., but because of periodic noncompliance, fluphenazine decanoate, 25 mg I.M., was added." Unfortunately, neither the interval nor the duration of administration is specified (an error that the reviewers should have caught). One can only presume that the dosages of oral medications were per day and the injections were given weekly or biweekly. Might I suggest that the author try the more commonly accepted guidelines for prescribing (1) and consider the following alternatives:

1. When side effects occur with neuroleptics, consider decreasing the dose before adding an antiparkinsonian agent.
2. Use antiparkinsonian agents only as necessary, in the lowest possible doses and for the shortest period of time. Generally, there is little need for long-term maintenance.
3. When noncompliance arises, thoroughly discuss the problem with the patient and look for other alternatives before initiating treatment with long-acting injectable preparations.

In addition, Dr. Schubert's case report leaves the following questions unanswered: Why was this patient treated with benztropine mesylate in the first place, and why was it continued? Why was a dosage of 8 mg necessary when 1–4 mg/day in divided doses is generally sufficient to counteract neuroleptic-induced parkinsonism? Why was the same dosage of neuroleptic necessary if the patient's "psychosis receded"? Why was compliance such an issue if he had previously been "relatively clear between episodes"? How was the diagnosis of chronic schizophrenia made, and was the possibility of an affective disorder considered in light of his illness course? And finally, why add another drug with an additional side effect profile and increase the risk of drug interactions?

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Dr. Schubert Replies

SIR: Sackett and Haynes (1) gave a weighted estimate of about 50% noncompliance with the therapeutic regimen for patients in all specialties. They then indicated that compliance is even poorer among psychiatric patients than those of other medical specialties. Blackwell (2) discussed side effects as one source of noncompliance. My paper was, as indicated in the title, about the use of bethanechol chloride with phenothiazines. This was an extension of Everett's suggestions (3) on the use of bethanechol chloride with tricyclic antidepressants because both phenothiazines and tricyclics possess anticholinergic activity as a side effect. Thus, my intent was to suggest a way to decrease side effects of phenothiazines when they are of the anticholinergic variety. Such symptoms can be severe and tend to lead to noncompliance. Benztropine mesylate shares the anticholinergic property with the phenothiazines and tricyclics and may produce more severe anticholinergic side effects.

I did not suggest that benztropine mesylate or other antiparkinsonian agents be used for parkinsonian-type side effects with phenothiazines. This I leave to others, such as Klein and Davis (4).

In addition to dealing with noncompliance, physicians should attempt to enhance the functioning and comfort of their patients. When medications are the treatment of choice, side effects should be taken as seriously and treated as vigorously as other syndromes.

Bethanechol chloride is also convenient as a test for elements of an anticholinergic (atropine) psychosis while continuing antipsychotic (or antidepressant) treatment.

With regard to Dr. Winstead's paragraph of questions, publication space is limited. However, I would challenge him to present a case that answers all possible questions.

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DANIEL SCHUBERT, M.D.
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Another Approach to Hyperpyrexia

SIR: Stephen C. Mann, M.D., and William P. Boger, M.D., did a service in reminding us of the hazards involved in the use of phenothiazines with patients exposed to high temperatures ("Psychotropic Drugs, Summer Heat and Humidity, and Hyperpyrexia: A Danger Restated," September 1978 issue). However, as Peele (1) noted in his letter commenting on their article and a related one (2): "We need to be careful to avoid automatically attributing a patient's hyperpyrexia to medication." He suggested that the diagnosis of lethal catatonia must also be considered. I would like to provide a case where a psychiatric illness, in a rather different way, was the cause of hyperpyrexia.

Mr. A, a 50-year-old man with chronic schizophrenia, was admitted to the hospital on a hot August day in a comatose state with rectal temperatures reported as 106° F. Vigorous measures were instituted and a rapid, extensive evaluation that included complete blood studies, a lumbar puncture, EEG, and CT scan revealed no specific etiology for the loss of consciousness other than the temperature. It was assumed at the time that the fever was due to heat stroke secondary to the combination of a phenothiazine and an antiparkinsonian agent (which were known to have been previously prescribed) resulting in faulty thermoregulation on a very hot day.

However, once the patient regained consciousness and his son became available for interview, it was learned that Mr. A had not taken his medication in months. He had become progressively more paranoid and was constantly terrified of "beams and rays" causing damage to his body and of strangers poisoning him. The patient began to wear six layers of clothing at all times to deflect the "rays," kept the windows of his tiny basement-level apartment sealed, and ate and drank little. This behavior continued during an August week of temperatures in the high 90s, at the end of which the patient was admitted. Thus, it appeared that the patient's psy-

chosis was ultimately responsible for the heat stroke through the intermediacy of paranoid delusions.

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Countering Diazepam Abuse

SIR: Diazepam has been the most frequently prescribed drug in the United States since the early 1970s (1). It is also a unique drug—overcompliance rather than noncompliance is often observed with diazepam. Indeed, about 7% of all psychiatric admissions have a history of such sedative abuse (2).

The pharmacy department at the Buffalo General Hospital Community Mental Health Center surveyed all new diazepam prescriptions written in the emergency room during one year. The most disturbing finding was that a number of patients who were wise to the shift rotations of different psychiatrists had managed to receive several prescriptions for diazepam within a three- to four-day period. In two cases, individuals had gotten enough diazepam to last three months. The pharmacy department, wishing to combat this diazepam abuse, compiled a list of diazepam "addicts" (regular seekers of new diazepam prescriptions) and posted it in the emergency room for psychiatrists to note before prescribing diazepam to anyone. Furthermore, a procedure was developed whereby patients who received diazepam prescriptions were given only a seven-day supply, with no refills authorized.

The pharmacy department then monitored all diazepam prescriptions written during the next year. Although these measures did not completely prevent suspected diazepam abusers from trying to obtain a quick, easy diazepam order, 28% fewer prescriptions were written compared with the previous year. Many addicts were finally confronted and made to realize that diazepam was not the panacea for all their problems. Some 24 of the 31 major abusers almost doubled the number of their appointments with assigned counselors. This gave them more opportunity to ventilate anxiety and everyday pressures than the drug could provide.

We believe that stricter control over diazepam prescription practices may help decrease dependency rates and encourage patients to make more use of their assigned counselors in learning to live with anxiety; it is addiction to anxiety that can lead to addiction to diazepam.

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The Group Alternative in Private Practice

SIR: The Topical Papers on the future of mental health care delivery systems (November 1978 issue) are timely and deal with issues that should receive constant and continued attention.

However, in "Private Practice of Psychiatry: Future Roles," Zigmond M. Lebensohn, M.D., missed an important trend in private practice which offers some of the advantages of clinic practice without the red tape, the costs, or the mixed quality of services in a large bureaucratic system.

I refer to small, multidisciplinary, private group practice, which has increased rapidly in the past three years in Massachusetts. Groups run by psychiatrists (or psychologists), usually staffed by senior social workers and psychologists, supply long-term, short-term, group, and other modalities of services. They operate at the time of day they are needed, at a cost far below that possible in a community mental health center. The groups offer psychiatrists the opportunity to expand the use of their services while partaking of the skills of others. They also do away with the isolation of solo practice. I take issue with Dr. Lebensohn's statement on the futility of cost comparison. I believe specific attention should be paid to costs since this frequently determines where services are purchased and which services receive government support. Community mental health centers and community clinics are usually not neighborhood clinics, but serve a very broad catchment area. Statistics on the cost of these services are frequently suspect because they include only the clinic costs and not the cost of the state administrative hierarchy or the pension system, all of which eventually comes out of the taxpayers' pocket. The fact that these clinics do not pay taxes, which further increases their cost per unit of service to the community (compared with a private group which does pay) is also overlooked.

As a former general practitioner and a current community psychiatric practitioner, I am distressed over mental health facilities which look like community or neighborhood centers on planning maps or charts but are actually far removed from most of the recipients of their services. Those planning such mental health facilities ignore the existence of different types of neighborhoods in any small community, let alone in a catchment area.

Family practice is returning in medicine; family and community psychiatry should also be encouraged.

JOHN C. CONIARIS, M.D.
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Dr. Lebensohn Replies

SIR: Dr. Coniaris has called our attention to a form of group practice that can provide good psychiatric care without red tape or excessive costs. The points he made are important and well taken. I regret that shortage of space prevented me from developing them more fully.

In APA Task Force Report Number 6 (1) there is considerable discussion of multidisciplinary private group practice (pp. 9–10). The quality of care in such groups is usually excellent and can be delivered at a cost much lower than that of most community mental health centers. Unfortunately, the distribution of these groups is still spotty. Their growth depends on individual initiative and should be encouraged.

When comparing costs, I found it necessary to rely on published data. The studies by Sharfstein and associates (2)

of 53 CMCHs indicated that the cost of one hour's psychotherapy by a psychiatrist in a community mental health center "could be approximated at $45." However, some of the centers surveyed estimated $70 per hour. I would agree with Dr. Coniaris that if one considered all the hidden expenses, a treatment hour in a community mental health center might cost nearly $100. Dr. Coniaris stressed the importance of including the cost of the state administrators and retirement system which "comes out of the taxpayer's pocket." The fact that these clinics do not pay taxes is also significant.

I agree that community clinics often serve rather broad catchment areas and are not really neighborhood clinics. This point deserves repeated emphasis. I have written about this in greater detail in "Pilgrim's Progress: Or the Tortuous Road to Mental Health" (3).

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Amantadine and α-Methyldopa: Effects and Interactions

SIR: In "Amantadine-Induced Psychosis in a Geriatric Patient with Renal Disease" (January 1979 issue) Richard Borison, M.D., Ph.D., made the point that amantadine "should be used only after careful clinical evaluation of renal function, particularly in the case of the geriatric patient." The case history well illustrates this point. However, it might also as well be used to illustrate some perhaps more fundamental principles of drug treatment that probably would have prevented the patient's ensuing complications.

1. One should avoid polypharmacy if possible. Parkinson's disease was only suspected, apparently on the basis only of a "bilateral resting and intention tremor of the hands." The patient was, however, described as caring adequately for herself and actively engaged socially in her church organization. Patients with early parkinsonism may have clinical features that warrant medical consultation, but if these symptoms cause no more than mild embarrassment or minimal inconvenience it may be best not to treat them because the chances of adverse effects with any of the available drug treatments probably exceed any likely benefits (1). Adverse effects would, of course, be of even more concern in a patient already taking multiple medications for multiple chronic medical problems. In addition, the patient's standing medication included α-methyldopa, 250 mg q.i.d., which in itself may cause parkinsonism as a side effect (2). Thus if the patient's symptoms were severe enough to warrant treatment, one might first consider altering the antihypertensive medication before adding a new drug.

2. If multiple drugs are prescribed, one should be aware of their possible interactions. As noted, the patient was taking α-methyldopa, which has profound effects, not to mention its main therapeutic mechanism of action on CNS dopaminergic systems, the same neurotransmitter systems that amantadine presumably affects in its therapeutic usefulness for parkinsonism. α-Methyldopa has been implicated, usually in association with other drugs that also affect central dopaminergic systems (e.g., haloperidol), in the causation of paranoid hallucinatory states (3). Dr. Borison correctly referred to Postma and Tilburg's 13 collected cases of apparent amantadine-induced psychosis in patients who were elderly, some in renal failure. However, 11 of their 13 patients were also taking other medications known to affect CNS neurotransmitter systems (one was taking methyldopa, 250 mg three times a day), which makes it pertinent to consider drug interaction in the etiology of these patients' psychoses (4).

3. In prescribing a particular drug, one should be aware of its pharmacokinetics and the clinical guidelines on dosage, which should be broken only with good reason. This patient was started on amantadine, 100 mg t.i.d. It is unusual for therapeutic benefits of amantadine to outweigh side effects at dosages above 200 mg. In the *Physicians' Desk Reference* the recommended starting dose of amantadine is 100 mg b.i.d., or in patients with serious medical problems, 100 mg q.d. Patients taking amantadine can develop congestive heart failure, which this patient already suffered from. In addition, as Dr. Borison stated, amantadine is in great part not metabolized but rather excreted in the kidney, and dosage therefore should be carefully introduced in someone who is known to have chronic renal failure. However, methyldopa is similarly excreted rather than metabolized, and the interaction of these drugs may have been therefore intensified. Indeed, bradycardia is a reported side effect of methyldopa, and one wonders about its relationship to the patient's chief complaint of bradycardia.

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Dr. Borison Replies

SIR: I agree wholeheartedly that the use of multiple drugs presents the possibility of potentially toxic drug interactions, particularly in a patient with multisystem disease. In response to the first and third points discussed by Dr. Cohen, I agree that the patient's apparent early parkinsonism was treated too aggressively. In fact, on closer neurologic examination, the patient's muscle tone was not affected, there was no bradykinesia, nor was there a compromise of postural reflexes. When further history was obtained, the patient revealed that her father and paternal grandfather both had tremors of the upper extremities. With this information, the

final diagnosis arrived at was benign familial tremor, rather than Parkinson's disease.

I must take issue with Dr. Cohen's strong emphasis on the interaction of α-methyldopa and amantadine as being potentially responsible for the patient's psychosis. Both drugs do affect dopamine systems; however, they do so in a differential fashion. Methyldopa is metabolized to the partial agonist methyldopamine, which depletes brain dopamine by serving as a false neurotransmitter, whereas amantadine has been suggested to either release or block the reuptake of presynaptic dopamine. Thus, if amantadine blocks reuptake, it would attenuate the actions of methyldopa. Alternatively, if amantadine releases stored presynaptic amines, it would release methyldopamine, which is a partial dopamine receptor agonist. Therefore, in either case, the interaction of amantadine and methyldopa would not be expected to result in a psychotic state. I also take issue with the comparison of the methyldopa-haloperidol interaction with a methyldopa-amantadine interaction. Haloperidol is a blocker of postsynaptic dopamine receptors, a mechanism of action clearly different than that of amantadine. Moreover, the addition of methyldopa to haloperidol therapy may in fact enhance its antipsychotic actions (1).

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Psychoanalytic Theory and the Dilemma of Precision

SIR: Anthony L. LaBruzza, M.D., in "The Activation-Synthesis Hypothesis of Dreams: A Theoretical Note," his criticism of Drs. Hobson and McCarley (December 1978 issue), may not have realized how his reliance on the concept of confusion of independent logical orders weakens his argument. The more psychoanalytic theory defends itself by refusing even the modest, "less exact" isomorphism used by Drs. McCarley and Hobson, the more it resembles an enterprise like scholastic philosophy. While a medieval theologian transported into a modern physics department might appear to ward off critics by the "no isomorphism" argument, that very security would erode his claims to describe reality as a candid look at the history of ideas demonstrates. The analogy to scholasticism suggests that the precision aspired to by psychoanalytic theory opens it to attack from other logical orders. Hard science leaves more modest psychologies and vaguer theologies in peace, if only out of politeness. A theory's explanatory power may not be subject to refutation for reasons summed up by Dr. LaBruzza and Gerald W. Vogel, M.D., in "An Alternative View of the Neurobiology of Dreaming," but its own inability to meet rough and ready challenges from other, more rigorously testable logical orders may weaken it fatally. An irrefutable place is not necessarily a safe place to be.

I thought Dr. Vogel's neurophysiologic comments a much more formidable criticism of McCarley and Hobson, although not supportive of analytic theory.

HUGH C. MURRAY, M.D.
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Dr. LaBruzza Replies

SIR: If Dr. Murray feels that, like a modern Thomas Aquinas, I have come to the defense of psychoanalytic dogma with "irrefutable" logic, I am flattered by the comparison, but he has misunderstood me.

I simply wanted to point out a major logical flaw in Hobson and McCarley's reasoning and thus challenge their conclusion that "the primary motive for the dream language and dream process cannot be disguise if the prime force of dreams is not an instinct or repressed 'wish' in need of disguise" (p. 1219). The terms "motive" and "force" belong to different logical orders and are not freely translatable to one another.

There is no argument that the firing of cells in the pons or anywhere in the nervous system is motivationally neutral. Are we to conclude, however, that because the neurobiological substrate for human behavior is the motivationally neutral firing of neurons, human behavior cannot be motivated? This ineluctable conclusion of Hobson and McCarley's line of reasoning flies in the face of clinical and human experience.

Hobson and McCarley appeal to a mind-body isomorphism that has not yet been scientifically demonstrated. At this point in the history of science such an isomorphism is a matter of faith. I do believe that an isomorphism or a superordinate framework for discussing meaning and physiology simultaneously will be discovered. I am not taking a stand for psychoanalysis and against empirical science. Both are necessary and valuable; neither should be accepted uncritically.

It would be unfortunate if our profession became polarized into warring factions, with psychoanalysis at one extreme and hard science at the other. Thoughtful scientists and theorists are sensitive to issues of multiple levels of explanation and their distinct, nontranslatable languages and methodologies (1–3). I highly recommend Kety's article (1) in this regard.

To cite a familiar proverb, we are all blind men touching upon various parts of the elephant. Together we might catch a glimpse of the whole.

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BOOK REVIEWS

Legal and Ethical Issues in Human Research and Treatment: Psychopharmacologic Considerations, *edited by Donald M. Gallant, M.D., and Robert Force, LL.M. New York, N.Y., SP Medical & Scientific Books (Spectrum Publications) (Halsted Press, John Wiley & Sons, distributor), 1978, 180 pp., $15.00.*

This volume is the result of a symposium sponsored by the American College of Neuropsychopharmacology. The list of contributors is distinguished, representing the fields of law, ethics, philosophy, and psychiatric research.

The book is divided into six chapters with a preface. The first chapter is a presentation of the American College of Neuropsychopharmacology's principles of ethical conduct for research on human subjects. This document has been extensively reviewed and serves as a useful guideline for all researchers. The next two chapters review issues of current litigation and law as they affect psychopharmacological research and treatment. Chapters four and five review the ethical issues of psychopharmacological research and treatment, respectively. The last chapter, developed by the two editors, details some specific recommendations for public policy and individual action in this area.

Each chapter is followed by comments from a variety of experts in the field. Judicious editing has made these discourses lively and relevant. The comments are directly related to the position papers they follow and present opposing or clarifying points of view.

The area of human rights and research has been widely reviewed and debated since the end of World War II. The Nuremberg trials served to direct attention to the potential for abuse of individual rights and human dignity in the name of scientific research. An outgrowth of those trials was the Nuremberg Code, which continues to set forth fundamental principles that should govern all human research. The last decade has seen an increased awareness of individual rights coupled with a distrust of intellectual and scientific pursuits. Such concerns, added to the history of Nuremberg, have stimulated a debate about human biomedical research. Added to these general concerns have been the awareness of the potential of behavior control and the misuse of psychiatry for political or social purposes. One wonders whether this latter controversy would have developed if there had not been solid basic scientific work with significant advances in clinical care. How else can one understand the demand for access to treatment concurrent with a move to curtail research?

Most of the experts in this volume develop positions that seem to polarize the interest of "the scientist" and "the public." It is interesting that the public position is detailed by "experts" who speak in behalf of a presumed public stance and/or ethical, moral position. One is left with the feeling that they have been sanctioned to act on the behalf of others; of course, this is not the case. In that sense, all of the public sector protagonists are acting *in loco parentis,* a curious position, it seems to me, for those who claim to speak for human rights, particularly the right of self-determination and individual dignity. This is a side issue because "the public" must always have a voice, even if it is a self-appointed one.

To my mind, the issue has been unfortunately limited, centering on such various concrete issues as informed consent, selection of subjects, and right to treatment. I think it is probably natural that the issues arise around specific issues; however, such narrowness risks missing the broader problems that lie behind these specifics. The conflicts of the values, priorities, and philosophical positions that are the basis for the controversy remain obscure. In our pluralistic society, particularly as social changes occur, it would be surprising if we did not have such arguments. It is unlikely that in the foreseeable future conflicts in values will be easily resolved. Furthermore, the debate is likely to remain narrow and concrete.

As for the book itself, I found it an excellent volume. There are good reviews of the current legal status of research and aspects of psychiatric care. Issues such as the right to treatment, informed consent, and malpractice applications to research are adequately covered. The legal opinions and statutes cited are interpreted by people of opposing views, thus leaving one a sense of the liveliness and continued evolution of this field. The liveliness of the field underlines the fact that any publication must be a bit out of date even when just published.

I would have found it a bit more satisfying to have the ethical issues presented before the review of the legal material so that the underlying issues could be identified and thus form the basis for the practical legal debate. It was somewhat disappointing that the ethical and philosophical sections do not include as broad a review as might be desirable of the basic debates and various historical positions concerning human dignity, individual and public rights, and related matters. For those not thoroughly familiar with the relevant aspects of philosophy, this would have been an extremely useful addition to the book that would have helped focus the various arguments. There are places in the volume where these matters are addressed, but usually in a narrow or fragmentary way. Occasionally, the positions are concretized into adversarial arguments that are narrow and legalistic and distort the philosophical assumptions that underlie them.

In spite of these reservations, I am happy to recommend the book. I believe it is the best volume I have seen on its subject. It balances the various positions in these controversial areas. I recommend it as must reading to all researchers who are interested in clinical as well as basic science problems. In addition, lawyers and the judiciary interested in psychiatric research and treatment will find the book interesting. Those involved in public policy and administration of hospitals and medical schools will also find something of use, as well as specialists in philosophy, bioethics, and history of science. These specialists may look to additional readings and wish for more detail in their own areas; nonetheless, the book is an outstanding source of current review.

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Phencyclidine (PCP) Abuse: An Appraisal. NIDA Research Monograph 21, *edited by Robert C. Petersen, Ph.D., and Richard C. Stillman, M.D. Rockville, Md., National Institute on Drug Abuse, 1978, 313 pp., no price listed (paper).*

Useful books are timeless or timely. This one is timely. The major clinical event of the 1970s has been phencyclidine abuse. It was virtually a nonexistent clinical entity before 1970, but now it constitutes a prominent hospital admission category in some cities. One of eight young adults has tried phencyclidine. More than the effects of abuse of any other substance, the organic brain syndrome associated with phencyclidine abuse seems to mimic schizophrenia, and phencyclidine use appears to produce an extreme exacerbation of the signs and symptoms of schizophrenic patients. However, phencyclidine has gone largely unnoticed by psychiatry.

This book evolved from a conference held by the National Institute on Drug Abuse in February 1978. So we have the rarity of a book published the same year as the conference on which it is based. The price for the speed is some redundancy, some errors, and no index. The redundancies are bearable for a fresh field; the errors are not lethal but consist of things like labeling chapter 17 as chapter 12. The lack of an index, however, is quite disappointing because this is the only available reference book.

Knowledge about phencyclidine (PCP, Sernyl, Sernylan, angel dust, elephant tranquilizer, and a dozen other street names) has developed only over the past two decades. It produces a stimulant response in mice, calms guinea pigs, and anesthetizes many other animals. Rhesus monkeys like it so much it is used as a reinforcing agent (unlike hallucinogens). For man, the possible effects include all of the above plus many others. It is lethal in high doses, and in lower doses it can produce a variety of behaviors that remind one of schizophrenia. One contributor to this book, Paul Luisada, states, only partly in jest, "The widespread use of phencyclidine in our catchment area might even be viewed as a crude 'screening test' for 'schizophrenicity.' " How the drug produces any of these phenomena is a mystery.

Also a mystery is why monkeys and man should willingly take the drug. It has always had a poor reputation on the street, and some negative effects are felt virtually every time it is taken. Along with the negative effects, two-thirds of the time there is a change in feelings that is regarded as positive enough to attract, even to habituate. Habituation, it is hypothesized, occurs with those who do not like their "normal" feelings or with those who enjoy a reduction in their own basic drives to the point where they can feel quite content with being nothing and having nothing. These hypotheses have considerable explanatory power, but the test for clinicians is the hypotheses' usefulness in treating patients, and day-to-day management does not yet include such concepts.

Treatment for phencyclidine abuse derives more directly from basic research than is usual with many mental illnesses. Because phencyclidine excretion has been found to be markedly increased in acidified urine, therapy consists of acidification of the urine. Because basic experiments found increased disturbances with increased stimuli, therapy consists of stimuli reduction. Laboratory experiments also suggest the use of haloperidol, not chlorpromazine, but this is not universally accepted.

What about prevention? Giving the drug bad publicity will not work alone—it has had a bad press for over a decade. Increasing penalties for illegal manufacturing and distribution will only put it out of the price range of a few. With today's limited prevention skills, phencyclidine use will probably run a course like cocaine use, with periods of popularity. Psychiatry will have to respond effectively to many citizens whose phencyclidine use produces a brain syndrome and to the few who seek treatment for their habituation. For the professional, this book provides all we know about phencyclidine.

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The Woman Patient: Medical and Psychological Interfaces, Vol. 1: Sexual and Reproductive Aspects of Women's Health, *edited by Malkah T. Notman, M.D., and Carol C. Nadelson, M.D. New York, N.Y., Plenum Press, 1978, 358 pp., $19.50.*

This 25-chapter book is generally well written and carefully thought out. It is understandable to interested people who are not in one of the health professions and presents in one volume contemporary feminist views on all aspects of women's sexual functioning. *The Woman Patient* can appropriately be used as a reference for both women and men who want to know more about what obstetricians, gynecologists, and psychiatrists are doing with and for their female patients. The information is sound and, in most instances, carefully documented. The references and the glossary should prove highly useful to patients for years to come.

Chapters that proved of special interest to me were "The COPE Story: A Service to Pregnant and Postpartum Women" by Turner and Izzi, the review of the history of contraception in the United States by Peter Reich, and Kahan and Gachill's chapter on "The Difficult Patient: Observations on the Staff-Patient Interaction."

COPE, which stands for Coping with the Overall Pregnancy/Parenting Experience, is a self-help group program started by Turner in 1972. It now has 20 chapters in the metropolitan Boston area. Turner and Izzi describe COPE groups as "supportive and focused on problem solving" with the immediate goal of tension reduction. As described in the chapter, COPE offers a prototype that, if it becomes more widespread, could prove invaluable to many women during and after their pregnancies.

Both men and women psychiatrists can profit from familiarizing themselves with the views so cogently expressed in this book. Few physicians would be hesitant to recommend the book to patients. By reading this book, all health-care professionals who deal with women would have a fuller understanding of these patients and their many worries. We can all look forward to the future volumes of this promising series.

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The Mind in Sleep: Psychology and Psychophysiology, *edited by Arthur M. Arkin, John S. Antrobus, and Steven J. Ellman. New York, N.Y., Lawrence Erlbaum Associates (Halsted Press, John Wiley & Sons, distributor), 1978, 624 pp., $29.95.*

This book is a comprehensive review of EEG studies of the psychology and psychophysiology of sleep through 1975. It contains no review of neurophysiological aspects of the sleep cycle but is otherwise a worthy successor to Kleit-

man's encyclopedic work (1), which summarized the initial phase of modern sleep research.

Individual chapters effectively discuss various topics that have generated much research and controversy, such as the question of non-REM dreaming. (The distinction between content elicited on awakening from REM and non-REM sleep is less clear than originally believed, although REM content is still considered to be more dreamlike.) Other chapters discuss mentation associated with sleep onset, which can also be dreamlike; studies of dream recall (factors besides repression seem most important in normal people); the connection between tonic state, phasic events such as eye movements, and sleep mentation (not impressive); the effects of various external stimuli and drugs on sleep experience; and the effects of deprivation of REM sleep, which are often unclear and apparently contradictory. Another long chapter reviews such material of psychiatric interest as normative dream content, relation of dreams to psychopathology, psychophysiological parallelism, and dream meaning. I missed only a chapter on the psychological effects of total sleep deprivation, which would have been relevant to recent contentions that sleep is an archaic instinct serving no necessary function (2).

With an approach like that of the *Psychological Bulletin*, the authors have made a major contribution by reducing a huge mass of data to an accessible and relatively digestible form. Similarly, they present meticulous consideration of methodological issues. These critiques run the gamut from incisive clarification of ambiguous procedures, which will be essential guides for future research, to irrelevant carping.

The principal limitations of the book are in the areas of conceptual background and integration of the material. Much of the conceptual background, such as psychoanalytic theory, structuralism, psycholinguistics, cognitive psychology, and neuropsychology, has recently been well reviewed by Foulkes (3). His volume would serve as a companion to the present volume. The issue of integration reflects an enduring problem in sleep research. Maintenance of the interdisciplinary perspective that spawned the field has become increasingly difficult as workers have become mired down in parochial matters. Integrative attempts are made in the final two chapters. Although I agree with most of the authors' conclusions, the ideas presented in these chapters are separated by a significant gulf from the data of the previous chapters and also leave out a lot.

In summary, this work is a good presentation of the state of the art in sleep psychology.

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Psychosomatic Disorders in Childhood, *by Melitta Sperling, M.D.; edited by Otto E. Sperling, M.D. New York, N.Y., Jason Aronson, 1978, 406 pp., $20.00.*

A volume that is given the all-inclusive title *Psychosomatic Disorders in Childhood* and is written by a highly respected psychoanalyst, pediatrician, and teacher promises much, and this is unfortunate. In some respects the reader will not be disappointed. Melitta Sperling spent a lifetime working with (and curing) children suffering from psychosomatic problems. This posthumous book gives eloquent testimony to the clinical sensitivity, the masterful technique, and the patient perseverance of a superb analyst and therapist. Virtually every chapter contains a treasure of clinical detail, thought-provoking psychodynamic hypotheses, and treatment strategies that will be read with much profit not only by those who work with disturbed children but by all therapists who grapple with the problems of regressed patients with infantile personality organization.

The reader who is led by the title to expect a comprehensive examination of the current state of psychosomatic disorders in childhood, however, will likely be frustrated. Unlike many recent writers on psychosomatic disorders who affectionately but firmly place classical psychoanalytic insights in the perspective afforded by experimental and clinical studies of the past two decades (e.g., Herbert Weiner [1]), Sperling largely ignores or disregards these studies. Of some 380 references, only 11, including 3 references to her own work, are from the present decade. There is a charming but clearly anachronistic 1950s quality throughout, especially with respect to the author's staunch assertion of a primary psychogenetic etiology for all the psychosomatic disorders and also for epilepsy. Although Sperling recognizes that "some of my readers who are used to thinking of epilepsy as an organic disease . . . might take exception to my attributing meaning to the epileptic amnesia," she regards the epileptic attack as protecting the patient from acting out unacceptable criminal, perverted, aggressive, or sexual impulses.

The book consists of 25 chapters divided into 9 sections: an introductory section of 5 chapters devoted to theoretical and technical considerations and 8 clinical sections. These deal not only with classical psychosomatic conditions (ulcerative colitis, bronchial asthma, migraine, dermatoses) but also anorexia nervosa, tic disorders, and epilepsy. Otto Sperling, the late author's husband and a well-known psychoanalytic contributor, has edited the book and contributed an introduction, the opening chapter on "The Concept of Psychosomatic Disease," and two case studies on dermatitis and angioneurotic edema. Like his wife's clinical studies, these are interesting and instructive.

This book is well written and because of the fascinating clinical material it makes for pleasant and at times even stimulating reading. Seen in historical context, it is a valuable summary of the work of a psychoanalytic pioneer in the field of psychosomatic disease in children. Sperling recognized early the need to treat parents as well as the identified patient, the need to reduce and when possible to eliminate dependence on medication, the need to explore memories and fantasies from the earliest years in the lives of sick children, and the need to reduce the splitting and fragmentation that results from the "team approach" to these children. However, students of the mechanisms underlying the psychosomatic disorders will need to go beyond this book for a comprehensive and up-to-date understanding.

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FREDERICK H. LOWY, M.D.
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Medicine and the Reign of Technology, *by Stanley Joel Reiser. New York, N.Y., Cambridge University Press, 1978, 307 pp., $14.95.*

Medicine increasingly relies on technological progress to achieve better diagnostic refinement and, consequently, therapeutic results. Many of the technical tools, such as the thermometer, the sphygmomanometer, and X rays, have become such regular routines that it seems as if they have always been with us. This is not the case, however. The acceptance by physicians of these and other important medical tools was at times unpredictable and was often related to progress achieved in the hard sciences like physics and chemistry. Conversely, the use of these medical tools resulted in substantial changes in medical practices and ultimately in better public health. These are two main points of Reiser's book. For each of the new tools, long controversies, often marked by bitterness and bias, ensued. In the end, clinical judgment and anatomopathological observation led to their daily use. This applies to the stethoscope, the ophthalmoscope, the laryngoscope, photography, X rays, the microscope, the sphygmomanometer, the electrocardiograph, the thermometer, and the various tests of urine, blood, gastric juice, and other fluids of the body—all developed some time during the past century.

The level of sophistication necessary to master the techniques related to these new tools contributed to the trend toward specialization by the medical profession and the public's increasing acceptance of the hospital and of private group practice. In retrospect, the advantage provided by refined technology in the hands of competent specialists was somewhat overshadowed by the loss of the old-fashioned family physician, a father figure who attended to the emotional as well as the physical needs of his patients and their families.

As Reiser puts it, in the first two decades of our century psychoanalysis focused on the importance of the patient's history; "conscious psychotherapy" was developed as an alternative means of evaluating and relieving emotional problems. However, he acknowledges that before World War II American physicians generally did not spend too much time exploring their patients' social and emotional experiences, and psychoanalysis also raised opposition from patients.

In spite of the shortcomings related to different evaluations of tests by various technicians, advances in telecommunication and in automation led to the introduction of highly specialized methodology, notably the cytoanalyzer and the computer in medicine. Regardless of the issue of how far the computer will go in replacing the specialist, the author concludes that "the healing of illness requires more than healing parts of the body; it also requires intensive efforts to communicate with patients." Further, he says, "The problems of illness hatched from beliefs, illusions, values, and other facets of cultural and mental life . . . are as forceful and significant as the biological problems of illness approached through technology and scientific learning."

This interesting and easily read volume by a clinician and medical historian, founder and director of the History of Medicine Program at Harvard Medical School, is directed at a wide audience. As such, it is largely expository and does not delve into a deeper analysis of sociocultural aspects related to technological developments (for instance, the so-called biophysical movement of the 1840s in German universities, which led to the level of excellence of their medical schools, and the interplay of physiological and political concepts by many scientists, beginning with Virchow).

Although it is not directed at psychiatrists, I think this volume may also be relevant for psychiatry, at least on two counts: on the one hand, technique appears to develop to the point of reaching a counterproductive level; on the other hand, the historical development of modern medicine is characterized by an increasing ambivalence between machine and humanism. These two issues are crucial to today's psychiatry.

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Down's Syndrome and the Family: The Early Years, *by Ann Gath. London, England, Academic Press, 1978, 128 pp., $13.25.*

In this short book Ann Gath reports her detailed medical and psychiatric observations on 30 families of infants with Down's syndrome and 30 well-matched families of normal infants. I know of no comparable controlled study involving a prospective follow-up of consecutively born infants with Down's syndrome and their families.

A total of 40 infants with Down's syndrome were born during the time of the study; 4 died in early infancy, 5 did not fulfill the criterion of living at home, and the family of 1 withdrew from the study. The physical and mental health of parents and siblings was assessed by interviews and questionnaires over a two-year period from the time the infant was diagnosed as having Down's syndrome. Teachers' rating scales were also used to assess siblings' behavior.

The most prominent finding in this study was that of greater marital disharmony in the families of infants with Down's syndrome than in the control families. Among the parents of the infants with Down's syndrome there were 2 separations and 1 divorce; a total of 9 of these couples were given a poor marital relationship rating. This was in contrast to no divorces or separations and no overall poor marital relationship rating among the control parents. In spite of the greater incidence of marital discord among parents of infants with Down's syndrome, the majority of the parents adapted well and did not have more medical or psychiatric problems than parents of the control infants. From the parents' marital histories, it appeared that marital disharmony and divorce or separation occurred mainly in those who had the poorest relationship before the birth of the handicapped infant. This logical and perhaps commonly observed phenomenon has not been well documented in previous studies. Gath's study also demonstrated that the degree of marital disharmony was not associated with the severity of developmental problems in the handicapped infant, since parents of infants with congenital heart disease, severe hypotonia, or severe feeding problems were not more likely to have marital disharmony.

It also appeared that couples with the most problems were the ones who were most socially isolated. They tended not to want public health nurse or social work contacts, although they did accept well the researcher's visits into their homes. In all, 12 couples had no contact at all with other parents of infants with Down's syndrome or with any professional agencies. Most of these parents did not seek support from the Society for Mentally Handicapped Children, and only 2 parents approached their local social service agency for help.

There were several less striking but equally important observations among the infants with Down's syndrome and their families. For example, one of the subjects was found to

have an inherited translocation, which created a great deal of anxiety and resentment in the carrier family; several members refused examination. Although this was the reaction of only 1 family, it is evident that greater understanding of the process of disclosure of such genetic findings is needed.

Another interesting finding was that subsequent reproduction was not affected by the birth of the abnormal child, since 9 parents of infants with Down's syndrome and 9 parents of control infants went on to have another child.

As suggested by previous retrospective studies, parents wanted to know their infant's diagnosis as soon as possible. Any delay in detection or disclosure of diagnosis appeared to create long-lasting anger and hostility toward the health professionals involved. The author also reports that the parents found medical clinics staffed with rotating doctors in pediatric training programs the least helpful, and, as would be expected, that specialized clinics for the care of handicapped children, where there was consistent contact with the same pediatrician, were the most helpful. Finally, although several mothers of infants with Down's syndrome were informed of hospital respite services, these were used in only one instance, and it appeared that even a short respite hospitalization suggested to the mother that the baby was being "put away."

The main shortcoming of this study is that two years is not long enough to measure the full effect of the handicapped child on the family. Especially in Down's syndrome, the infancy and preschool years may be the easiest period for the family. The author reviews previous studies of her own and others illustrating problems in the parents and siblings of older individuals with Down's syndrome (e.g., sisters of individuals with Down's syndrome have more antisocial adjustment). It was also disappointing that the author's study is strictly descriptive and does not attempt to measure the effect of any form of early family intervention. There is also an editorial problem caused by repeated referral to material from the author's previously reported survey of the siblings of individuals with Down's syndrome, which is not included in the text as an appendix.

In her final recommendations the author reviews the theory that parents may be most susceptible to intervention during the first year after the birth of a handicapped child (1) and suggests some form of early crisis intervention for parents of infants with Down's syndrome. Since marital disharmony was greatest in parents with the most problems before the birth of the handicapped child, intervention could be directed toward these higher-risk families. My associates and I have proposed a model for such intervention (2), but experience has shown that parents are not likely to participate in weekly counseling sessions without some evidence of direct benefit to the affected child. The author's finding that parents who did not want public health nurse or social work contacts were still accepting of the researcher's regular home visits suggests that future attempts at early family intervention may come about through the professional's presence in the home. This may be of particular benefit for parents who are most likely to be burdened by the care of a handicapped child.

In summary, the observations and recommendations put forth in this book are most timely and welcome and should benefit health professionals involved in the care of handicapped families.

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A Social Interactional Theory of Emotions, *by Theodore D. Kemper. New York, N.Y., Wiley-Interscience (John Wiley & Sons), 1978, 434 pp., $19.95.*

In order to understand the interplay of emotions in social relationships, Kemper has ambitiously sought "a principle that can perform the same function as gravity in classical mechanics." He has chosen the "power-status" model of social interaction for this purpose. This book consists of an exposition of his theory based on an extensive review of the literature. Kemper has selected about 900 references on the physiology and psychology of emotion and the psychology and sociology of social relationships; no new data are presented. Topical chapters include a detailed structural formulation of emotions as these are affected by the power-status variables existing between two people, an analysis of the physiology of emotions in human relationships, a theory of schizophrenia and depression in power-status terms, and analyses of "punishment" and "love" from the standpoint of this theory.

Despite the diversity of academic fields and reports surveyed, the author presents his material in a lucid, flowing, and readable form. Simply as a review of the broad literature on emotions and the power-status model of social relationships, the work is valuable. At times, however, the author seems to be forcing all available observations into a single theory unable to contain them.

Psychiatrists will be disappointed with the author's treatment of depression and schizophrenia: his reinterpretation of clinical "facts" often fails to ring true and is largely a rehash (although using new terminology) of Freudian theory on the etiology of adult psychiatric disorders in child raising practices. The author's theory itself, with 1,701 "possible" emotions and 168 "likely" emotions, does not lend itself to ready clinical application, but it would definitely interest many psychological or physiological researchers studying emotion. Child and adolescent psychiatrists, as well as forensic and prison psychiatrists, should find the chapter on punishment and "distressful emotions" useful. Most psychiatrists, and especially those seeing couples, would gain from the two chapters on "love as a social relationship," with their cogent exposition of intimate relationships from the conceptual viewpoint of power and status.

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Beyond the Double Bind: Communication and Family Systems, Theories, and Techniques with Schizophrenics, *edited by Milton M. Berger, M.D. New York, N.Y., Brunner/Mazel, 1978, 254 pp., $16.95.*

Twenty years after the presentation of the now famous paper "Toward a Theory of Schizophrenia" by Bateson and associates (1), a follow-up conference was held titled Beyond the Double Bind. (The reader will, I trust, be aware

of the concept of the double bind theory as it applies to the understanding of schizophrenia—if not, this book presents the original paper for review.) The aim of the conference was to explore the heuristic effects of the double bind theory as seen by leading family therapists, including all of the living authors of the original paper. In addition, such notable communications theorists or family therapists as Bowen, Scheflen, Wynne, and Whitaker present their current thinking about psychotherapy and the double bind concept as it applies to the treatment of severely disturbed individuals and families. The conference papers have been carefully edited for presentation in book form.

This is not a book for beginning family therapists. The authors do not develop their theories of psychotherapy with schizophrenic patients in a sequential manner. They assume that the reader has some background in communications theory and family systems therapy. The presentation of the original double bind paper at the beginning of the book is not intended to provide adequate background for the novice therapist but, rather, to pay respect to the concepts being explored and to serve as general background for the presentations. The book has considerable value, however, to the more experienced therapist who wishes to reevaluate his or her thinking on the treatment of severely disturbed individuals and families.

This book provides a rare opportunity to, as it were, sit alongside leaders in the field of family therapy and learn from their experience over the past 20 years in the treatment of schizophrenia.

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Toward a Theory of Psychosomatic Disorders: Alexithymia, Pensée Opératoire, Psychosomatisches Phänomen, *edited by W. Bräutigam and M. von Rad. Basel, Switzerland, S. Karger, 1977, 388 pp., $53.50.*

In their incisive introduction describing papers given at the 11th European Conference on Psychosomatic Research, Bräutigam and von Rad outline the major themes considered in this volume. They point first to the two diverging attitudes toward psychosomatic research: one tends more to specify disease, the other to find a common denominator in different disorders. The latter approach, they say, was magnificently formulated by Jurgen Ruesch in 1948; he described the affective deficiency in communication, in fantasy, and in social behavior as belonging to an "infantile personality." It is against this background that the new terminology of alexithymia, pensée opératoire, and psychosomatic phenomena appear.

The editors also saw the meeting as embracing endeavors to differentiate psychosomatic patients from psychoneurotic patients. The definition of alexithymia by Sifneos and Nemiah puts a seal of significance on the observations of many others dating from the 1940s and 1950s. Their efforts to clarify and measure this concept put us in their debt. Concurrently the French workers Marty and de M'Uzan have developed a parallel concept called pensée opératoire. Both deal with the difficulty many patients, especially those with psychosomatic disorders, have in communicating feelings and expressing fantasy. Other European psychiatrists speak of psychosomatic phenomena in a similar way, but the three terms are by no means synonymous despite their appearance on the cover of this book. Furthermore, these terms often mean somewhat different things to different people. The more than 40 papers in this book show how widely the term "psychosomatic" varies from the classic "holy seven."

The book begins with 4 splendid papers on basic concepts. This is followed by a section titled Observations that begins with Sifneos and associates' studies of alexithymia in neurotic and psychosomatic patients. The other 17 papers add useful variations on the assessment of alexithymia in different conditions, including commissurotomy, ulcerative colitis, and pensée opératoire in obesity. Nemiah and associates add a new dimension with their study of oxygen consumption in response to affect-provoking thoughts. The causes of and mechanisms involved in alexithymia excited much discussion, but no consensus emerged.

The third section, titled Theories, begins with Nemiah on theoretical considerations followed by MacLean on the triune brain and the Heibergs on a study of twins. These are stunningly good contributions. The other 7 papers are useful and provide many significant elaborations, especially on the role of the family.

In the fourth section therapeutic approaches are elucidated. Bastiaans emphasizes the importance of the patient's feeling of safety denied by deficient coping strategies, particularly in the area of communication. Therapy in psychosomatic disorders should aim to restore a sense of safety, which may be encouraged by supportive and group therapy. There seems to be a consensus that formal psychoanalysis is contraindicated because dynamic insight therapy can be used only after the milder approaches, such as relaxation and group therapy, have decreased the patient's rigidity. The authors most referred to in this section are Winnicott for his sage explanations of early relationships and McDougall for her insightful explication of early developmental processes, of importance to an understanding of alexithymia and pensée opératoire in 1974. The authors might well have quoted one of McDougall's most pungent remarks: "Research into the meaning and treatment of psychosomatic illness is at the crossroads of various scientific disciplines." This is a succinct commentary applicable to the field and to this meeting.

The final 2 papers, in a section titled Varia, try to discuss the lives of Blaise Pascal and Pinocchio from a psychosomatic point of view. It is a pity that the conference did not finish with a more rigorous scientific approach because this is the main criticism of the whole book. Since there was considerable diversity of opinion as to what psychosomatic phenomena or disorders should be considered the proper area of interest, it is not surprising that considerable nebulousness reigns. Despite the fact that it was called a research conference, too many papers with no research orientation marred the program.

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Aging: The Process and the People, *edited by Gene Usdin, M.D., and Charles K. Hofling, M.D. New York, N.Y., Brunner/Mazel, 1978, 239 pp., $15.00.*

This volume from the American College of Psychiatrists clearly presents current knowledge and theories of the aging.

process and elderly people. It should be useful as a basic text for students of any discipline concerned with gerontology or with services for aging citizens. In the introduction Gene Usdin points out that interest and understanding of normal and pathological aging is intriguing and challenging, should benefit older patients, and, from a selfish view, should smooth our own later years.

The "Overview on Aging" by Robert Butler provides demographic information and considers the role of finances, emotional and mental problems, and "professional neglect." He stresses the need for research, education, service, and attitudinal changes.

In "Social and Cultural Context of Aging" George Madox uses "differential gerontology" to accentuate the wide variation in functional capacity, socioeconomic status, and coping styles of older people of the same chronological age. He stresses the need for creativity in meeting needs in such areas as finances, health care, and transportation. He also questions society's values and attitudes.

Theories and current research on the "Biology of Aging and Its Role in Depression" are outlined by Morris Lipton and Charles Memeroff and emphasized by their conceptual figure of "a multivalent model of depression." Their tables on age-related changes in catecholamines and related enzymes, on indoleamines, monoamine oxidase, and cholinergic systems, and on amino acid transmitters provide an excellent basis for information on current thinking and should give direction to further research.

In "Psychophysiologic and Cognitive Studies in the Aged" Carl Eisdorfer describes the difficulties in studying and differentiating normal aging and a pathological process. He reviews the psychophysiologic studies that have shown significant findings and have been replicated, but in spite of the progress made in the past decade the need for research into accurate measures of normalcy and disease is obvious.

Ewald Busse's "Review and Critique of Aging Research" points to further areas of study, such as EEG, sleep, sex differences, and sexuality. He defines research and its subtypes, making a plea for continued basic research.

In "Death and Dying" Thomas Holmes describes the attitudes and expectations of societies and the interaction of these stimuli with the psychophysiologic mechanisms that result in the response of death.

Bennett Gurian and Marjorie Cantor deplore the neglect of the elderly consumer in "Mental Health and Community Support Systems for the Elderly." As one who has focused on geriatric services in a community mental health center for many years, I would argue with some of their statements. For example, I do not agree with Robert Weiss that the helper must be perceived as an ally by the distressed individual to give support. Confrontation of the dysfunctional older person is the role of those in a support system, even if the patient views the supporter as an enemy. Although I agree that services should be provided in a setting in which "there is interdisciplinary training and a wide range of services, such as a teaching hospital," an interested psychiatrist can be a one-man team in any setting.

"Aged Patients, Their Families, and Physicians" by Charles Gaitz sums up many of the areas in working with an older patient for consideration. Although there is overlap of material in many chapters, the attitudinal consensus and the individual viewpoint of each writer make this volume a valuable addition to the literature.

SHIRLEY M. COLTHART, M.D.
Rochester, N.Y.

Progress in Experimental Personality Research, Vol. 8, *edited by Brendan A. Maher. New York, N.Y., Academic Press, 1978, 340 pp., $23.00.*

Progress is a difficult thing to recognize in personality research. Findings accumulate and conceptualizations become more sophisticated, but the degree to which our knowledge is higher, better, or more advanced is uncertain. The six articles in this volume cover a variety of topics (empathy, violence, drug abuse, schizophrenia deficits, female criminality, socialization), but progress is not a recurring theme. Nonetheless, all the articles provide thorough and interesting overviews of complex subjects.

Schizophrenia researchers will find the Oltmanns' and Neale's discussion of deficit research very worthwhile and challenging. After reviewing the theories of Vygotsky, Goldstein, and Arieti and then critiquing data from object sorting, proverb, and other reasoning tests, the authors present a brief but compelling section on fundamental aspects of methodology in schizophrenia research. We are reminded, for example, that even schizophrenia research analyzed with t tests or analyses of variance is basically correlational and not experimental because the "critical variable—schizophrenic control—has not been manipulated nor have subjects been randomly assigned to either of these conditions." Matching schizophrenic and control groups, for instance, is a common aspect of the experimental paradigm, but the matching procedure itself can introduce severe artifacts because of "third" variable influences. Everything about schizophrenia continues to lure researchers from many fields; this article is worthy of their careful attention.

A lively review by Pihl and Spiers of research on the addictive personality concept will be discouraging to would-be researchers in this area. After a careful and illuminating consideration of previous work, the authors essentially are left with the generalization that the addicted individual is troubled. This area is ripe for creative research strategies. Perhaps as genetic and biological predispositions to drug addiction are researched more carefully the role of personality and social factors will become more clear.

Geen's paper on the effects of observing violence and Fesbach's paper on empathy in children are the best examples in the volume of the experimental approach to personality research. The Geen article summarizes findings in the field and provides an overview of how researchers study a phenomenon in the laboratory and in the real world. Fesbach's paper concentrates on reporting the results of a specific project and is therefore a bit more narrow in appeal, but it too illustrates the experimental approach. Violence and empathy are favorite topics for personality researchers, but progress in these areas is particularly difficult to judge because of the problems associated with generalizing laboratory findings to the real world.

Female criminality is the subject of Widom's paper; her review highlights many important problems. Typing offenders on the basis of personality characteristics rather than for the crimes committed appears to be one promising area for more research, as does exploring further the relationship between crime and psychopathology. Social deviance always has been of special interest to personality researchers, and the fundamental question of sex differences in this area deserves much more attention.

The last paper in the volume is an informative piece on socialization in the Soviet child by Barlett. Since it is not empirical it seems a bit out of place with the other papers and with the volume's title, but it provides a noteworthy example

of the interplay of cultural assumptions, social policy, and social science. If experimental personality research is ever to have impact on social policy, a controversial prospect, then more topics like this need exploration.

There *is* progress in personality research, but it is slow and undramatic. Its promise in most areas has yet to be realized, but the field is vital and the questions remain provocative.

RICHARD J. HAIER, PH.D.
Bethesda, Md.

Progess in Clinical Neurophysiology, Vol. 3: Language and Hemispheric Specialization in Man: Cerebral Event-Related Potentials, *edited by John E. Desmedt. Basel, Switzerland, S. Karger, 1977, 282 pp., $47.25.*

This volume contains 18 chapters presenting studies on the topics mentioned in the title. Two deal with theoretical issues, 4 with perception of language, 3 with speech production, and the remainder with hemispheric asymmetries in linguistic and nonlinguistic tasks. Too much material is presented to review in detail; I will therefore concentrate on highlights that seem to me potentially most fruitful.

The introductory chapters on the psychobiology of speech and language make ample reference not only to the neurological literature establishing a dominant role for the left hemisphere in linguistic function but also to the burgeoning recent linguistic and psycholinguistic writings that have enunciated a theoretical framework for the detailed description and explanation of linguistic structures and, to some extent, aspects of their use in talking and in comprehending speech. Unfortunately, aside from a passing comment by Donchin and associates, these considerations are not evident in the studies reported. No studies bear on event-related potentials related to any of the important linguistic structures now articulated within this framework, nor to any of the well-defined hypotheses available from psycholinguistics regarding the processing of sentences. In part this is due to perceived limitations of the event-related potential technique, which requires repeated stimuli and experimental paradigms not easily constructable with these independent variables. I suspect it is also partly due to experimental clinical neurophysiologists' relative lack of familiarity with this literature and the concern they have with other psychological factors, such as attention, which figures notably in the volume. Donchin and associates' comments that interesting event-related potential effects, particularly in the late components of potentials, may reflect such variables seem to me most cogent and a possible direction for further work.

What is reported with respect to language is a good deal of material on event-related potentials and spectra-analyses related to perception of lexical and categorical ambiguity of individual words, perception of Haskins-generated synthetic syllables (with similar but nonlanguage material used as controls), and perception of such semantic aspects of words as their place on the Osgood semantic differential. In speech production, the book presents important cautions dealing with respiratory and oro-facial-pharyngeal-lingual motor contamination of cerebral potentials. The effects on the event-related potentials are not robust in the perception work, and much of the production work seems like a retreat from earlier claims regarding lateralization of speech-specific potentials. Many analyses of the perceptual data are post hoc, and, as Molfese reports, different analyses can lead to radically different results. This is not to say that the reports are uninteresting; indeed, I think some of them are remarkable, such as Chapman and associates' discovery of an event-related potential effect of the semantic differential. As a whole they provide evidence that abstract information coded in the linguistic signal is measurable neurophysiologically over selected sites, primarily the left hemisphere. It is to be hoped that further investigation, perhaps along the lines I suggested at the outset, will provide more data regarding these effects.

The studies on hemispheric specialization use a variety of techniques: spectra-analysis (both for frequencies and amplitudes), event-related potential data derived from tasks thought to engage one hemisphere preferentially, and event-related potential effects of interfering or competing with a task engaging primarily one hemisphere. Molfese's paper traces the ontogeny of some of the event-related potentials to a select but well-defined stimulus set. Almost without exception, the investigators found what they expected: visuospatial tasks engage the right hemisphere and language tasks the left. Of some interest (and concern) to me is the fact that interpretation of the data as a reflection of hemispheric involvement is to some extent arbitrary. Thus, most investigators in the third section of the volume accept the view that desynchronization of the EEG and decrease in its amplitude indicates increased processing in an area. How does this square with claims (such as in Roemer and Teyler) that larger amplitude variation in event-related potentials reflects local processing?

The authors are sensitive to these issues. The papers by Desmedt and Robertson and by Donchin and associates begin to develop a taxonomy of potentials with respect to psychological variables. The key division of event-related potentials in their view is along temporal lines. Early potentials are considered stimulus-specific, perhaps reflecting lemniscal and other fast-pathway transmission, and are lateralized to the opposite hemisphere. Potentials in the 50–200-msec range reflect attentional processes and stimulus characteristics such as frequency and modality. Late potentials, like the P300 and possibly the CNV, reflect abstract stimulus variables, are modality-independent, and are the consequence of "complex" and "endogenous" psychological manipulation of stimuli. Clearly, such taxonomies are only preliminary and are to be extended to encompass more of the variation of potentials and EEG spectra than can now be done, with the goal of developing a theory of the relevant neurophysiology.

Many authors remark that modern analysis of EEG spectra and event-related potentials constitutes our first foothold in the area of the neurophysiology of cognitive processes in intact man. The field is in its infancy; much empirical work and theoretical formulation will precede the kind of analysis that we have of information encoding and flow for peripheral nerve and many nonhuman perceptual and motor systems. This volume gathers together valuable papers that are representative of results and thinking in the field.

DAVID CAPLAN, PH.D., M.D.
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Aging and Behavior: A Comprehensive Integration of Research Findings, 2nd ed., *by Jack Botwinick. New York, N.Y., Springer Publishing Co., 1978, 398 pp., $16.50.*

The title of this book, which is the second edition of a 1973 publication, is somewhat misleading in that the word "ag-

ing" suggests a continuous process but the book deals almost exclusively with the aged. The concept of process (the vital process of aging) is referred to minimally. Furthermore, and as a corollary, connections between early life and its impact on aging and the aged are denied. The author's allusion to a longitudinal process is a negative one, and he appears to be biased in his intentional exclusion of psychodynamic concepts. He attempts to discredit what is referred to as "the orthodox psychoanalytic view" in his chapter on simple rigidity:

> Rigidity has been thought to be a global aspect of man. Many psychologists, until relatively recently at least, behaved as if they also had this view of rigidity. *This was typified most extremely, perhaps, by the orthodox psychoanalytic view.* If there is fixation at a particular stage of psychosexual development, that is, if childhood development is not complete, having stopped at a particular point in the process, the resulting adult will be stunted in effectiveness by an obsessive thought system and by an excessive rigidity of behavior. (italics mine) (p. 87)

A few sentences later he writes, "Even if we were to endorse the premise that young adults can be classified on a continuum of general rigidity, and *even if we were to accept the notion that rigid personality is the result of earlier life experiences . . .*" (italics mine). It is this particular point of view that I find disturbing because it ignores the research and clinical studies of Spitz, Mahler, Freud, Erikson, Harlow, and Fraiberg, to mention a few. It should further be noted that in a bibliography of 674 references, there is no mention of the *Journal of Geriatric Psychiatry*, a publication that for more than 12 years has advocated a longitudinal and dynamic view of the process of aging. Indeed, the author's approach to behavior appears to be more physiological, as indicated, for example, by his chapter eleven. Titled "Processing Sense Information," it contains the following subheadings, Sequential Integration: Stimulus Fusion, Stimulus Masking, and Stimulus Enhancement, Stimuli Reflecting Common Experience, Sustained Attention; Temporal Integration of Non Sequential Information: Spiral Aftereffect, Color Afterimage, Time to Perceive Complex Stimuli, Illusions; Spatial Integration: Part-Whole Relationships, Modification of Percepts; Anoxia, Another Model; and Compensatory Adjustments.

One might have hoped that Dr. Botwinick could have presented a more rounded view of aging and behavior. Although he may not hold to the concepts of dynamic theory, a complete discussion of aging and behavior should indicate that such concepts exist and are more often than not the basis for considerable exploration, research, and writing.

MARTIN A. BEREZIN, M.D.
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Violence and Responsibility: The Individual, the Family and Society, *edited by Robert L. Sadoff, M.D. New York, N.Y., SP Medical & Scientific Books (Spectrum Publications) (Halsted Press, John Wiley & Sons, distributor), 1978, 133 pp., $14.95.*

The increasing public concern about violence and its social ramifications served as the impetus for a symposium on the subject at Philadelphia's Friends Hospital in 1976. This volume is a collection of nine essays delivered at the symposium by various distinguished authors. They discuss violence in intrapsychic, neurological, legal, and social contexts.

In general, the individual authors are to be commended for their clarity of thinking and readable style. Nevertheless, the essays represent only brief outlines of the frustrations and dilemmas in so broad a topic, serving to raise consciousness through an emotional appeal to the audience.

Unfortunately, there is little new information or research contained here. Some recommendations are advanced as to possible approaches, but few actual data are provided as to the efficacy of these solutions. In summary, the book offers an introduction to the subject of violence, directed toward the neophyte. As such, it presents a provocative first step to understanding but falls short of making any major contribution.

THOMAS C. BOND, M.D.
Boston, Mass.

Progress in Behavior Modification, Vol. 6, *edited by Michel Hersen, Richard M. Eisler, and Peter M. Miller. New York, N.Y., Academic Press (Harcourt Brace Jovanovich), 1978, 248 pp., $18.50.*

The purpose of this series on behavior modification is to present timely issues, new directions, and future trends in the field. Each of the six volumes' reviews begins with a short outline that aids the reader in maintaining organization. Despite the large number of reviews in the six volumes, there is very little overlap in subject matter. The previous volumes reviewed in the *American Journal of Psychiatry* have received excellent ratings.

There are seven original review articles in volume 6. These articles are well referenced and cover their topics in depth. "Behavioral Treatments for Insomnia," "Behavioral Approaches to Stuttering," and "Assessment and Treatment of Enuresis and Encopresis in Children" provide some practical treatment approaches to difficult clinical problems. "Behavior Modification in Latin America," "The Social Identity of Behavior Modification," "New Directions in Behavior Modification with Children," and "The Therapist-Client Relationship in Behavior Therapy" offer little therapeutic technique but provide excellent historical and theoretical discourses.

I feel that the first three articles in volume 6 have some value for clinicians. Overall, however, this is more of a reference book for the psychiatrist particularly interested in behavior modification.

GEORGE W. MIDDLEKAUFF, M.D., PH.D.
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Adults and Their Parents in Family Therapy: A New Direction in Treatment, *by Lee Headley. New York, N.Y., Plenum Press, 1977, 190 pp., $14.95.*

The purpose of this slender book is to describe the value of including in psychotherapy the parents of adult patients who are being seen for either individual or marital problems. The author delineates her views of types of patients for whom this inclusion is valuable, the necessary conditions for proceeding, the types of problems encountered, and the

benefits of the experience for both the adult patients and their parents. The book is easy to read and enlivened by many case examples that convey the way the author does her work. However, this book is not a significant addition to the literature on family or individual therapy.

The nature and importance of intergenerational ties have been well delineated by Ivan Boszormenyi-Nagy and Geraldine Spark in Invisible Loyalties (1), which is unfortunately too little read. Although Nagy and Spark described their work with adults and their parents, they did not focus on the characteristics of the psychotherapy with this combination, which was described in thoughtful detail by James Framo (2). In a closely related area, Murray Bowen (3) has demonstrated that patients in individual therapy can be helped to return home to visit their parents and to change the characteristic and unrewarding patterns of interaction between them and their parents in the direction of greater individuation and mutual satisfaction. This work and its importance are not discussed in the body of Headley's book, although the Framo and Bowen papers are mentioned in the introduction.

I believe the author's tendency to be clearly on the side of her patients can have unfortunate effects. Although she states that therapists should be even-handed, it is evident from her case material that in the therapist the patient has an ally—"a friendly facilitator, who will assist him/her in case there are any unsuccessful binds with the parent." When tensions rise, she is quick to end the interview, which is probably a good thing, but often painful feelings must be aired before they can be resolved. Too often one has the sense that Headley wants her adult patients and their parents to reach an agreement about how to handle some problem in a practical way before the issues dividing them have been adequately raised and their interactional nature clarified.

This book may be of some interest to adult patients who are contemplating having their parents participate in their therapy. For mental health professionals, it does not add to the literature in this area and is an insubstantial volume in both size and thought.

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2. Framo J: Family of origin as a therapeutic resource for adults in marital and family therapy: you can and should go home again. *Fam Proc* 15:193–209, 1976
3. Bowen M: Towards the differentiation of a self in one's own family, in Family Interaction. Edited by Framo J. New York, Springer Publishing Co, 1972

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Books Received

This listing acknowledges the receipt of recent books. Books of particular interest to the readers of this journal will be reviewed as space permits, and copies of the reviews will be sent to the publishers. Books cannot be returned to the publishers.

The Chronic Mental Patient: Problems, Solutions, and Recommendations for a Public Policy, *prepared by the American Psychiatric Association Ad Hoc Committee on the Chronic Mental Patient; edited by John A. Talbott, M.D. Washington, D.C., APA, 1978, 259 pp., $11.00.*

Task Force Report 14: Electroconvulsive Therapy, *by the American Psychiatric Association Task Force on Electroconvulsive Therapy. Washington, D.C., APA, 1978, 200 pp., $7.50 (paper).*

One to One: Self-Understanding Through Journal Writing, *by Christina Baldwin. New York, N.Y., M. Evans and Co., 1977, 186 pp., $3.95 (paper).*

Paul Schilder: On Neurosis, *edited by Lauretta Bender, M.D. New York, N.Y., International Universities Press, 1979, 378 pp., $22.50.*

The Adolescent Passage: Developmental Issues, *by Peter Blos. New York, N.Y., International Universities Press, 1979, 507 pp., no price listed.*

A Bibliography of Prostitution, *edited by Vern Bullough, Barrett Elcano, Margaret Deacon, and Bonnie Bullough. New York, N.Y., Garland Publishing, 386 pp., $38.00.*

Disulfiram in the Treatment of Alcoholism, Bibliographic Series 14, *compiled by S. Busse, C.T. Mulloy, and C.E. Weise. Toronto, Ont., Canada, Addiction Research Foundation, 1978, 301 pp., $8.00 (paper).*

The Annual of Psychoanalysis, Vol. VI/1978, *edited by the Chicago Institute for Psychoanalysis. New York, N.Y., International Universities Press, 1978, 481 pp., $20.00*

The Stranger in Your Bed: A Guide to Emotional Intimacy, *by Dean C. Dauw with Tom and Joan Watts. Chicago, Ill., Nelson-Hall, 1979, 109 pp., $9.95.*

Getting Motivated by Ernest Dichter: The Secret Behind Individual Motivations by the Man Who Was Not Afraid to Ask "Why?" *by Ernest Dichter, Ph.D. New York, N.Y., Pergamon Press, 1979, 193 pp., $10.95.*

Diagnosis and Therapy of Porphyrias and Lead Intoxication: International Symposium Clinical Biochemistry, *edited by Manfred Doss. New York, N.Y., Springer-Verlag, 1978, 305 pp., $34.00 (paper).*

Law, Psychology, and the Courts: Rethinking Treatment of the Young and Disturbed, *by Ellsworth A. Fersch, Jr., Ph.D., J.D. Springfield, Ill., Charles C Thomas, 1979, 164 pp., $14.75.*

Helping the Troubled Employee, *by Joseph F. Follmann, Jr. New York, N.Y., AMACOM (American Management Association), 1978, 246 pp., $15.00.*

Dream Interpretation: A Comparative Study, *edited by James L. Fosshage, Ph.D., and Clemens A. Loew, Ph.D. New York, N.Y., SP Medical & Scientific Books (Spectrum Publications), 1978, 298 pp., $20.00.*

Discipline and Punish: The Birth of the Prison, *by Michel Foucault; translated by Alan Sheridan. New York, N.Y., Vintage Books (Random House), 1979, 333 pp., $4.95 (paper).*

Current Themes in Psychiatry, *edited by Raghu N. Gaind and Barbara L. Hudson. London, England, Macmillan Press, 1978, 379 pp., £12.00 (British pounds).*

Maximizing Treatment Gains: Transfer Enhancement in Psychotherapy, *edited by Arnold P. Goldstein and Frederick H. Kanfer. New York, N.Y., Academic Press, 1979, 484 pp., $25.00.*

Overcoming Impotence: What Every Husband and Wife Should Know, *by Leo I. Jacobs, M.D. Chicago, Ill., Contemporary Books, 1978, 74 pp., $6.95.*

L'Identité du Psychanalyste, *edited by Edward D. Joseph and Daniel Widlocher. Paris, France, Presses Universitaires de France, 1979, 294 pp., no price listed.*

Conditioning: An Image Approach, *by Donald L. King. New York, N.Y., Gardner Press (Halsted Press, John Wiley & Sons, distributor), 1979, 483 pp., $27.50.*

The Autonomic Nervous System: Physiological Basis of Psychosomatic Therapy, *by Fuad Lechin, M.D., Bertha van der Dijs, M.D., and Ernesto Lechin, M.D. Barcelona, Spain, Editorial Científico-Médica, 1979, 159 pp., $23.00.*

Freud's Early Psychology of the Neuroses: A Historical Perspective, *by Kenneth Levin. Pittsburgh, Pa., University of Pittsburgh Press, 1978, 302 pp., $12.95.*

The World Technique, *by Margaret Lowenfeld. Winchester, Mass., Allen & Unwin, 1979, 281 pp., $27.50.*

Learning from Clients: Interpersonal Helping as Viewed by Clients and Social Workers, *by Anthony N. Maluccio. New York, N.Y. , Free Press (Macmillan Publishing Co.), 1979, 315 pp., $13.95.*

1978 Annual Review of Child Abuse and Neglect Research, *prepared by Mary Porter Martin and Susan L. Klaus. Washington, D.C., U.S. Department of Health, Education, and Welfare, 1978, 93 pp., no price listed (paper).*

Therapeutic Hypnosis, *by Michael Miller, M.D. New York, N.Y., Human Sciences Press, 1979, 357 pp., $18.95.*

Science of PSI: ESP and PK, *by Carroll B. Nash, Ph.D. Springfield, Ill., Charles C Thomas, 1978, 280 pp., $16.95.*

The Social Skills Basis of Psychopathology: Alternatives to Abnormal Psychology and Psychiatry, *by E. Lakin Phillips, Ph.D. New York, N.Y., Grune & Stratton, 1978, 267 pp., $16.50.*

Family Secrets: The Experience of Emotional Crisis, *by Mel Roman and Sara Blackburn. New York, N.Y., Times Books, 1979, 152 pp., $10.95.*

Weathering: How the Atmosphere Conditions Your Body, Your Mind, Your Moods—and Your Health, *by Stephen Rosen. New York, N.Y., M. Evans and Co., 1979, 360 pp., $12.95.*

New Views on Older Lives: A Sampler of NIMH-Sponsored Research and Service Programs. National Institute of Mental Health Science Monograph, *by Anne H. Rosenfeld. Rockville, Md., U.S. Department of Health, Education, and Welfare, 1978, 181 pp., $3.00 (paper).*

Damnation and Deviance: The Protestant Ethic and the Spirit of Failure, *by Mordechai Rotenberg. New York, N.Y., Free Press (Macmillan Publishing Co.), 1978, 224 pp., $12.95.*

Sexual Excitement/Sexual Peace: The Place of Masturbation in Adult Relationships, *by Suzanne Sarnoff and Irving Sarnoff, Ph.D. New York, N.Y., M. Evans and Co., 1979, 314 pp., $12.50.*

The Childhood Emotional Pattern in Marriage, *by Leon J. Saul, M.D. New York, N.Y., Van Nostrand Reinhold Co. (Litton Educational Publishing), 1979, 447 pp., $15.95.*

Progress in Clinical and Biological Research, Vol. 27: Membrane Mechanisms of Drugs of Abuse, *edited by Charles W. Sharp and Leo G. Wood. New York, N.Y., Alan R. Liss, 1979, 263 pp., $22.00.*

Reversals: A Personal Account of Victory Over Dyslexia, *by Eileen Simpson. Boston, Mass., Houghton Mifflin Co., 1979, 246 pp., $8.95.*

Dying, Death, and Grief: A Critically Annotated Bibliography and Source Book of Thanatology and Terminal Care, *by Michael A. Simpson. New York, N.Y., Plenum Press, 1979, 288 pp., $21.95.*

Delusions of Infestation: Dermatozoehwahn—Ekbom's Syndrome. Reports from the Psychiatric Research Centre, St. Jörgen Hospital, University of Göteberg, *by Annika Skott, M.D. Gotab, Sweden, Kungälv, 1978, 132 pp., $8.00 (paper).*

The Psychopharmacology of Hallucinogens, *edited by Richard C. Stillman and Robert E. Willette. New York, N.Y., Pergamon Press, 1978, 329 pp., $32.50.*

Faces in a Cloud: Subjectivity in Personality Theory, *by Robert D. Stolorow, Ph.D., and George E. Atwood, Ph.D. New York, N.Y., Jason Aronson, 1979, 208 pp., $15.00.*

Progress in Behavior Therapy with Delinquents, *edited by Jerome S. Stumphauzer, Ph.D. Springfield, Ill., Charles C Thomas, 1979, 381 pp., $29.75.*

A Resident's Guide to Psychiatric Education, *edited by Michael G.G. Thompson, M.D. New York, N.Y., Plenum Medical Book Co., 1979, 273 pp., $14.95.*

Introduction à la Psychiatrie Biologique, *by R. Tissot with Y. Burnand and A.-M. Zutter. Paris, France, Masson, 1979, 163 pp., 68 French francs.*

Psychoanalytic Politics: Freud's French Revolution, *by Sherry Turkle. New York, N.Y., Basic Books, 1978, 267 pp., $12.50.*

People in Pain: A Guide to Pastoral Care, *by James A. Vanderpool, Ph.D. Springfield, Ill., Charles C Thomas, 1979, 185 pp., $12.75.*

Stepfamilies: A Guide to Working with Stepparents and Stepchildren, *by Emily B. Visher, Ph.D., and John S. Visher, M.D. New York, N.Y., Brunner/Mazel, 1979, 272 pp., $15.00.*

Law and Psychiatry II: Proceedings of the Second International Symposium Held at the Clarke Institute of Psychiatry, *edited by David N. Weisstub. New York, N.Y., Pergamon Press, 1979, 163 pp., $15.00.*

Nonverbal Communication: Readings with Commentary, 2nd ed., *edited by Shirley Weitz. New York, N.Y., Oxford University Press, 1979, 418 pp., $13.95.*

The Inner World of Childhood, *by Frances G. Wickes. Englewood Cliffs, N.J., Prentice-Hall, 1966, 277 pp., $10.95; $5.95 (paper).*

Sleep Disorders: Diagnosis and Treatment, *edited by Robert L. Williams, M.D., and Ismet Karacan, M.D., D.Sc. Somerset, N.J., John Wiley & Sons, 1978, 409 pp., $32.00.*

Clinical Diagnosis of Mental Disorders: A Handbook, *edited by Benjamin B. Wolman. New York, N.Y., Plenum Press, 1978, 909 pp., $50.00.*

Mental Disorders: Glossary and Guide to Their Classification in Accordance with the Ninth Revision of the International Classification of Diseases, *by the World Health Organization. Geneva, Switzerland, WHO, 1978, 82 pp., 12 Swiss francs (paper).*

Practical Approaches to Alcoholism Psychotherapy, *edited by Sheldon Zimberg, John Wallace, and Sheila B. Blume. New York, N.Y., Plenum Press, 1978, 277 pp., $20.00.*

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OFFICIAL ACTIONS

The American Board of Psychiatry and Neurology

The following successfully completed the Board examination given in Boston, MA, November 6-7, 1978.

PSYCHIATRY

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Berkelhammer, Edward M., M.D., Elizabeth, NJ
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Berv, Kenneth R., M.D., New Canaan, CT
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Katz, Myles M., M.D., Forest Hills, NY
Kessler, John A., M.D., New York, NY

Laureno, Robert, M.D., Rockville, MD
Lechtenberg, Richard, M.D., New York, NY
Lichtenfeld, Peter J., M.D., Great Neck, NY
Lieberman, Bryan, M.D., Attleboro, MA
Lipsius, Bruce D., M.D., Bradford, MA
Luban, Norman A., M.D., Chevy Chase, MD
Lusons, John, M.D., Bronxville, NY

Martinez, Robert D., M.D., Lafayette, LA
Matthew, Elizabeth, M.D., New York, NY
Mayeux, Richard, M.D., New York, NY
Mitsumoto, Hiroshi, M.D., Cleveland, OH
Moore, George R., M.D., Kingston, Ont., Canada
Mordes, Marvin, M.D., Pikesville, MD
Mounayer, Sami, M.D., Royal Oak, MI

Newman, Steven E., M.D., Bethesda, MD

O'Bannon, John M., III, M.D., Richmond, VA
O'Keefe, Dennis M., M.D., Albany, NY

Plaitakis, Andreas J., M.D., Flushing, NY
Polinsky, Ronald J., M.D., Columbia, MD
Ribner, Jeffrey A., M.D., Endwell, NY
Richert, John R., M.D., Chevy Chase, MD
Riley, Terrence L., M.D., Silver Spring, MD
Ronthal, Michael, M.D., Brockton, MA
Ross, Elliott D., M.D., Dallas, TX
Rubin, Jeffrey I., M.D., Montreal, Que., Canada

Sachdev, Kuldip K., M.D., New York, NY

Sahenk, Zarife, M.D., Columbus, OH
Samuels, Martin A., M.D., Boston, MA
Sanders, Mindy E., M.D., Schenectady, NY
Schafer, John A., M.D., Carmichael, CA
Seybold, William R., M.D., Colorado Springs, CO
Singh, Avtar, M.D., Scarsdale, NY
Sulibhavi, Dhruvi, M.D., Farmingdale, NY
Sullivan, Richard L., M.D., Portland, ME

Talman, William T., M.D., Summit, NJ
Taormina, Vincent, M.D., Lexington, KY
Troiano, Raymond G., M.D., Virginia Beach, VA

Victor, Stephen J., M.D., S. Orange, NJ

Weiner, Howard L., M.D., Brookline, MA
Weisman, Richard M., M.D., Baltimore, MD
Weiss, Howard D., M.D., Baltimore, MD
White, Barry E., M.D., Lakewood, OH
Williams, Armistead D., M.D., Norfolk, VA
Williamson, Edward L., M.D., Greensburg, PA
Willner, Joseph H., M.D., New York, NY
Wright, David G., M.D., Pittsburgh, PA

CHILD NEUROLOGY

Kurczynski, Thaddeus W., M.D., Richmond Heights, OH
Leahy, William R., Jr., M.D., Chevy Chase, MD
Lusser, Martha A., M.D., Allentown, PA
Marshall, Paul C., M.D., Worcester, MA
Pe Benito, Rhandy D., M.D., New York, NY
Rodriguez, Dolores, M.D., Danville, PA
Rosenblatt, Bernard, M.D., Hampstead, Que., Canada
Wessel, Henry B., M.D., Gibsonia, PA

The following successfully completed the Board examination given in San Francisco, CA, January 8-9, 1979.

PSYCHIATRY

Ahluwalia, Yoglackshan K., M.D., Chicago, IL
Akina, Eleanore Mayo Green, M.D., Kailua, HI
Alex, Neil Steven, M.D., San Diego, CA
Amarasinghe, Amarasinghe A. W., M.D., Augusta, GA
Armstrong, David Ligon, M.D., Orange, CA
Aroon, Javali B., M.D., Lexington, KY
Atkinson, Joseph H., Jr., M.D., San Diego, CA

Barnes, Patricia J., M.D., Renton, WA
Baumzweiger, William, M.D., Studio City, CA
Beaumont, Graham, M.D., San Francisco, CA
Benson, David S., M.D., Highland Park, IL
Bienenstock, Bruce, M.D., Mountain View, CA
Bitar, Adib Husni, M.D., Memphis, TN
Bolmann, Andrew R., M.D., Pottstown, PA
Boyer, David N., M.D., Hunt Beach, CA
Braga, Djalma A., M.D., Jacksonville, FL
Brast, Neil B., M.D., Palo Alto, CA
Breslow, Richard E., M.D., New York, NY
Brumfield, Daniel T., M.D., Del Mar, CA
Bryant, Etta Colish, M.D., San Mateo, CA

Caliboso-Viloria, Rosalinda, M.D., Bronxville, NY
Castner, Edward J., M.D., Arcadia, CA
Choi, Won Il, M.D., Temple City, CA
Chu, Hung-Ming, M.D., Flint, MI
Chun, Ryo Sook, M.D., New Canaan, CT
Conti, Paul A., M.D., El Granada, CA
Cook, Colin B., M.D., Stamford, CT
Coons, David J., M.D., Anchorage, AK
Cooperman, Oliver B., M.D., San Francisco, CA
Cotrufo, John H., D.O., Oakland, CA
Cruz-Barrios, Olga T., M.D., Williamsville, NY
Cruz-Peguero, Francisco G., M.D., Topeka, KS

D'Azzara, Lewis, M.D., New York, NY
De Lima, Clarence A., M.D., Dayton, OH
Denea, Russell, M.D., Santa Monica, CA
Diamond, Robert Paul, M.D., Los Angeles, CA
Dillon, David Charles, M.D., Woodland Hills, CA
Dorus, Walter, M.D., Chicago, IL
Douglas, Florence Maud, M.D., Palos Verdes, CA
Drlik, John M., M.D., Fremont, CA
El-Chammas, Sabah N., M.D., Del Mar, CA
Ericksen, Stephen E., M.D., Sacramento, CA
Ettari, Charles Vincent, M.D., La Jolla, CA
Evans, Philip J., M.D., Berkeley, CA
Ezra, Joseph, M.D., Tarzana, CA

Finkelstein, Adrian, M.D., Northbrook, IL
Fluss, Hella R., M.D., Berkeley, CA
Ford, John Suffern, M.D., Webster, TX
Fowler, James Thomas, III, M.D., La Jolla, CA
Fox, Colin L., M.D., Millbrae, CA
Frank, John Lewis, M.D., Philadelphia, PA
Friend, R. Claire, M.D., Rancho Palos Verdes, CA
Friend, Robert London, M.D., San Francisco, CA
Fukushima, Susan, M.D., Los Angeles, CA

Gamble, Michael Miller, M.D., Berkeley, CA
Gardner, Daniel, M.D., La Jolla, CA
Garrie, Stuart A., M.D., Seattle, WA
Geller, Stephanie Lansman, M.D., Beverly Hills, CA
Gilbert, Paul, Jr., M.D., San Francisco, CA
Gillick, Marvin, M.D., Reseda, CA
Ginsberg, Harold N., M.D., Travis AFB, CA
Gode, Richard O., M.D., Seattle, WA
Green, Ben Ezra, M.D., Portland, OR
Greenberg, Peter M., M.D., San Francisco, CA
Grinstein, Alberto, M.D., Portage, MI
Groban, Stephen Edward, M.D., La Mesa, CA
Gross, Jonothon, M.D., Berkeley, CA
Groves, James Elton, M.D., Boston, MA
Grubb, David George, M.D., Oakland, CA

Hagen, Ralph L., M.D., Portland, OR
Hahn, Susan, M.D., Washington Crossing, PA
Hakanson, Glenn G., M.D., Sacramento, CA
Hamm, John Edward, M.D., Bainbridge Island, WA
Hammond, Kenric W., M.D., Redlands, CA
Hayes, Stephen G., M.D., Los Angeles, CA
Heidel, Stephen Hunt, M.D., San Diego, CA
Hemachandra, M. G. Sarath, M.D., Bloomfield Hills, MI
Hilliard, Ida McCree, M.D., Oakland, CA
Hirz, Ronald H., M.D., Laguna Beach, CA
Hoke, Bob, M.D., Long Beach, CA
Hondl, Edeltraud A., M.D., Seattle, WA
Hong, Kang-E Michael, M.D., Roseville, MN

Horvath, Thomas B., M.D., Stanford, CA
Hunt, David Daniel, M.D., Seattle, WA
Hunt, Robert D., M.D., New Haven, CT
Hyde, Geoffrey Noel L., M.D., Portland, OR

Jacobson, Eric Michael, M.D., Carmel, CA
James, Robert L., M.D., Santa Monica, CA
Johnson, Matthew Edward, M.D., Rock Hill, SC
Johnson, Richard Haviland, M.D., Pendleton, OR

Kadkhodaian, Hooshmand, M.D., Arlington Heights, IL
Kakkar, Hakumat Rai, M.D., Pacific Palisades, CA
Kakkar, Promila Rai, M.D., Pacific Palisades, CA
Kang, Jin Koo, M.D., Brightwaters, NY
Kaplan, Harriet Smith, M.D., Rancho Palos Verdes, CA
Karme Laila, M.D., Pasadena, CA
Kashfi, Ali A., M.D., Winter Haven, FL
Kaye, Walter, M.D., Bethesda, MD
Kelly, William L., M.D., Reno, NV
Kilduff, Raymond F., M.D., Berkeley, CA
King, Hugh A., Jr., M.D., Pineville, LA
Kline, David M., M.D., Oakland, CA
Kohn, Ben, M.D., Beverly Hills, CA
Kostick, Neil S., M.D., Albany, CA
Kram, Leonard W., M.D., Los Angeles, CA
Krimerman, Eleanor J., M.D., New Orleans, LA
Kwon, In Won, M.D., Roseburg, OR

Larrabee, James F., Jr., M.D., Culver City, CA
Lavrence, Thomas I., M.D., Morristown, NJ
Leffert, Mark, M.D., Del Mar, CA
Levin, Edmund C., M.D., Berkeley, CA
Levy, Michael A., M.D., Fort Ord, CA
Lewis, Argus Wayne, M.D., Foster City, CA
Lewis, Jonathan D., M.D., Chicago, IL
Limsuwan, Nongpanga, M.D., Austin, TX
Liu, Pung Show, M.D., Petersburg, VA
Loesch, John George, M.D., Chicago, IL
Lomas, Harvey D., M.D., Redlands, CA
Luce, Eleanor M., M.D., Berkeley, CA

Malinak, Dennis Paul, M.D., San Rafael, CA
Marcel, Leonard J., M.D., Portland, OR
Martin, Harvey C., M.D., Wichita Falls, TX
Mee-Lee, Denis, M.D., Honolulu, HI
Miley, Dennis P., M.D., San Diego, CA
Miller, J. Allen, M.D., Sacramento, CA
Missett, James R., M.D., Palo Alto, CA
Moore, Ardean Carol, M.D., San Francisco, CA
Mortimer, Raymond E., M.D., W. Palm Beach, FL
Motlagh, Mojtaba Rastgar, M.D., Lawrenceville, NJ
Muir, Keith M., M.D., New York, NY

Nadel, Stanley Mark, M.D., El Cajon, CA
Nash, Gerald M., M.D., Santa Cruz, CA
Natu, Himani W., M.D., Aptos, CA
Newstadt, Geoffrey J., M.D., Los Angeles, CA

Okimoto, Joseph T., M.D., Seattle, WA

Pakier, Oscar, M.D., Van Nuys, CA
Pally, Regina, M.D., Los Angeles, CA

Paul, Henry, M.D., Brockport, NY
Pentz, Paul Gordon, M.D., Hartford, CT
Perlo, Solomon, M.D., Tarzana, CA
Prasad, Prem Shanker, M.D., Farmington Hills, MI
Pravda, Marvin, M.D., Cincinnati, OH
Pro, John Dennis, M.D., Seattle, WA
Prosen, Melvin, M.D., Glencoe, IL

Rajendran, Somasundaram, M.D., Lincoln, NB
Ramchandani, Deepika R., M.D., New Hyde Park, NY
Reby, Morris, M.D., Elberon, NJ
Reding, Michael Edward, M.D., Sacramento, CA
Reid, Laurance S., M.D., Pasadena, CA
Renson, Jean, M.D., San Carlos, CA
Ritvo, David Z., M.D., San Francisco, CA
Robbins, Jeffrey M., M.D., Los Angeles, CA
Rogers, Sheldon, M.D., Portsmouth, OH
Rosenzweig, Marvin W., M.D., Berkeley, CA
Rowlett, David B., III, M.D., Carnation, WA

Sachdev, Usha, M.D., Fountain Valley, CA
Schmits, George Michael, M.D., Orangevale, CA
Schoenfeld, Frank Barry, M.D., San Francisco, CA
Schultz, Steven L., M.D., Scottsdale, AZ
Schwartz, David A., M.D., San Anselmo, CA
Scott, Robert Charles, M.D., San Francisco, CA
Shakir, Saad Adnan, M.D., Palo Alto, CA
Sheldon, Robert Burnett, M.D., Bryan, TX
Simmons, Ethel Sultana A., M.D., Carson, CA
Simon, Travis Theodore, M.D., Vancouver, B.C., Canada
Smolowe, John S., M.D., Menlo Park, CA
Spar, James Edward, M.D., Los Angeles, CA
Steiner, Hans, M.D., Stanford, CA
Steip, Richard H., M.D., Torrance, CA
Stewart, James H., M.D., Santa Barbara, CA
Stipes, Albert H., M.D., Elmhurst, IL
Switkes, Daniel A., M.D., Berkeley, CA

Tardy, Walter J., Jr., M.D., Jamaica, NY
Trachtenberg, David P., M.D., Walnut Creek, CA
Tsung, Der-cheng, M.D., Staten Island, NY
Tusel, Donald J., M.D., San Francisco, CA

Undavia, Suresh V., M.D., Binghamton, NY

Vagshenian, Gregory Simon, M.D., Palo Alto, CA
Veith, Richard Charles, M.D., Seattle, WA
Vlahakis, George J., Jr., M.D., Yakima, WA

Watson, Stanley J., Jr., M.D., Ann Arbor, MI
Weiss, Michael Stuart, M.D., Santa Monica, CA
Werbach, Melvyn R., M.D., Tarzana, CA
West, Joel Jay, M.D., Los Angeles, CA
Wilkins, Jeffery Neal, M.D., Santa Monica, CA
Williams, Charles Donald, M.D., Yakima, WA
Wilson, Charles L., M.D., Whittier, CA
Winocur, Emanuel M., M.D., San Bernardino, CA
Winston, Robert Alan, M.D., Redondo Beach, CA

Yasin, Fazle M., M.D., St. Louis, MO
Yballe, Sonia B., M.D., Chicago, IL
Yeu, Hokun, M.D., Pearl River, NY
Young, Brian Richard, M.D., Oakland, CA

Zablotny, Raymond, M.D., San Francisco, CA
Zeiguer, Luis V., M.D., New York, NY

NEUROLOGY

Allen, Aaron Russell, M.D., Laguna Niguel, CA
Atkinson, Richard P., M.D., Sacramento, CA

Belafsky, Melvin A., M.D., Encino, CA
Bounds, Jeffrey A., M.D., Loma Linda, CA

Chance, Janet M., M.D., Irvine, CA
Coull, Bruce M., M.D., Portland, OR

Dauben, Richard Dennis, M.D., Orange, CA
Diamond, Ellis R., M.D., Oceanside, CA
Dobkin, Bruce H., M.D., Los Angeles, CA

Flippin, Arthur M., M.D., San Diego, CA
Foa, Richard Paul, M.D., Portland, OR

Friedland, Robert P., M.D., Martinez, CA
Fung, Peter C., M.D., San Jose, CA

Ganji, Stalin, M.D., Allen Park, MI
Glackman, Richard S., M.D., San Pedro, CA
Goldstein, Jerome, M.D., San Francisco, CA

Healey, Edwin M., M.D., Springfield, VA
Helme, Robert D., M.D., Victoria, Australia
Hooge, John P., M.D., Vancouver, B.C., Canada
Hopkins, Elwood W., III, M.D., San Francisco, CA

Jurkowitz, Jay, M.D., Long Beach, CA

Laxer, Kenneth D., M.D., Portland, OR
Leonard, Hubert A., M.D., Portland, OR
Ludwig, Barry I., M.D., Santa Monica, CA

Miller, Jack Edwin, M.D., San Rafael, CA

Neville, John R., M.D., Oakland, CA

O'Connor, Edward J., M.D., Santa Monica, CA
Olsen, Noel Helge, M.D., Adamsville, RI
Osborn, Kurt M., M.D., Eureka, CA
Overfield, W. Dale, M.D., Puyallup, WA

Prusiner, Stanley B., M.D., San Francisco, CA

Raffer, Paul K., M.D., Chula Vista, CA
Rosenbaum, Richard Barry, M.D., Portland, OR

Schwartz, Michael R., M.D., Kensington, MD
Simon, Robert E., M.D., Mission Viejo, CA
Slyter, Howard M., Jr., M.D., San Francisco, CA
Snider, Donald J., M.D., Anaheim, CA
Suranyi, Leslie, M.D., Cornwall, Ont., Canada

Tahmoush, Albert J., M.D., Rockville, MD

Varda, Darryl J., M.D., Ada, MI

Weir, William S., M.D., Northampton, MA
Williams, Michael H., M.D., Anchorage, AK

Yaffe, Michael Gerard, M.D., Whittier, CA

Zarchin, Lawrence Edward, M.D., Fairfax, VA

CHILD NEUROLOGY

Ch'ien, Lawrence Tien-Tso, M.D., Germantown, TN
Jackson, Anthony H., M.D., Springfield, MA
Knauss, Thomas A., M.D., Bellevue, WA
Sunder, Theodore R., M.D., Oceanside, CA

The following successfully completed the Board examination given by the Committee on Certification in Child Psychiatry in Pittsburgh, PA, February 25–27, 1979.

Arazan, Virginia, M.D., Sterling Heights, MI
Ardali, Cahit, M.D., Goshen, NY
Ardon, Marjorie S., M.D., Downers Grove, IL

Barber, Joan K., M.D., Arlington, VA
Barthel, Richard P., M.D., West Allis, WI
Berney, Jerome D., M.D., San Francisco, CA
Bezdek, Patrick T., M.D., Santa Monica, CA
Braga, Wander D., M.D., Delmar, NY
Brain, Lawrence A., M.D., Chevy Chase, MD
Brooks, Beth A., M.D., Detroit, MI
Brown, Craig A., M.D., Del Mar, CA
Burger, Frances L., M.D., Santa Monica, CA

Capers, Olivia, M.D., Philadelphia, PA
Carrera, Frank, III, M.D., Gainesville, FL
Chase, Gary A., M.D., Los Angeles, CA
Chaudhry, Dewat R., M.D., Rock Island, IL
Chung, Sul, M.D., Glendale, WI
Cladel, Charles E., Jr., M.D., Hummelstown, PA
Cochran, Stephen R., M.D., Ann Arbor, MI
Corman, Clifford L., M.D., Golden Valley, MN
Cresci, Joseph V., Jr., M.D., Cincinnati, OH
Curran, Barbara E., M.D., Dedham, MA

Dakin, Alice R., M.D., Oak Park, IL
Denaro, Anthony T., M.D., Avon, CT
Derivan, Albert T., M.D., Timonium, MD
Desai, Angeline C., M.D., Port St. Lucie, FL
Desai, Sureshchandra N., M.D., Port St. Lucie, FL
De Sa Pereira, Elisabeth A., M.D., Chicago, IL
Dozier, J. Emmett, Jr., M.D., Franklin, TN
Durfee, Michael J., M.D., S. Pasadena, CA

Evans, John W., M.D., San Anselmo, CA

Fagelman, Frances D., M.D., Dallas, TX
Falit, Harvey H., M.D., Ann Arbor, MI
Feinstein, Carl B., M.D., Washington, DC
Felch, Richard A., D.O., Delmar, NY
Ferre, Richard C., M.D., Salt Lake City, UT
Fink, Kenneth M., M.D., Huntington, WV
Fishler, Lois A., M.D., Valhalla, NY
Fox, David A., M.D., Fresno, CA
Friedman, Matthew R., M.D., Longmeadow, MA
Fuchs, Ruth, M.D., New York, NY

Gelber, Gary S., M.D., Mill Valley, CA
Gelpi, Jose A., M.D., York, PA
Geraty, Ronald D., M.D., Stoneham, MA
Gilmour, Edward E., M.D., New York, NY
Gislason, I. Lee, M.D., Orange, CA
Goetz, Patricia L., M.D., Pittsburgh, PA
Gordon, Barbara Groome, M.D., New York, NY
Grubbs, James H., M.D., Dallas, TX

Haines, Philip C., M.D., Detroit, MI
Hajal, Fady S., M.D., Ann Arbor, MI
Heller, J. Ronald, M.D., Charlottesville, VA
Hodas, Gordon R., M.D., Philadelphia, PA
Hollingsworth, Charles E., M.D., La Jolla, CA
Hong, K. Michael, M.D., Minneapolis, MN
Howe, Don D., M.D., Helotes, TX
Huebner, Hans F., M.D., New York, NY
Hugg, Terry W., M.D., New York, NY

Imani, Pouran A., M.D., Chicago, IL

Jankowski, Joseph J., M.D., Wellesley, MA
Javaid, Ghazala, M.D., Great Neck, NY
Jordan, Kent, M.D., Encinitas, CA
Joseph, Brian W., M.D., Topeka, KS

Josiah, Stuart E., M.D., Fairfield, CT

Kallen, Lowell H., M.D., San Francisco, CA
Kaplan, Sandra J., M.D., Port Washington, NY
Kennedy, Paul V., M.D., New York, NY
Kessler, Mina D., M.D., Pittsburgh, PA
Kiehl, Carl G., M.D., Nyack, NY
Killoran, Sean M., M.D., Bremerton, WA
Korrol, Charles R., D.O., E. Northport, NY

Ladov, Norman H., M.D., Wayne, NJ
Lear, Charles E., M.D., Missoula, MT
Lew, Arthur, M.D., New Rochelle, NY
Licamele, William L., M.D., Arlington, VA

Maloney, Michael J., M.D., Cincinnati, OH
Marcus, Rose M., M.D., Flushing, NY
Meisel, Frederick L., M.D., Cambridge, MA

Najarian, Louis M., M.D., Manhasset, NY
Naylor, Kenneth A., M.D., Raleigh, NC
Nelson, Gerald E., M.D., Del Mar, CA

Oldham, Robert L., M.D., Glendale, CA
Osborn, Michael V., M.D., Hastings/Hudson, NY

Pach, Lleni, M.D., Syracuse, NY
Pagan, Domingo, M.D., Ottawa, Ont., Canada
Palmer, Robert C., M.D., Pomona, NY
Payton, James B., M.D., Cincinnati, OH
Pearlman, Mary I., M.D., Madison, WI
Petersen, P. Brent, M.D., Salt Lake City, UT
Porcher, William J., M.D., Austin, TX
Price, Lloyd F., M.D., Boston, MA

Rabinovich, Harris, M.D., New York, NY
Rashkis, Shirley R., M.D., Gladwyne, PA
Ray, Ritz C., Jr., M.D., Winston-Salem, NC
Reichenthal, Jeffrey A., M.D., Palo Alto, CA
Reiskin, Ira M., M.D., Newton, MA
Reynolds, Elizabeth, M.D., Napa, CA
Rivera-Lara, Ruth M., M.D., Philadelphia, PA
Rivinus, Timothy M., M.D., Boston, MA
Rutt, Carl N., M.D., Lennox, SD

Samuelly, Michaela, M.D., Brooklyn, NY
Schonfeld, Burton G., M.D., Washington, DC
Schussler, Irwin, D.O., Fort Worth, TX
Simmons, Randon C., M.D., Pittsburgh, PA
Slack, Robert G., M.D., Dayton, OH
Sondheimer, Adrian N., M.D., New York, NY
Stambler, Morris J., M.D., Watertown, MA
Steckler, Eric A., M.D., Fairfax, VA
Sternbach, Susanne E., M.D., New York, NY
Steude, Philip G., M.D., Columbia, SC
Stine, John J., M.D., Chappaqua, NY

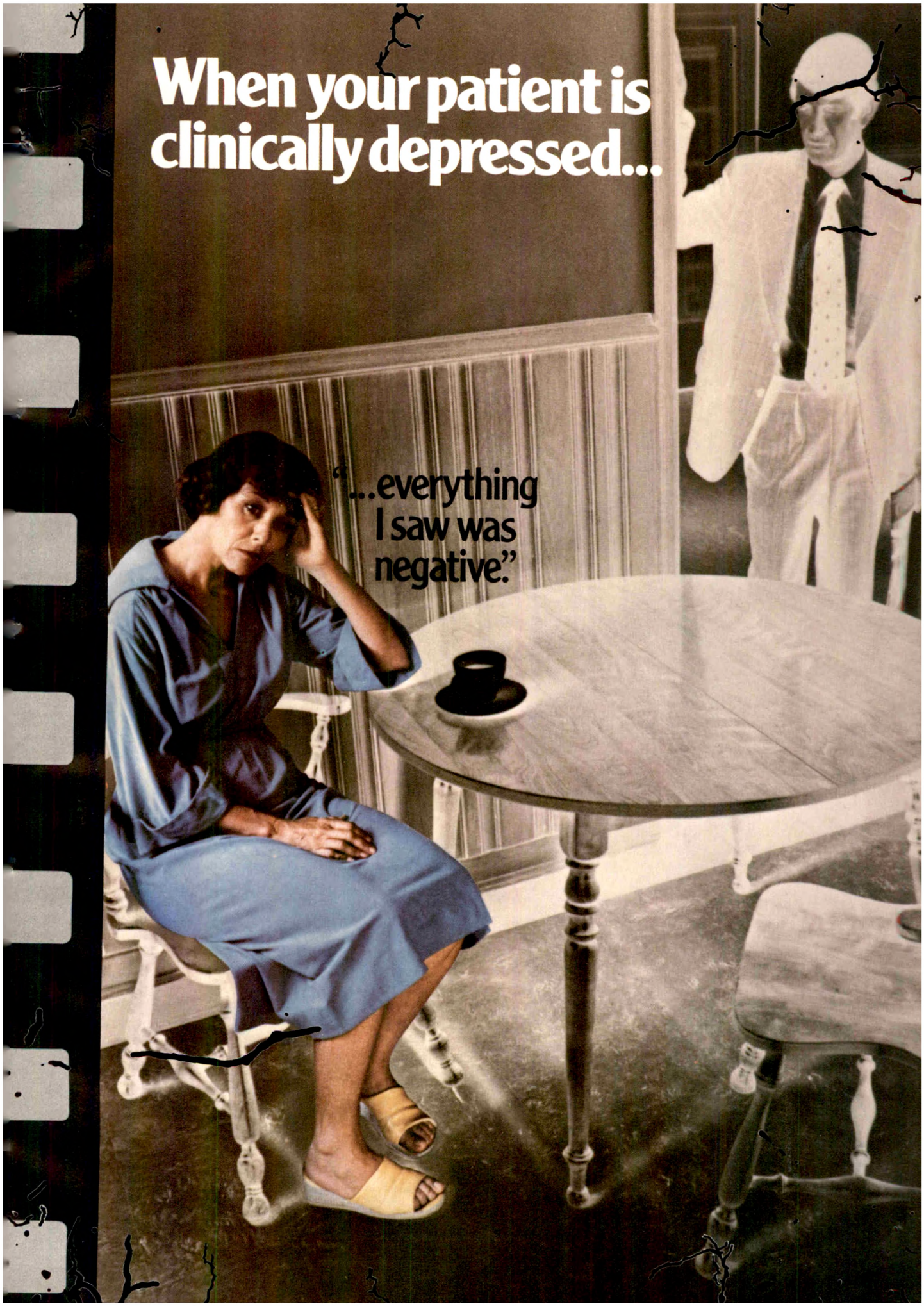
Tambyraja, Samuel M., M.D., Akron, OH
Terry, William T., M.D., Reno, NY
Thompson, Dennis S., D.O., Chicago, IL

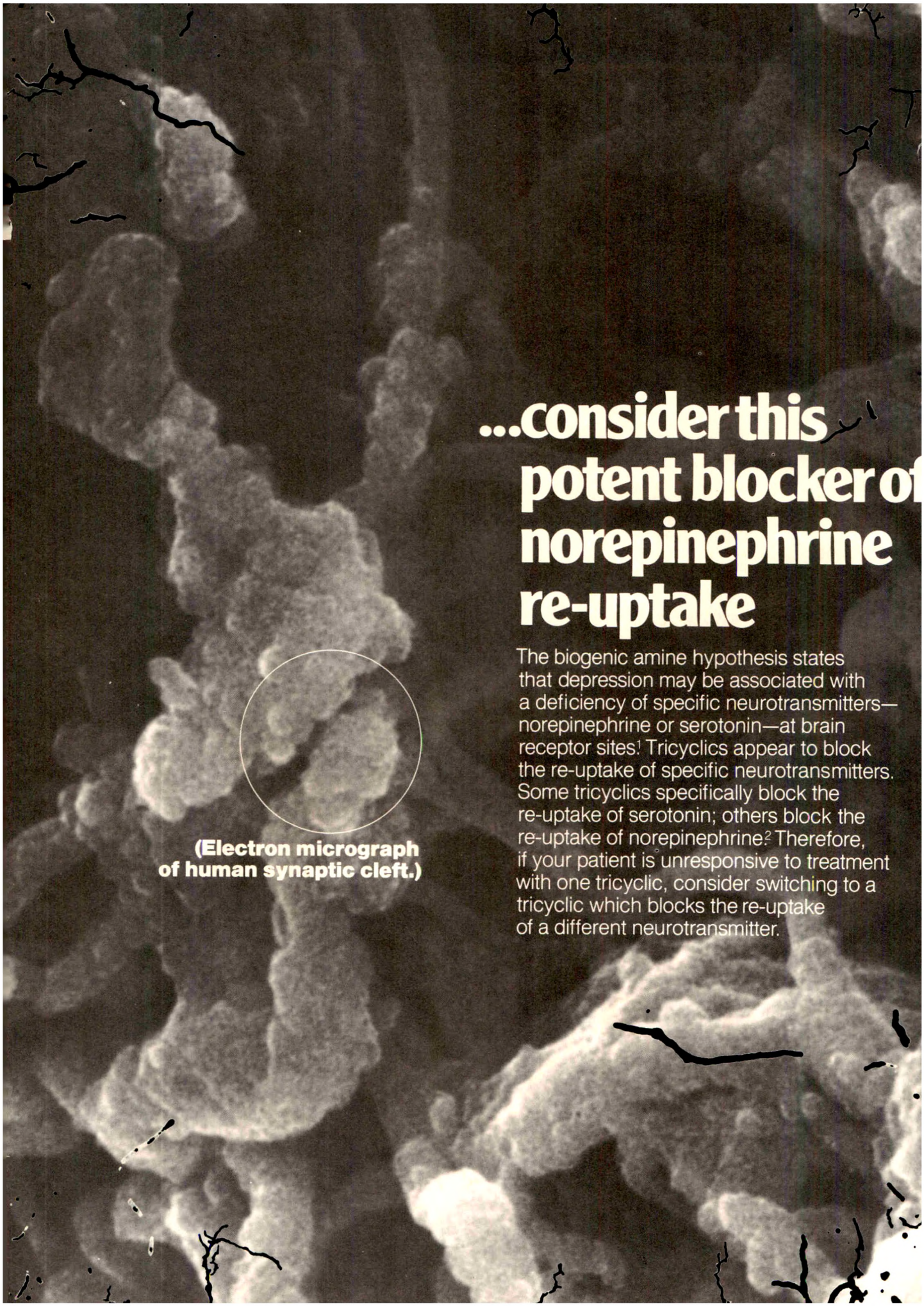
Vary, Marshall G., M.D., Boulder, CO
Vintas, Gustavo H., M.D., New York, NY

Wiggins, Kenneth M., M.D., Richardson, TX
Wong, Ruth, M.D., Paterson, NJ
Woolston, Joseph L., M.D., Madison, CT

Zegarra de Polo, Myrna, M.D., Rio Piedras, PR
Ziegler, Robert G., M.D., Cambridge, MA

Bernstein, Basil G., M.D., Los Angeles, CA, was among those who successfully completed the Board examination given by the Committee on Certification in Child Psychiatry in New Orleans, LA, February 26–28, 1979. The other successful candidates were listed in the June 1978 issue of the *Journal*.


When your patient is clinically depressed...
"...everything I saw was negative."



...consider this potent blocker of norepinephrine re-uptake

The biogenic amine hypothesis states that depression may be associated with a deficiency of specific neurotransmitters—norepinephrine or serotonin—at brain receptor sites.[1] Tricyclics appear to block the re-uptake of specific neurotransmitters. Some tricyclics specifically block the re-uptake of serotonin; others block the re-uptake of norepinephrine.[2] Therefore, if your patient is unresponsive to treatment with one tricyclic, consider switching to a tricyclic which blocks the re-uptake of a different neurotransmitter.

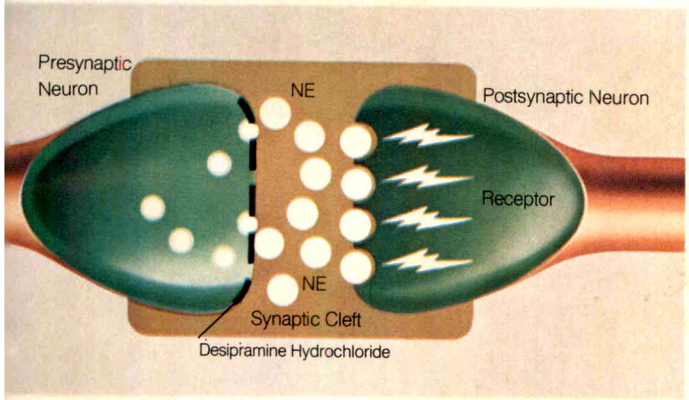
(Electron micrograph of human synaptic cleft.)

Norpramin®
(desipramine hydrochloride tablets NF)

25, 50, 75, 100, 150 mg.

Potent blocker of norepinephrine re-uptake

According to current theory[1,3] some tricyclic antidepressants may fail in certain types of depression because they are not sufficiently potent blockers of norepinephrine re-uptake. Laboratory studies have shown that desipramine is the most potent inhibitor of norepinephrine re-uptake.[4] Norpramin may work in some patients when other tricyclic antidepressants do not.



Consider it now, consider it next.

Less anticholinergic activity

(See Warnings section.)

Studies in animals[5] and in normal human subjects[6,7] have shown that desipramine has significantly less anticholinergic activity than amitriptyline or doxepin. The single bond side chain is associated with lower anticholinergic activity than these other leading antidepressants. This may mean:

- less dry mouth
- less blurred vision
- less urinary retention

Helps patients remain calm but active

Norpramin does not usually inhibit normal activity although patients should be cautioned as to driving or operating machinery if drowsiness occurs.

In addition...

Begins to improve sleep patterns within one week in some patients[8]

- Norpramin helps relieve the sleep disturbances that often accompany depression.
- As the depression is relieved, the difficulty in falling asleep, restlessness, and early morning awakening diminish.

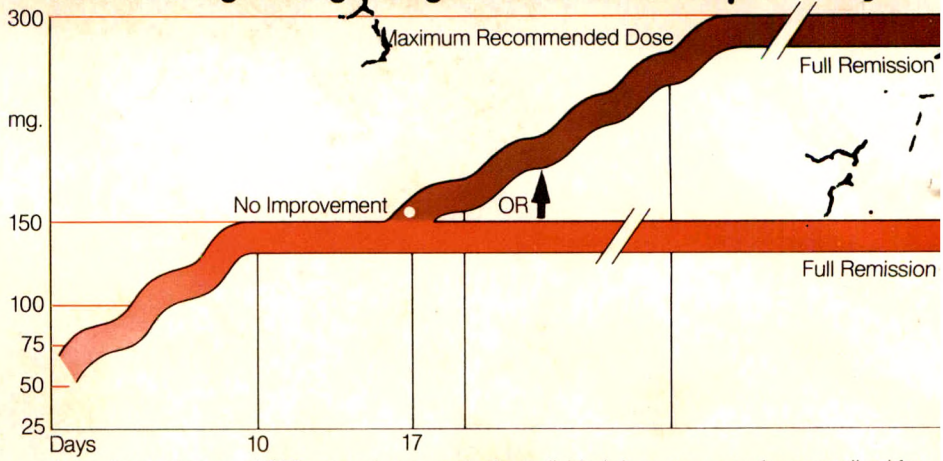
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1. Maas, J.W.: Biogenic amines and depression. Biochemical and pharmacological separation of two types of depression. Arch. Gen. Psychiat. 32:1357-1361, 1975. **2.** Mendels, J., Stern, S., and Frazer, A.: Dis. Nerv. Syst. 37:3, 1976. **3.** Davis, J.M.: Central biogenic amines and theories of depression and mania. In, Phenomenology and Treatment of Depression, W.E. Fann, I. Karacan, A.D. Pokorny, and R.L. Williams, Ed., New York, Spectrum Publications, Inc., 1977, pp. 17-32. **4.** Ross, S.B. and Renyi, A.L.: Tricyclic antidepressant agents. 1. Comparison of the inhibition of the uptake of ^{3}H-noradrenaline and ^{14}C-5-hydroxytryptamine in slices and crude synaptosome preparations of the midbrain-hypothalamus region of the rat brain. Acta Pharmacol. (Kobenhavn) 36:382-394, 1975. **5.** Snyder, S.H. and Yamamura, H.I.: Antidepressants and the muscarinic acetylcholine receptor. Arch. Gen. Psychiat. 34:236-239, 1977. **6.** Blackwell, B., Stefopoulos, A., Enders, P., Kuzma, R., and Adolphe, A.: The anticholinergic activity of two tricyclic antidepressants. Amer. J. Psychiat., 135:722-724, June 1978. **7.** Peterson, G.R., Hostetler, R.M., Blackwell, B., Kuzma, R., and Adolphe, A.B.: Effects of the tricyclic antidepressants desipramine and doxepin on anticholinergic and CNS activity in non-depressed volunteers. Neuroscience Abstr. 3:447, 1977. **8.** Zung, W.W.K.: The pharmacology of disordered sleep. A laboratory approach. J.A.M.A. 211:1532-1534, 1970.

(For prescribing information see next page.)

Merrell

Now 300 mg. dosage range broadens clinical flexibility



A maximum dosage of 300 mg. a day, as a single or divided dose, may now be prescribed for Norpramin. Since inadequate dosage is thought to be one of the major causes of treatment failure, the new therapeutic range should prove clinically helpful. Plasma desipramine measurement would constitute the optimal guide to dosage monitoring.

Norpramin®
(desipramine hydrochloride tablets NF)

AVAILABLE ONLY ON PRESCRIPTION

Brief Summary

MECHANISM OF ACTION: Available evidence suggests that many depressions have a biochemical basis in the form of a relative deficiency of neurotransmitters such as norepinephrine and serotonin. Norepinephrine deficiency may be associated with relatively low urinary 3-methoxy-4-hydroxyphenyl glycol (MHPG) levels, while serotonin deficiencies may be associated with low spinal fluid levels of 5-hydroxyindolacetic acid.

While the precise mechanism of action of the tricyclic antidepressants is unknown, a leading theory suggests that they restore normal levels of neurotransmitters by blocking the re-uptake of these substances from the synapse in the central nervous system.

Evidence indicates that the secondary amine tricyclic antidepressants, including Norpramin, may have greater activity in blocking the re-uptake of norepinephrine. Tertiary amine tricyclic antidepressants, such as amitriptyline, may have greater effect on serotonin re-uptake.

Norpramin (desipramine hydrochloride) is not a monoamine oxidase (MAO) inhibitor and does not act primarily as a central nervous system stimulant. It has been found in some studies to have a more rapid onset of action than imipramine. Earliest therapeutic effects may occasionally be seen in 2 to 5 days, but full treatment benefit usually requires 2 to 3 weeks to obtain.

INDICATIONS: Norpramin (desipramine hydrochloride) is indicated for relief of symptoms in various depressive syndromes, especially endogenous depression.

CONTRAINDICATIONS: Desipramine hydrochloride should not be given in conjunction with, or within 2 weeks of, treatment with an MAO inhibitor drug; hyperpyretic crises, severe convulsions, and death have occurred in patients taking MAO inhibitors and tricyclic antidepressants. When Norpramin (desipramine hydrochloride) is substituted for an MAO inhibitor, at least 2 weeks should elapse between treatments. Norpramin should then be started cautiously and should be increased gradually.

The drug is contraindicated in the acute recovery period following myocardial infarction. It should not be used in those who have shown prior hypersensitivity to the drug. Cross sensitivity between this and other dibenzazepines is a possibility.

WARNINGS: 1. Extreme caution should be used when this drug is given in the following situations: a. In patients with cardiovascular disease, because of the possibility of conduction defects, arrhythmias, tachycardias, strokes, and acute myocardial infarction. b. In patients with a history of urinary retention or glaucoma, because of the anticholinergic properties of the drug. c. In patients with thyroid disease or those taking thyroid medication, because of the possibility of cardiovascular toxicity, including arrhythmias. d. In patients with a history of seizure disorder, because this drug has been shown to lower the seizure threshold. **2.** This drug is capable of blocking the antihypertensive effect of guanethidine and similarly acting compounds. **3.** USE IN PREGNANCY: Safe use of desipramine hydrochloride during pregnancy and lactation has not been established; therefore, if it is to be given to pregnant patients, nursing mothers, or women of childbearing potential, the possible benefits must be weighed against the possible hazards to mother and child. Animal reproductive studies have been inconclusive. **4.** USE IN CHILDREN: Norpramin (desipramine hydrochloride) is not recommended for use in children since safety and effectiveness in the pediatric age group have not been established. **5.** The patient should be cautioned that this drug may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery. **6.** In patients who may use alcohol excessively, it should be borne in mind that the potentiation may increase the danger inherent in any suicide attempt or overdosage.

PRECAUTIONS: 1. It is important that this drug be dispensed in the least possible quantities to depressed outpatients, since suicide has been accomplished with this class of drug. Ordinary prudence requires that children not have access to this drug or to potent drugs of any kind; if possible this drug should be dispensed in containers with child-resistant safety closures. Storage of this drug in the home must be supervised responsibly. **2.** If serious adverse effects occur, dosage should be reduced or treatment should be altered. **3.** Norpramin (desipramine hydrochloride) therapy in patients with manic-depressive illness may induce a hypomanic state after the depressive phase terminates. **4.** The drug may cause exacerbation of psychosis in schizophrenic patients. **5.** Close supervision and careful adjustment of dosage are required when this drug is given concomitantly with anticholinergic or sympathomimetic drugs. **6.** Patients should be warned that while taking this drug their response to alcoholic beverages may be exaggerated. **7.** Clinical experience in the concurrent administration of ECT and antidepressant drugs is limited. Thus, if such treatment is essential, the possibility of increased risk relative to benefits should be considered. **8.** The sedative effects of Norpramin and benzodiazepines (*e.g.*, chlordiazepoxide or diazepam) are additive. Both the sedative and anticholinergic effects of the major tranquilizers are additive to those of Norpramin. **9.** This drug should be discontinued as soon as possible prior to elective surgery because of the possible cardiovascular effects. Hypertensive episodes have been observed during surgery in patients taking desipramine hydrochloride. **10.** Both elevation and lowering of blood sugar levels have been reported. **11.** Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during therapy; the drug should be discontinued if there is evidence of pathologic neutrophil depression.

ADVERSE REACTIONS: Note: Included in the following listing are a few adverse reactions that have not been reported with this specific drug. However, the pharmacologic similarities among the tricyclic antidepressant drugs require that each of the reactions be considered when Norpramin (desipramine hydrochloride) is given.

Cardiovascular: hypotension, hypertension, tachycardia, palpitation, arrhythmias, heart block, myocardial infarction, stroke.

Psychiatric: confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia and nightmares; hypomania; exacerbation of psychosis.

Neurologic: numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures; alteration in EEG patterns; tinnitus.

Anticholinergic: dry mouth, and rarely associated sublingual adenitis; blurred vision, disturbance of accommodation, mydriasis, increased intraocular pressure; constipation, paralytic ileus; urinary retention, delayed micturition, dilatation of urinary tract.

Allergic: skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight), edema (of face and tongue or general), drug fever, cross sensitivity with other tricyclic drugs.

Hematologic: bone marrow depressions including agranulocytosis, eosinophilia, purpura, thrombocytopenia.

Gastrointestinal: anorexia, nausea and vomiting, epigastric distress, peculiar taste, abdominal cramps, diarrhea, stomatitis, black tongue.

Endocrine: gynecomastia in the male, breast enlargement and galactorrhea in the female; increased or decreased libido, impotence, testicular swelling; elevation or depression of blood sugar levels.

Other: jaundice (simulating obstructive), altered liver function; weight gain or loss; perspiration, flushing; urinary frequency, nocturia; parotid swelling; drowsiness, dizziness, weakness and fatigue, headache; alopecia.

Withdrawal Symptoms: Though not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache, and malaise.

DOSAGE AND ADMINISTRATION: Not recommended for use in children. Lower dosages are recommended for elderly patients and adolescents. Lower dosages are also recommended for outpatients compared to hospitalized patients, who are closely supervised. Dosage should be initiated at a low level and increased according to clinical response and any evidence of intolerance. Following remission, maintenance medication may be required for a period of time and should be at the lowest dose that will maintain remission.

Usual Adult Dose: The usual adult dose is 100 to 200 mg. per day. In more severely ill patients, dosage may be further increased gradually to 300 mg./day if necessary. Dosages above 300 mg./day are not recommended.

Dosage should be initiated at a lower level and increased according to tolerance and clinical response.

Treatment of patients requiring as much as 300 mg. should generally be initiated in hospitals, where regular visits by the physician, skilled nursing care, and frequent electrocardiograms (ECG's) are available.

The best available evidence of impending toxicity from very high doses of Norpramin is prolongation of the QRS or QT intervals on the ECG. Prolongation of the PR interval is also significant, but less closely correlated with plasma levels. Clinical symptoms of intolerance, especially drowsiness, dizziness, and postural hypotension, should also alert the physician to the need for reduction in dosage. Plasma desipramine measurement would constitute the optimal guide to dosage monitoring.

Initial therapy may be administered in divided doses or a single daily dose.

Maintenance therapy may be given on a once-daily schedule for patient convenience and compliance.

Adolescent and Geriatric Dose: The usual adolescent and geriatric dose is 25 to 100 mg. daily.

Dosage should be initiated at a lower level and increased according to tolerance and clinical response to a usual maximum of 100 mg. daily. In more severely ill patients, dosage may be further increased to 150 mg./day. Doses above 150 mg./day are not recommended for these age groups.

Initial therapy may be administered in divided doses or a single daily dose.

Maintenance therapy may be given on a once-daily schedule for patient convenience and compliance.

OVERDOSAGE: See prescribing information for a discussion of symptoms and treatment of overdose.

Product Information as of April, 1978

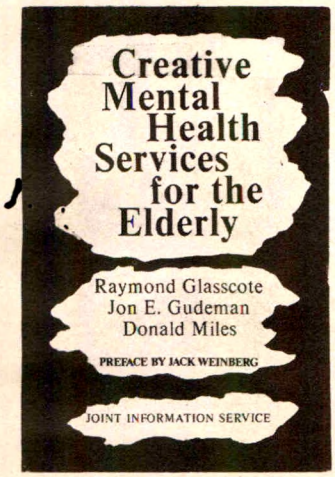
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8-3856 (Y546A)*

The Joint Information Service of the American Psychiatric Association and the Mental Health Association releases Two New Volumes That

Focus on the Elderly



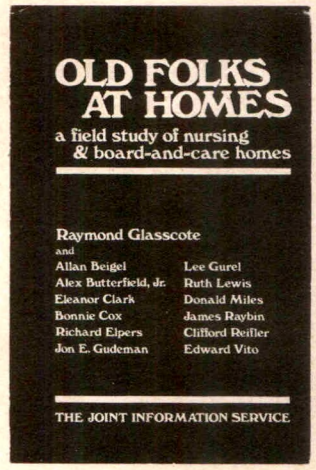
This carefully documented book reports a field study of ten programs, four in America and six in England and Scandinavia, which serve as practical demonstrations of creative mental health services for the elderly. They include:

- a novel "Neighborhood Family" providing vigorous support to elderly residents of several trailer parks
- a high school for the elderly which sends its students abroad for study trips
- a "Human Development Project" that focuses on responding to the psychological needs of the elderly
- a carefully coordinated system of "respite hospitalization," which promotes the health of the elderly while allowing maximum use of hospital beds and family resources
- a "Lucy Booth," patterned after the Peanuts comic strip, which provides easy access to many kinds of services
- and many other innovative, successful approaches.

Each program is subjected to intensive scrutiny. The resulting document becomes a handbook, and, as well, a forum through which some of the world's most experienced practitioners of "the psychiatry of old age" present their views.

Dr. Wilma Donohue, director of the International Center for Social Gerontology, characterizes this volume as "a masterful job of putting everything in relief . . . the guidelines for action are right here."

190 pages. Casebound. Price $8.50.



Concerned about the very bad reputation that nursing homes have received from many quarters in recent years, the Joint Information Service set up a field study to visit a systematically chosen sample of nursing homes, and board-and-care homes as well, to see to what extent the care of and the quality of life for the patients differed from the geriatrics units of state hospitals. Altogether, sixty nursing homes and 31 board-and-care homes in ten locales were visited, in each case by the senior author accompanied by two mental health professionals.

The teams were surpised to learn that care and quality of life in many of the nursing and board-and-care homes they visited were better—sometimes dramatically better—than that in the mental health facilities and often at lower cost.

This unique publication provides an overview of all facilities visited plus vignettes of approximately half. Federal and state rules and regulations are also expertly synthesized into brief and readable style.

148 pages. Casebound. Price $6.50

Publication Sales Division
American Psychiatric Association
1700 18th St., N.W., Washington, D.C. 20009

Please send: _____ copies of *Creative Mental Health Services for the Elderly* @ $8.50 per copy
_____ copies of *Old Folks at Homes* @ $6.50 per copy
_____ sets (one copy of each volume) at the *special combination price of $13.50* (a savings of $1.50 over the regular combined price of $15.00)

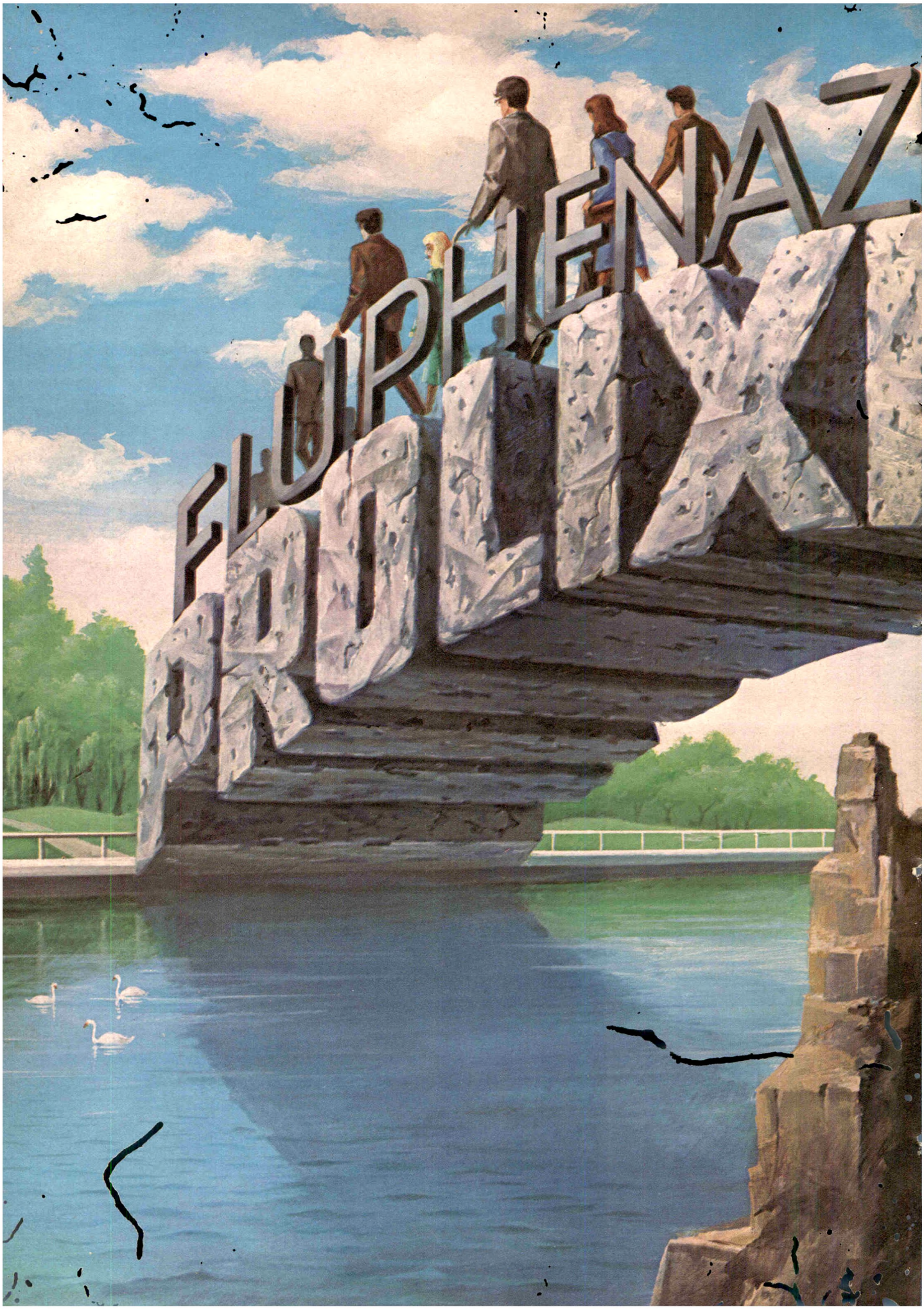
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The Prolixin® (Fluphenazine) system of schizophrenia management—

spanning virtually every clinical situation

There is scarcely a patient-type presenting with schizophrenia that cannot be successfully managed with one of the numerous forms of Prolixin (Fluphenazine).

For the newly admitted, agitated patient, a first choice would be short-acting Prolixin Injection (Fluphenazine Hydrochloride Injection USP).

For long-term management of more "dependable" patients, you have the titration flexibility of four potencies of Prolixin Tablets (Fluphenazine Hydrochloride Tablets USP).

For patients who "cheek" or otherwise resist tablet medication, you can utilize pleasant tasting, orange-flavored Prolixin Elixir (Fluphenazine Hydrochloride Elixir USP).

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Unimatic® Single Dose Syringe
25 mg in 1 ml

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Hydrochloride
Injection USP

10 ml multiple dose vial
2.5 mg per ml

5 ml
multiple dose vial
25 mg per ml

Prolixin® Elixir
Fluphenazine
Hydrochloride
Elixir USP

Prolixin® Tablets
Fluphenazine
Hydrochloride
Tablets USP

10 mg

5 mg

2.5 mg

1 mg

0.5 mg per ml

See next page for brief summaries.

PROLIXIN® (Fluphenazine Hydrochloride)
TABLETS/ELIXIR/INJECTION

Prolixin Tablets (Fluphenazine Hydrochloride Tablets USP) provide 1, 2.5, 5, or 10 mg fluphenazine hydrochloride per tablet. Prolixin Elixir (Fluphenazine Hydrochloride Elixir USP) provides 0.5 mg fluphenazine hydrochloride per ml (2.5 mg per 5 ml teaspoonful) with 14% alcohol by volume. Prolixin Injection (Fluphenazine Hydrochloride Injection USP) provides 2.5 mg fluphenazine hydrochloride per ml; it contains 0.1% methylparaben and 0.01% propylparaben as preservatives.

CONTRAINDICATIONS: In presence of suspected or established subcortical brain damage. In patients who have a blood dyscrasia or liver damage, or who are receiving large doses of hypnotics, or who are comatose or severely depressed. In patients who have shown hypersensitivity to fluphenazine; cross-sensitivity to phenothiazine derivatives may occur.

WARNINGS: Mental and physical abilities required for driving a car or operating heavy machinery may be impaired by use of this drug. Potentiation of effects of alcohol may occur. Safety and efficacy in children have not been established because of inadequate experience in use in children.

Usage in Pregnancy: Safety for use during pregnancy has not been established; weigh possible hazards against potential benefits if administering this drug to pregnant patients.

PRECAUTIONS: Caution must be exercised if another phenothiazine compound caused cholestatic jaundice, dermatoses or other allergic reactions because of the possibility of cross-sensitivity. When psychotic patients on large doses of a phenothiazine drug are to undergo surgery, hypotensive phenomena should be watched for; less anesthetics or central nervous system depressants may be required. Because of added anticholinergic effects, fluphenazine may potentiate the effects of atropine.

Use fluphenazine cautiously in patients exposed to extreme heat or phosphorus insecticides; in patients with a history of convulsive disorders since grand mal convulsions have occurred; and in patients with special medical disorders such as mitral insufficiency or other cardiovascular diseases, and pheochromocytoma. Bear in mind that with prolonged therapy there is the possibility of liver damage, pigmentary retinopathy, lenticular and corneal deposits, and development of irreversible dyskinesia.

There is sufficient experimental evidence to conclude that chronic administration of antipsychotic drugs which increase prolactin secretion has the potential to induce mammary neoplasms in rodents under the appropriate conditions. There are recognized differences in the physiological role of prolactin between rodents and humans. Since there are, at present, no adequate epidemiological studies, the relevance to human mammary cancer risk from prolonged exposure to fluphenazine hydrochloride and other antipsychotic drugs is not known.

Periodic checking of hepatic and renal functions and blood picture should be done. Monitor renal function of patients on long-term therapy; if BUN becomes abnormal, discontinue fluphenazine. "Silent pneumonias" are possible.

Abrupt Withdrawal: In general, phenothiazines do not produce psychic dependence. However, gastritis, nausea and vomiting, dizziness, and tremulousness have been reported following abrupt cessation of high dose therapy; reports suggest that these symptoms can be reduced if concomitant antiparkinsonian agents are continued for several weeks after the phenothiazine is withdrawn.

ADVERSE REACTIONS: *Central Nervous System*—Extrapyramidal symptoms are most frequently reported. Most often these symptoms are reversible, but they may be persistent. They include pseudoparkinsonism, dystonia, dyskinesia, akathisia, oculogyric crises, opisthotonos, hyperreflexia. The incidence and severity of such reactions will depend more on individual patient sensitivity, but dosage level and patient age are also determinants. As these reactions may be alarming, the patient should be forewarned and reassured. These reactions can usually be controlled by administration of an anti-parkinsonian drug such as benztropine mesylate and by subsequent reduction in dosage.

Persistent Tardive Dyskinesia: As with all antipsychotic agents, persistent and sometimes irreversible tardive dyskinesia may appear in some patients on long-term therapy or may occur after discontinuation of drug. The risk seems greater in elderly patients, especially females, on high dosages. The syndrome is characterized by rhythmical involuntary movements of tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and may be accompanied by involuntary movements of extremities. There is no known effective therapy for tardive dyskinesia; usually the symptoms are not alleviated by antiparkinsonism agents. If the symptoms appear, discontinuation of all antipsychotic agents is suggested. The syndrome may be masked if treatment is reinstituted, or drug dosage increased, or a different antipsychotic agent used. Reports are that fine vermicular movements of the tongue may be an early sign of the syndrome which may not develop if medication is stopped at that time.

Phenothiazine derivatives have been known to cause restlessness, excitement, or bizarre dreams; reactivation or aggravation of psychotic processes may be encountered. If drowsiness or lethargy occur, the dosage may need to be reduced. Dosages, far in excess of the recommended amounts, may induce a catatonic-like state.

Autonomic Nervous System—Hypertension and fluctuations in blood pressure have been reported. Although hypotension is rarely a problem, patients with pheochromocytoma, cerebral vascular or renal insufficiency or severe cardiac reserve deficiency such as mitral insufficiency appear to be particularly prone to this reaction and should be observed carefully. Supportive measures including intravenous vasopressor drugs should be instituted immediately should severe hypotension occur; Levarterenol Bitartrate Injection is the most suitable drug; *epinephrine should not be used* since phenothiazine derivatives have been found to reverse its action. Nausea, loss of appetite, salivation, polyuria, perspiration, dry mouth, headache and constipation may occur. Reducing or temporarily discontinuing the dosage will usually control these effects. Blurred vision, glaucoma, bladder paralysis, fecal impaction, paralytic ileus, tachycardia, or nasal congestion have occurred in some patients on phenothiazine derivatives.

Metabolic and Endocrine—Weight change, peripheral edema, abnormal lactation, gynecomastia, menstrual irregularities, false results on pregnancy tests, impotency in men and increased libido in women have occurred in some patients on phenothiazine therapy.

Allergic Reactions—Itching, erythema, urticaria, seborrhea, photosensitivity, eczema and exfoliative dermatitis have been reported with phenothiazines. The possibility of anaphylactoid reactions should be borne in mind.

Hematologic—Blood dyscrasias including leukopenia, agranulocytosis, thrombocytopenic or nonthrombocytopenic purpura, eosinophilia, and pancytopenia have been observed with phenothiazines. If soreness of the mouth, gums or throat or any symptoms of upper respiratory infection occur and confirmatory leukocyte count indicates cellular depression, therapy should be discontinued and other appropriate measures instituted immediately.

Hepatic—Liver damage manifested by cholestatic jaundice, particularly during the first months of therapy, may occur; treatment should be discontinued. A cephalin flocculation increase, sometimes accompanied by alterations in other liver function tests, has been reported in patients who have had no clinical evidence of liver damage.

Others—Sudden deaths have been reported in hospitalized patients on phenothiazines. Previous brain damage or seizures may be predisposing factors. High doses should be avoided in known seizure patients. Shortly before death, several patients showed flare-ups of psychotic behavior patterns. Autopsy findings have usually revealed acute fulminating pneumonia or pneumonitis, aspiration of gastric contents, or intramyocardial lesions. Although not a general feature of fluphenazine, potentiation of central nervous system depressants such as opiates, analgesics, antihistamines, barbiturates, and alcohol may occur.

Systemic lupus erythematosus-like syndrome, hypotension severe enough to cause fatal cardiac arrest, altered electrocardiographic and electroencephalographic tracings, altered cerebrospinal fluid proteins, cerebral edema, asthma, laryngeal edema, and angioneurotic edema; with long-term use, skin pigmentation and lenticular and corneal opacities have occurred with phenothiazines.

For full prescribing information, consult package inserts.

HOW SUPPLIED: *Tablets*—5 mg in bottles of 50 and 500; 2.5 mg and 5 mg in bottles of 50 and 500 and in Unimatic® single-dose cartons of 100; 10 mg in bottles of 50 and 500. *Elixir*—in bottles of 473 ml (1 pint) and in 60 ml dropper-assembly bottles with dropper calibrated at 0.5 ml (0.25 mg), 1 ml (0.5 mg), 1.5 ml (0.75 mg), and 2 ml (1 mg). *Injection*—in multiple-dose vials of 10 ml.

PROLIXIN DECANOATE®
Fluphenazine Decanoate Injection

Prolixin Decanoate (Fluphenazine Decanoate Injection) provides 25 mg fluphenazine decanoate per ml in a sesame oil vehicle with 1.2% (w/v) benzyl alcohol as a preservative.

CONTRAINDICATIONS: In presence of suspected or established subcortical brain damage; patients who have a blood dyscrasia or liver damage, or who are receiving large doses of hypnotics, or who are comatose or severely depressed. In patients who have shown hypersensitivity to fluphenazine; cross-sensitivity to phenothiazine derivatives may occur.

Not intended for use in children under 12.

WARNINGS: Mental and physical abilities required for driving a car or operating heavy machinery may be impaired by use of this drug. Physicians should be alert to the possibility of severe adverse reactions may occur which require immediate medical attention. Potentiation effects of alcohol may occur. Safety and efficacy in children have not been established because of inadequate experience in use in children.

Usage in Pregnancy: Safety for use during pregnancy has not been established; weigh possible hazards against potential benefits if administering this drug to pregnant patients.

PRECAUTIONS: Caution must be exercised if another phenothiazine compound caused cholestatic jaundice, dermatoses or other allergic reactions because of the possibility of cross-sensitivity. When psychotic patients on large doses of a phenothiazine drug are to undergo surgery, hypotensive phenomena should be watched for; less anesthetics or central nervous system depressants may be required. Because of added anticholinergic effects, fluphenazine may potentiate the effects of atropine.

Use fluphenazine decanoate cautiously in patients exposed to extreme heat or phosphorus insecticides; in patients with a history of convulsive disorders since grand mal convulsions have occurred; and in patients with special medical disorders such as mitral insufficiency or other cardiovascular diseases, and pheochromocytoma. Bear in mind that with prolonged therapy there is the possibility of liver damage, pigmentary retinopathy, lenticular and corneal deposits, and development of irreversible dyskinesia.

Fluphenazine decanoate should be administered under the direction of a physician experienced in the clinical use of psychotropic drugs. Periodic checking of hepatic and renal function and blood picture should be done. Renal function of patients on long-term therapy should be monitored; if BUN becomes abnormal, treatment should be discontinued. "Silent pneumonias" are possible.

There is sufficient experimental evidence to conclude that chronic administration of antipsychotic drugs which increase prolactin secretion has the potential to induce mammary neoplasms in rodents under the appropriate conditions. There are recognized differences in the physiological role of prolactin between rodents and humans. Since there are, at present, no adequate epidemiological studies, the relevance to human mammary cancer risk from prolonged exposure to fluphenazine decanoate and other antipsychotic drugs is not known.

ADVERSE REACTIONS: *Central Nervous System*—Extrapyramidal symptoms are most frequently reported. Most often these symptoms are reversible, but they may be persistent. They include pseudoparkinsonism, dystonia, dyskinesia, akathisia, oculogyric crises, opisthotonos, hyperreflexia. Muscle rigidity sometimes accompanied by hyperthermia has been reported following use of fluphenazine decanoate. One can expect a higher incidence of such reactions with fluphenazine decanoate than with less potent piperazine derivatives or straight-chain phenothiazines. The incidence and severity will depend more on individual patient sensitivity, dosage level and patient age are also determinants. As these reactions may be alarming, the patient should be forewarned and reassured. These reactions can usually be controlled by administration of an antiparkinsonian drug such as benztropine mesylate and by subsequent reduction in dosage.

Persistent Tardive Dyskinesia: As with all antipsychotic agents, persistent and sometimes irreversible tardive dyskinesia may appear in some patients on long-term therapy or may occur after discontinuation of drug. The risk seems greater in elderly patients, especially females, on high dosages. The syndrome is characterized by rhythmical involuntary movements of tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and may be accompanied by involuntary movements of extremities. There is no known effective therapy for tardive dyskinesia; usually the symptoms are not alleviated by antiparkinsonism agents. If the symptoms appear, discontinuation of all antipsychotic agents is suggested. The syndrome may be masked if treatment is reinstituted, or drug dosage increased, or a different antipsychotic agent used. Reports are that fine vermicular movements of the tongue may be an early sign of the syndrome which may not develop if medication is stopped at that time.

Phenothiazine derivatives have been known to cause restlessness, excitement, or bizarre dreams; reactivation or aggravation of psychotic processes may be encountered. If drowsiness or lethargy occur, the dosage may need to be reduced. Dosages, far in excess of the recommended amounts, may induce a catatonic-like state.

Autonomic Nervous System—Hypertension and fluctuations in blood pressure have been reported. Although hypotension is rarely a problem, patients with pheochromocytoma, cerebral vascular or renal insufficiency or severe cardiac reserve deficiency such as mitral insufficiency appear to be particularly prone to this reaction and should be observed carefully. Supportive measures including intravenous vasopressor drugs should be instituted immediately should severe hypotension occur; Levarterenol Bitartrate Injection is the most suitable drug; *epinephrine should not be used* since phenothiazine derivatives have been found to reverse its action. Nausea, loss of appetite, salivation, polyuria, perspiration, dry mouth, headache and constipation may occur. Reducing or temporarily discontinuing the dosage will usually control these effects. Blurred vision, glaucoma, bladder paralysis, fecal impaction, paralytic ileus, tachycardia, or nasal congestion have occurred in some patients on phenothiazine derivatives.

Metabolic and Endocrine—Weight change, peripheral edema, abnormal lactation, gynecomastia, menstrual irregularities, false results on pregnancy tests, impotency in men and increased libido in women have occurred in some patients on phenothiazine therapy.

Allergic Reactions—Itching, erythema, urticaria, seborrhea, photosensitivity, eczema and exfoliative dermatitis have been reported with phenothiazines. The possibility of anaphylactoid reactions should be borne in mind.

Hematologic—Blood dyscrasias including leukopenia, agranulocytosis, thrombocytopenic or nonthrombocytopenic purpura, eosinophilia, and pancytopenia have been observed with phenothiazines. If soreness of the mouth, gums or throat or any symptoms of upper respiratory infection occur and confirmatory leukocyte count indicates cellular depression, therapy should be discontinued and other appropriate measures instituted immediately.

Hepatic—Liver damage manifested by cholestatic jaundice, particularly during the first months of therapy, may occur; treatment should be discontinued. A cephalin flocculation increase, sometimes accompanied by alterations in other liver function tests, has been reported in patients who have had no clinical evidence of liver damage.

Others—Sudden deaths have been reported in hospitalized patients on phenothiazines. Previous brain damage or seizures may be predisposing factors. High doses should be avoided in known seizure patients. Shortly before death, several patients showed flare-ups of psychotic behavior patterns. Autopsy findings have usually revealed acute fulminating pneumonia or pneumonitis, aspiration of gastric contents, or intramyocardial lesions. Although not a general feature of fluphenazine, potentiation of central nervous system depressants such as opiates, analgesics, antihistamines, barbiturates, and alcohol may occur.

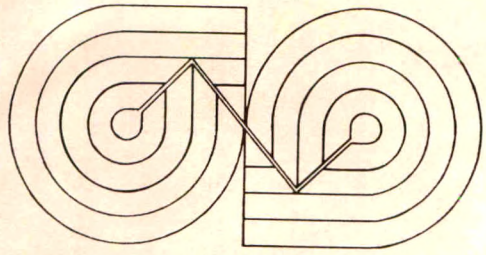
Systemic lupus erythematosus-like syndrome, hypotension severe enough to cause fatal cardiac arrest, altered electrocardiographic and electroencephalographic tracings, altered cerebrospinal fluid proteins, cerebral edema, asthma, laryngeal edema, and angioneurotic edema; with long-term use, skin pigmentation and lenticular and corneal opacities have occurred with phenothiazines. Local tissue reactions occur only rarely with injections of fluphenazine decanoate.

For full prescribing information, consult package insert.

HOW SUPPLIED: 1 ml Unimatic® single-dose preassembled syringes and cartridge units and 5 ml vials.

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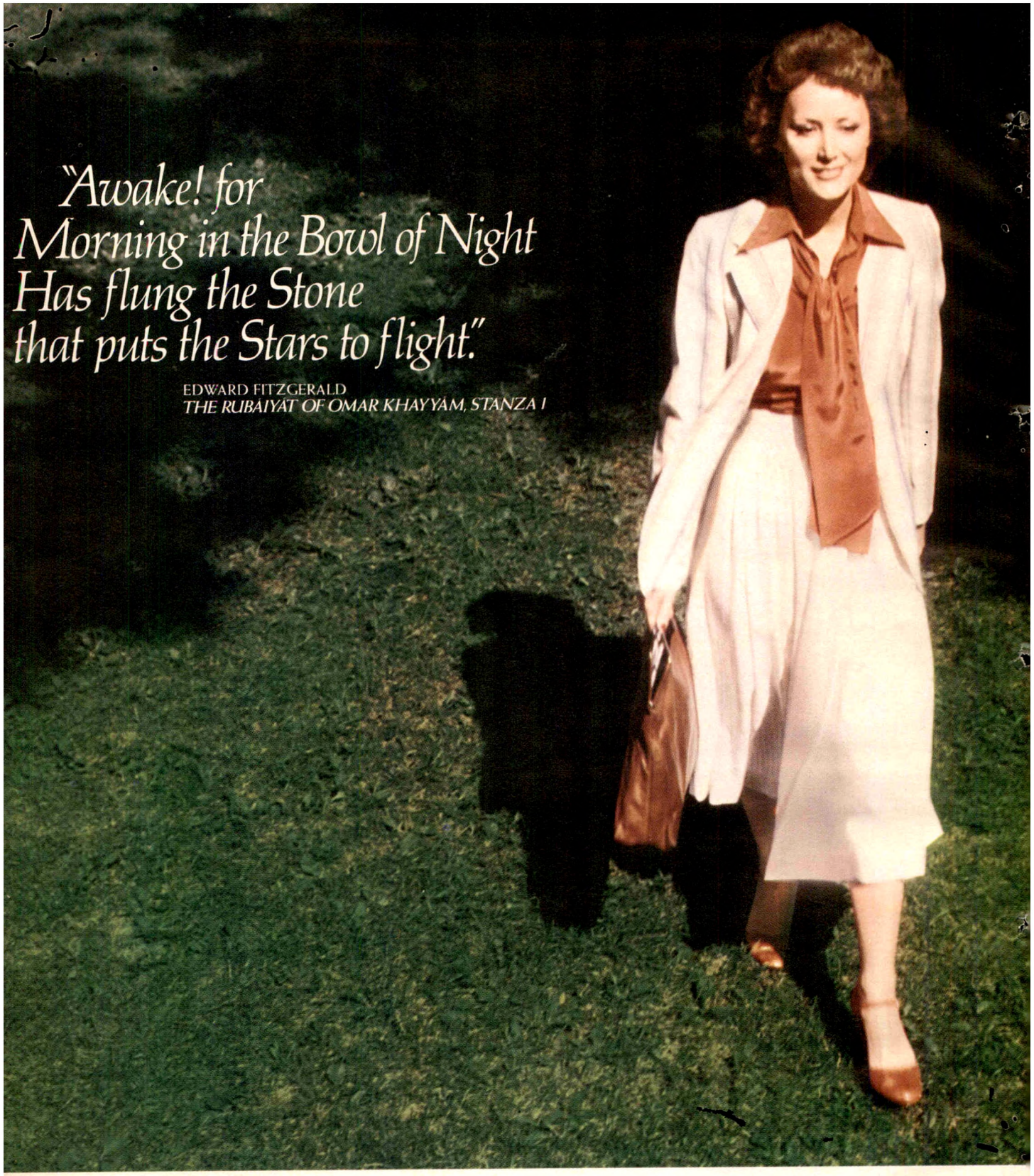
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☐ Psychiatric Resident-In-Training, APA MEMBER	$20.00
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Brief Summary of Prescribing Information

ADAPIN® (doxepin HCl) Capsules

Indications—Relief of symptoms of anxiety and depression.

Contraindications—Glaucoma, tendency toward urinary retention, or hypersensitivity to doxepin.

Warnings—Adapin has not been evaluated for safety in pregnancy. No evidence of harm to the animal fetus has been shown in reproductive studies. There are no data concerning secretion in human milk, nor on effect in nursing infants.

Usage in children under 12 years of age is not recommended. MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with this drug, as serious side-effects and death have been reported with the concomitant use of certain drugs and MAO inhibitors.

In patients who may use alcohol excessively potentiation may increase the danger inherent in any suicide attempt or overdosage.

Precautions—Drowsiness may occur and patients should be cautioned against driving a motor vehicle or operating hazardous machinery. Since suicide is an inherent risk in depressed patients they should be closely supervised while receiving treatment. Although Adapin has shown effective tranquilizing activity, the possibility of activating or unmasking latent psychotic symptoms should be kept in mind.

Adverse Reactions—Dry mouth, blurred vision and constipation have been reported. Drowsiness has also been observed.

Adverse effects occurring infrequently include extrapyramidal symptoms, gastrointestinal reactions, secretory effects such as sweating, tachycardia and hypotension. Weakness, dizziness, fatigue, weight gain, edema, paresthesias, flushing, chills, tinnitus, photophobia, decreased libido, rash and pruritus may also occur.

Dosage and Administration—In mild to moderate anxiety and/or depression: 25 mg t.i.d. Increase or decrease the dosage according to individual response. Daily dosage, up to 150 mg may be taken at bedtime without loss of effectiveness. Usual optimum daily dosage is 75 mg to 150 mg per day not to exceed 300 mg per day.

Antianxiety effect usually precedes the antidepressant effect by two or three weeks.

How Supplied—Each capsule contains doxepin, as the hydrochloride: **10 mg, 25 mg, 50 mg, and 100 mg** capsules in bottles of 100 and 1000.

For complete prescribing information please see package insert or PDR.



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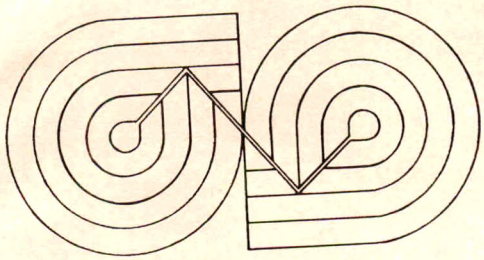
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Antianxiety effect usually precedes the antidepressant effect by two or three weeks.

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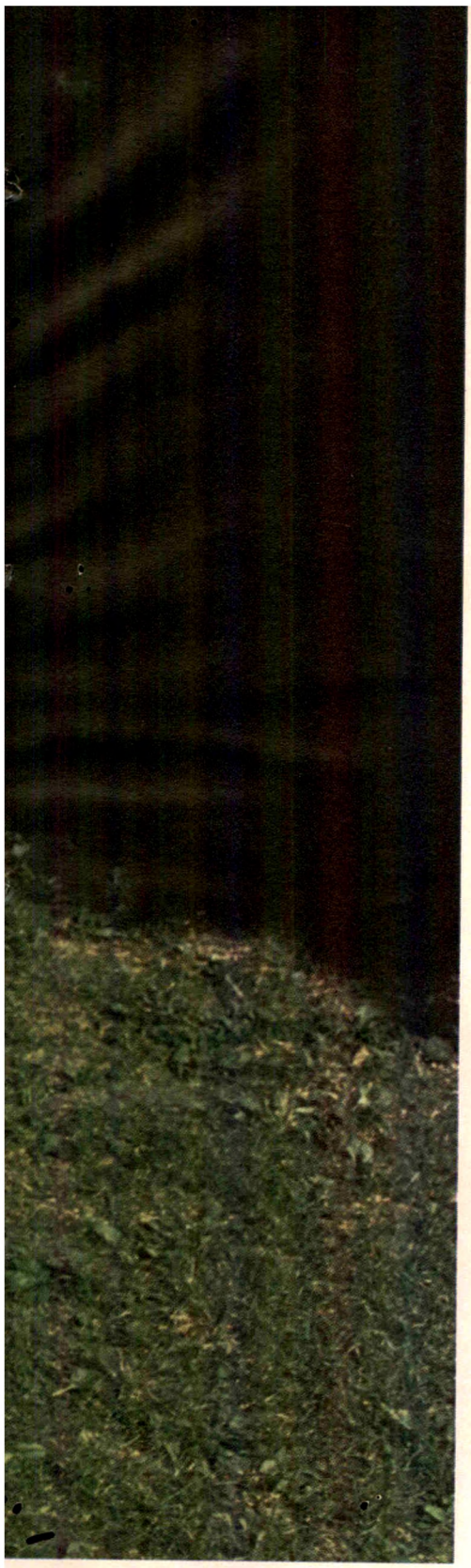
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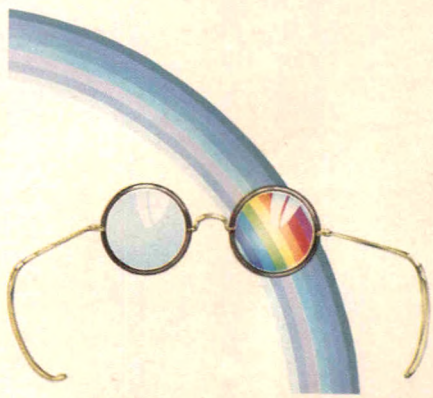
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Most depressed patients experience some form of sleep disturbance. Adapin® helps relieve the accompanying anxiety, and restores a more normal sleep pattern right from the start of therapy.

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The usual optimal dose is 75 to 150 mg daily. The entire dose may be given at night without loss of therapeutic effect. Adapin is available as 10 mg, 25 mg, 50 mg, and 100 mg capsules.

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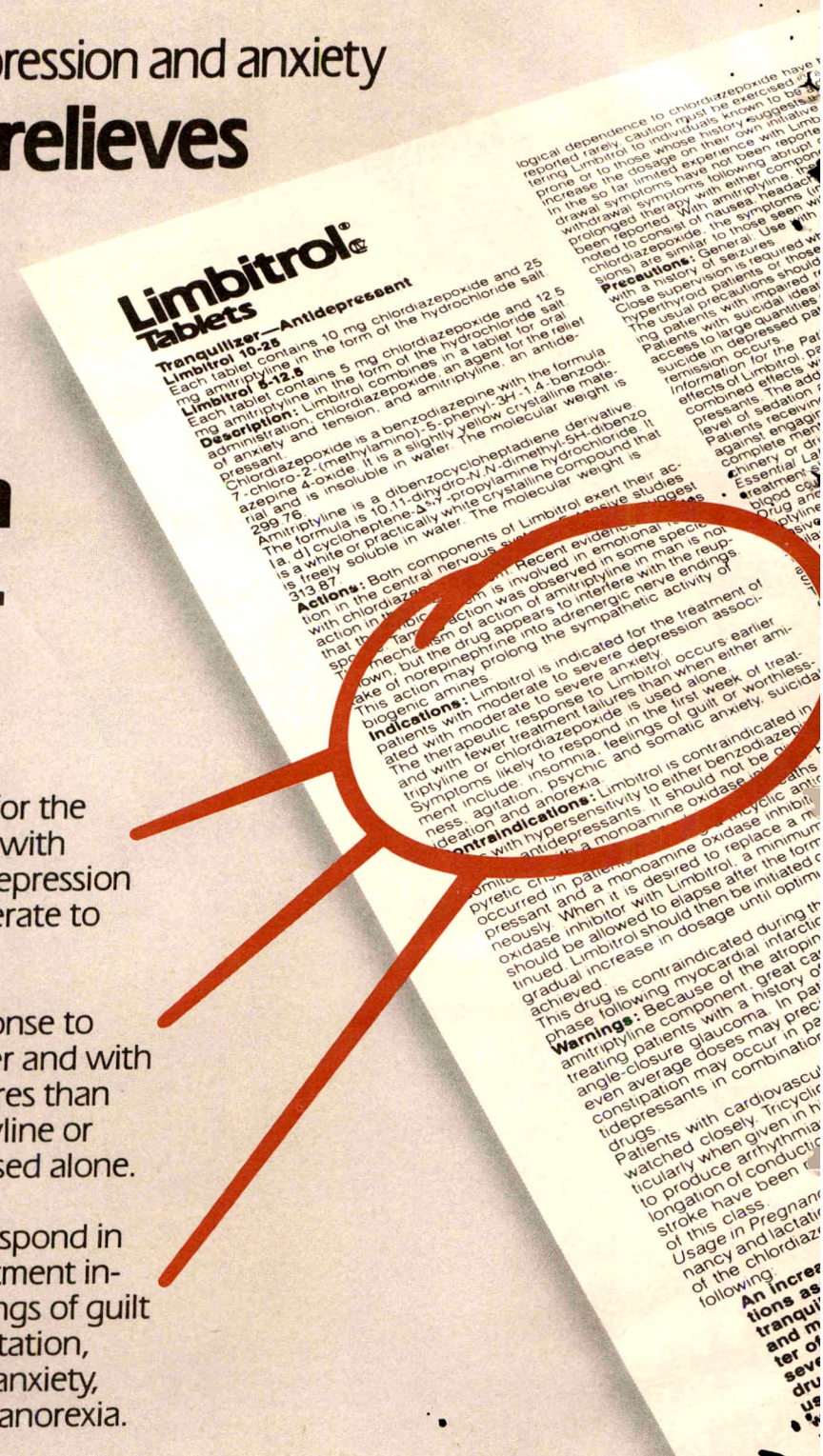
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In moderate depression and anxiety

Only Limbitrol relieves this range of symptoms this rapidly.

And without a phenothiazine.


Limbitrol®
Tablets

Tranquilizer—Antidepressant

Limbitrol 10-25
Each tablet contains 10 mg chlordiazepoxide and 25 mg amitriptyline in the form of the hydrochloride salt.

Limbitrol 5-12.5
Each tablet contains 5 mg chlordiazepoxide and 12.5 mg amitriptyline in the form of the hydrochloride salt.

Description: Limbitrol combines in a tablet for oral administration, chlordiazepoxide, an agent for the relief of anxiety and tension, and amitriptyline, an antidepressant.

Chlordiazepoxide is a benzodiazepine with the formula 7-chloro-2-(methylamino)-5-phenyl-3H-1,4-benzodiazepine 4-oxide. It is a slightly yellow crystalline material and is insoluble in water. The molecular weight is 299.76.

Amitriptyline is a dibenzocycloheptadiene derivative. The formula is 10,11-dihydro-N,N-dimethyl-5H-dibenzo [a, d] cycloheptene-Δ5,γ-propylamine hydrochloride. It is a white or practically white crystalline compound that is freely soluble in water. The molecular weight is 313.87.

Actions: Both components of Limbitrol exert their action in the central nervous system. Recent evidence suggests that the mechanism of action was observed in emotional states. This action may prolong the sympathetic activity of biogenic amines.

Indications: Limbitrol is indicated for the treatment of patients with moderate to severe depression associated with moderate to severe anxiety.

The therapeutic response to Limbitrol occurs earlier and with fewer treatment failures than when either amitriptyline or chlordiazepoxide is used alone.

Symptoms likely to respond in the first week of treatment include: insomnia, feelings of guilt or worthlessness, agitation, psychic and somatic anxiety, suicidal ideation and anorexia.

Contraindications: Limbitrol is contraindicated in patients with hypersensitivity to either benzodiazepines or tricyclic antidepressants. It should not be given in the presence of a monoamine oxidase inhibitor.

Pyretic crises, reactions resembling acute schizophrenic episodes, and convulsions have occurred in patients receiving amitriptyline alone or in combination with other drugs, including tricyclic antidepressant and a monoamine oxidase inhibitor. When it is desired to replace a monoamine oxidase inhibitor with Limbitrol, a minimum of 14 days should be allowed to elapse after the former is discontinued. Limbitrol should then be initiated at a low dosage and gradual increase in dosage until optimum response is achieved.

This drug is contraindicated during the acute recovery phase following myocardial infarction. **Warnings:** Because of the atropine-like effects of amitriptyline component, great caution should be used in treating patients with a history of urinary retention, angle-closure glaucoma. In patients receiving even average doses may occur in paralytic ileus. Constipation may occur in patients receiving tricyclic antidepressants in combination with drugs.

Patients with cardiovascular disease should be watched closely. Tricyclic antidepressants particularly when given in high doses, have been known to produce arrhythmias, sinus tachycardia, prolongation of conduction time, myocardial infarction, and stroke have been reported with the use of this class.

Usage in Pregnancy and lactation: Safe use of the chlordiazepoxide during pregnancy has not been established. Therefore, the following:

An increased incidence of congenital malformations associated with the use of minor tranquilizers and meprobamate during pregnancy has been reported in several studies. Barbiturates, chlordiazepoxide, and meprobamate have been shown to be teratogenic in animals. Limbitrol should not be used during pregnancy, especially during the first trimester of pregnancy, unless in the opinion of the physician the potential benefits outweigh the possible hazards.

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Limbitrol.
Dual therapy with greater specificity...without classical phenothiazine drawbacks.

osage and recommendations for treatment are based on clinical experience with its components. Primary concern should be with the dangers associated with amitriptyline overdosage. Deaths by deliberate or accidental overdosage have occurred with this class of drugs.

All patients suspected of having an overdosage of Limbitrol should be admitted to a hospital as soon as possible.

Manifestations: High doses may cause drowsiness, temporary confusion, disturbed concentration or transient visual hallucinations. Overdosage may cause hypothermia, tachycardia and conduction disturbances (evidence of impaired conduction such as bundle branch block), congestive heart failure, dilated pupils, convulsions, severe hypotension, stupor and coma. Other symptoms may be agitation, hyperpyrexia or any of those muscle rigidity, vomiting, hyperactive reflexes, listed under Adverse Reactions.

Treatment: Empty the stomach as quickly as possible by emesis or lavage. In the comatose patient a cuff endotracheal tube should be placed in position prior to either of these measures. The stomach also should be considered. If charcoal into the stomach and nursing care may be required. It the patient is stuporous but responds to stimuli, only close observation and nursing care may be required. It essential to maintain an adequate airway and fluid intake. Body temperature should be watched closely and appropriate measures taken should deviations occur.

The intramuscular or slow intravenous administration of 1 to 3 mg in adults (or 0.5 mg in children) of physostigmine salicylate (Antilirium®) has been reported to reverse the manifestations of amitriptyline overdosage. Because of its relatively short half-life, additional doses may be needed at intervals of 30 minutes to 2 hours. Convulsions may be treated by the use of barbiturates. Cardiac anesthetic rather than the use of an inhalation or other antiarrhythmic agents should be considered if monitoring is advisable, and the cautious use of cardiac serious cardiovascular abnormalities occur. Serum potassium levels should be monitored and kept within normal limits by the use of appropriate I.V. fluids. Standard measures including oxygen, I.V. fluids, plasma expanders and corticosteroids may be used to control circulatory shock.

Dialysis is unlikely to be of value, as it has not proven useful in overdosages of either amitriptyline or chlordiazepoxide. Since many suicidal attempts involve multiple drugs including barbiturates, the possibility of dialysis being beneficial for removal of other drugs should not be overlooked.

Treatment should be continued for at least 48 hours, along with cardiac monitoring in patients who do not respond to therapy promptly. Since relapses are frequent, patients should be hospitalized until their conditions remain stable without physostigmine for at least 24 hours.

Since overdosage is often deliberate, patients may attempt suicide by other means during the recovery phase.

References:

1. Granacher RP, Baldessarini RJ: Physostigmine. Its use in acute anticholinergic syndrome with antidepressant and antiparkinson drugs. Arch Gen Psychiatry 32:375-380, Mar 1975.
2. Burks JS, Walker JE, Rumack BH, Ott JE: Tricyclic antidepressant poisoning. Reversal of coma, choreoathetosis, and myoclonus by physostigmine. JAMA 230:1405-1407, Dec 9, 1974.
3. Snyder BD, Blonde L, McWhirter WR: Reversal of amitriptyline intoxication by physostigmine. JAMA 230:1433-1434, Dec 9, 1974.

Dosage and Administration: Optimum dosage varies with the severity of the symptoms and the response of the individual patient. When a satisfactory response is obtained, dosage should be reduced to the smallest amount needed to maintain the remission. The larger portion of the total daily dose may be taken at bedtime. In some patients, a single dose at bedtime may be sufficient. In general, lower dosages are recommended for elderly patients.

Limbitrol 10-25 is recommended in an initial dosage of three or four tablets daily in divided doses; this may be increased to six tablets daily as required. Some patients respond to smaller doses and can be maintained on two tablets daily.

Limbitrol 5-12.5 in an initial dosage of three or four tablets daily in divided doses may be satisfactory in patients who do not tolerate higher doses.

How Supplied: White, film-coated tablets, each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt) and blue, film-coated tablets, each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 50.

THIS PRODUCT INFORMATION ISSUED OCTOBER 1978.

ROCHE LABORATORIES
Division of Hoffmann-La Roche Inc.
Nutley, New Jersey 07110

Limbitrol®

Tablets 10-25 each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt)

Tablets 5-12.5 each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)

Please see summary of product information on last page of this advertisement.

LIMBITROL® TABLETS Tranquilizer—Antidepressant

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Relief of moderate to severe depression associated with moderate to severe anxiety.

Contraindications: Known hypersensitivity to benzodiazepines or tricyclic antidepressants. Do not use with monoamine oxidase (MAO) inhibitors or within 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use; then initiate cautiously, gradually increasing dosage until optimal response is achieved. Contraindicated during acute recovery phase following myocardial infarction.

Warnings: Use with great care in patients with history of urinary retention or angle-closure glaucoma. Severe constipation may occur in patients taking tricyclic antidepressants and anticholinergic-type drugs. Closely supervise cardiovascular patients. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, especially high doses. Myocardial infarction and stroke reported with use of this class of drugs.)

***Usage in Pregnancy:* Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Since physical and psychological dependence to chlordiazepoxide have been reported rarely, use caution in administering Limbitrol to addiction-prone individuals or those who might increase dosage; withdrawal symptoms following discontinuation of either component alone have been reported (nausea, headache and malaise for amitriptyline; symptoms [including convulsions] similar to those of barbiturate withdrawal for chlordiazepoxide).

Precautions: Use with caution in patients with a history of seizures, in hyperthyroid patients or those on thyroid medication, and in patients with impaired renal or hepatic function. Because of the possibility of suicide in depressed patients, do not permit easy access to large quantities in these patients. Caution patients about possible combined effects with alcohol and other CNS depressants and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Periodic liver function tests and blood counts are recommended during prolonged treatment. Amitriptyline component may block action of guanethidine or similar antihypertensives. Concomitant use with other psychotropic drugs has not been evaluated: sedative effects may be additive. Discontinue several days before surgery. Limit concomitant administration of ECT to essential treatment. See Warnings for precautions about pregnancy. Limbitrol should not be taken during the nursing period. Not recommended in children under 12.

In the elderly and debilitated, limit to smallest effective dosage to preclude ataxia, oversedation, confusion or anticholinergic effects.

Adverse Reactions: Most frequently reported are those associated with either component alone: drowsiness, dry mouth, constipation, blurred vision, dizziness and bloating. Less frequently occurring reactions include vivid dreams, impotence, tremor, confusion and nasal congestion. Many depressive symptoms including anorexia, fatigue, weakness, restlessness and lethargy have been reported as side effects of both Limbitrol and amitriptyline. Granulocytopenia, jaundice and hepatic dysfunction have been observed rarely.

This list includes adverse reactions not reported with Limbitrol but requiring consideration because they have been reported with one or both components or closely related drugs:

Cardiovascular: Hypotension, hypertension, tachycardia, palpitations, myocardial infarction, arrhythmias, heart block, stroke.

Psychiatric: Euphoria, apprehension, poor concentration, delusions, hallucinations, hypomania and increased or decreased libido.

Neurologic: Incoordination, ataxia, numbness, tingling and paresthesias of the extremities, extrapyramidal symptoms, syncope, changes in EEG patterns.

Anticholinergic: Disturbance of accommodation, paralytic ileus, urinary retention, dilatation of urinary tract.

Allergic: Skin rash, urticaria, photosensitization, edema of face and tongue, pruritus.

Hematologic: Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia.

Gastrointestinal: Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, black tongue.

Endocrine: Testicular swelling and gynecomastia in the male, breast enlargement, galactorrhea and minor menstrual irregularities in the female and elevation and lowering of blood sugar levels.

Other: Headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, jaundice, alopecia, parotid swelling.

Overdosage: Immediately hospitalize patient suspected of having taken an overdose. Treatment is symptomatic and supportive. I.V. administration of 1 to 3 mg physostigmine salicylate has been reported to reverse the symptoms of amitriptyline poisoning. See complete product information for manifestation and treatment.

Dosage: Individualize according to symptom severity and patient response. Reduce to smallest effective dosage when satisfactory response is obtained. Larger portion of daily dose may be taken at bedtime. Single *h.s.* dose may suffice for some patients. Lower dosages are recommended for the elderly.

Limbitrol 10-25, initial dosage of three to four tablets daily in divided doses, increased up to six tablets or decreased to two tablets daily as required. Limbitrol 5-12.5, initial dosage of three to four tablets daily in divided doses, for patients who do not tolerate higher doses.

Supplied: Limbitrol 10-25 tablets each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt) and Limbitrol 5-12.5 tablets each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)—bottles of 100 and 500; Tel-E-Dose* packages of 100; Prescription Paks of 50.

ROCHE

Roche Laboratories
Division of Hoffmann-La Roche Inc.
Nutley, New Jersey 07110

How to initiate and maintain therapy

Select dosage strength appropriate for each patient

☐ Limbitrol 10-25 is recommended for most patients

☐ Limbitrol 5-12.5 may be indicated for those who do not tolerate higher doses and for elderly patients

Specify daily dosage based on symptom severity

☐ An initial dosage of three tablets is recommended.

☐ Dosage may be increased to six tablets or decreased to two tablets daily as necessary.

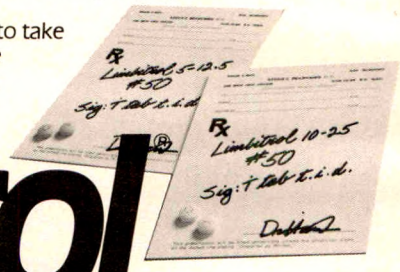
☐ Once a satisfactory response is obtained, evaluate response and, if necessary, reduce dosage to smallest amount needed.

Utilize dosage options to best accommodate individual patient needs

☐ T.I.D. or Q.I.D., familiar regimens most suited for patients who tolerate medication without undue drowsiness

☐ Two tablets one hour before bedtime and one tablet midday may minimize daytime drowsiness and help relieve a common target symptom—insomnia

☐ Entire dosage h.s. to take maximum advantage of the sedative effect



Limbitrol

Tablets 10-25 each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt)

Tablets 5-12.5 each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)

How to make each patient an informed patient

1. Discuss with patients the probability that they will experience drowsiness, especially during the first week.

2. Reassure your patients that drowsiness is one indication that the medication is working and that it may help alleviate their insomnia.

3. Encourage patients to report if drowsiness becomes troublesome so that, if necessary, dosage schedule can be adjusted or the symptom treated.

4. Caution patients about the combined effects with alcohol or other CNS depressants. Let them know that the additive effects may produce a harmful level of sedation and CNS depression.

5. Caution patients about activities requiring complete mental alertness, such as operating machinery or driving a car.

6. Warn pregnant patients and patients of childbearing age that the safety of Limbitrol in pregnancy has not yet been established.

Please see complete product disclosure for other pertinent information.

Limbitrol should not be used under the following circumstances:

1. Hypersensitivity to benzodiazepines or tricyclic antidepressants.
2. Should not be given with an MAO inhibitor. To replace an MAO inhibitor with Limbitrol, discontinue MAO inhibitor for a minimum of 14 days before cautiously initiating Limbitrol therapy.
3. During the acute recovery phase following myocardial infarction.

A50

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for institutions and agencies concerned with the care of the mentally disabled

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Hospital & Community Psychiatry is just one of the benefits of membership in the H&CP Service. Others include a film library containing more than a hundred films specially chosen for their usefulness in staff development and community education programs; supplementary mailings of important books, reports, articles, or other material of special interest to administrators or clinicians; reduced registration fees at the annual fall Institute on Hospital & Community Psychiatry; and, on request, information and consultation from the professional staff of the American Psychiatric Association. The H&CP Service also sponsors the annual Achievement Awards competition, which gives special recognition to outstanding programs for the mentally ill and mentally retarded.

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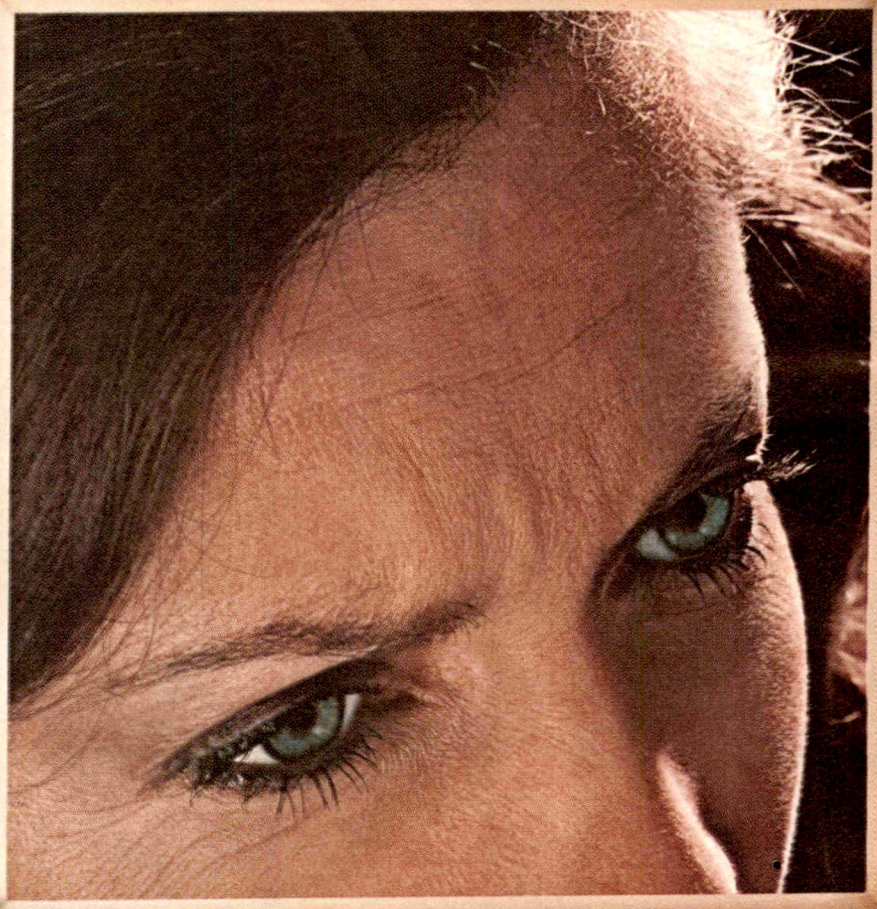
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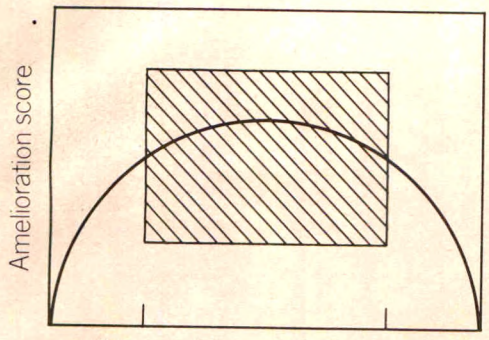
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A51

The therapeutic window helps you see the difference between Pamelor® (nortriptyline HCl) and other antidepressants.





The Pamelor Therapeutic Window: a well-defined range of therapeutic blood levels.

All tricyclics have a range of steady-state plasma levels that produce maximal antidepressant effects and minimal toxic effects.[1] When this range is well-defined it is called the "therapeutic window." To date, conclusive evidence for a therapeutic window has been obtained for only one tricyclic—nortriptyline.[2]

Highly predictable determination of optimal dosage

The more accurately the limits of the therapeutic window are defined, the more predictably individual dosage requirements for a tricyclic agent can be determined.[2] That's why an initial starting dose of 75 mg Pamelor daily can achieve optimal plasma levels for 70% of depressed patients.[3] A single dosage adjustment to 100 mg Pamelor daily will achieve therapeutic blood levels for most patients who have not responded to 75 mg.

Minimal titration

With other tricyclic agents, the therapeutic dose is usually determined by increasing the daily dose from 25 mg upward to 300 mg. This time-consuming and often random process of titration is virtually eliminated with Pamelor. Because the initial dose is usually a therapeutic dose, you can spend less time on dosage adjustment and more time on psychotherapy.

Minimal significant side effects

Pamelor therapy is well tolerated. Daytime drowsiness is rarely a problem. As with all antidepressants, however, patients should be cautioned against driving or operating hazardous machinery. Anticholinergic side effects are also minimal, although some patients may experience dry mouth.

Pamelor therapy is clinically effective

Improvement of symptoms of depression can often begin to be seen in some patients within a week. These include relief of depressed mood, sleep disturbances, and fatigue. Global improvement is usually observed by the second week. Maximum improvement with Pamelor, as with other antidepressants, may require longer therapy particularly in severe depressive illnesses.



Pamelor®
(nortriptyline HCl) NF
25mg. Capsules

Helps make the therapeutic dosage easy to determine.

For brief summary please see last page of this advertisement.

SANDOZ
PHARMACEUTICALS
East Hanover, N.J. 07936



Pamelor® (nortriptyline HCl) NF 25mg. Capsules

Helps make the therapeutic dosage easy to determine for depressed patients.

- ☐ highly predictable determination of optimal dosage
- ☐ minimal titration
- ☐ minimal significant side effects
- ☐ Pamelor therapy is clinically effective

Indications: For relief of depressive symptoms. Endogenous depressions are more likely to be alleviated than others.

Contraindications: Hypersensitivity. Should not be given concomitantly with MAO inhibitors or within 2 weeks of the use of this drug since hyperpyretic crises, severe convulsions, and fatalities have occurred when similar tricyclic antidepressants were used in such combinations. Cross-sensitivity with other dibenzazepines is a possibility. Contraindicated during acute recovery period after myocardial infarction.

Warnings: Use with caution in patients with cardiovascular disease because of tendency to produce sinus tachycardia and prolong conduction time. Myocardial infarction, arrhythmia, and strokes have occurred. May block antihypertensive action of guanethidine and similar agents. Because of anticholinergic activity, use cautiously in patients with glaucoma or a history of urinary retention. Patients with a history of seizures should be followed closely because the drug is known to lower the convulsive threshold. Great care is required for hyperthyroid patients and those taking thyroid medication because of possible development of cardiac arrhythmia. Caution patients about possibility of impaired mental and/or physical ability to operate a motor vehicle or dangerous machinery. Response to alcoholic beverages may be exaggerated and may lead to suicidal attempts. Safe use during pregnancy, lactation, and women of childbearing potential has not been established and the drug should not be given unless clinical situation warrants potential risk. Not recommended for use in children.

Precautions: Psychotic symptoms may be exacerbated in schizophrenic patients. Increased anxiety and agitation may occur in overactive or agitated patients. Manic-depressive patients may experience shift to manic phase. Hostility may be aroused. Concomitant administration of reserpine may produce a "stimulating" effect. Watch for possible epileptiform seizures during treatment. Use cautiously with anticholinergic or sympathomimetic drugs. Concurrent electroconvulsive therapy may increase hazards associated with nortriptyline HCl. When possible, discontinue drug several days prior to surgery. Potentially suicidal patients require supervision and protective measures during therapy. Prescriptions should be limited to the least possible quantity. Both elevation and lowering of blood sugar levels have been reported.

Adverse Reactions: Note: The pharmacologic similarities among the tricyclic antidepressant drugs require that each of the following reactions be considered when nortriptyline is administered.

Cardiovascular: Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke.

Psychiatric: Confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia, panic, nightmares; hypomania; exacerbation of psychosis.

Neurologic: Numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures, alteration in EEG patterns; tinnitus.

Anticholinergic: Dry mouth and rarely, associated sublingual adenitis; blurred vision, disturbance of accommodation, mydriasis; constipation, paralytic ileus; urinary retention, delayed micturition, dilation of the urinary tract.

Allergic: Skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight); edema (general or of face and tongue), drug fever, cross-sensitivity with other tricyclic drugs.

Hematologic: Bone-marrow depression, including agranulocytosis; eosinophilia; purpura; thrombocytopenia.

Gastrointestinal: Nausea and vomiting, anorexia, epigastric distress, diarrhea, peculiar taste, stomatitis, abdominal cramps, black-tongue.

Endocrine: Gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, impotence, testicular swelling; elevation or depression of blood sugar levels.

Other: Jaundice (simulating obstructive); altered liver function; weight gain or loss; perspiration; flushing; urinary frequency, nocturia; drowsiness, dizziness, weakness, fatigue; headache; parotid swelling; alopecia.

Withdrawal Symptoms: Though these are not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache, and malaise.

Dosage and Administration: *Usual adult dose*—25 mg. three or four times daily; dosage should begin at a low level and increase as required. As an alternate regimen, the total daily dosage may be given once-a-day. *Elderly and Adolescent*—30 to 50 mg. per day, in divided doses, or the total dosage may be given once-a-day. Doses above 100 mg. per day and use in children are not recommended. If a patient develops minor side effects, the dosage should be reduced. The drug should be discontinued promptly if adverse effects of a serious nature or allergic manifestations occur.

How Supplied: Capsules 10 mg. and 25 mg.; solution 10 mg./5 cc.

For more detailed information see full prescribing information.

References:

1. Åsberg M, Crönholm B, Sjöqvist F, et al: Relationship between plasma level and therapeutic effect of nortriptyline. *Br Med J* 3:331-334, 1971.
2. Ziegler VE: Improved clinical response to tricyclic antidepressants: Provisions for faster, more effective, and safer therapy. Presented as a scientific exhibit at the 30th Institute on Hospital & Community Psychiatry of the American Psychiatric Association, Kansas City, Mo, September 18-20, 1978. p 9.
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The American Psychiatric Association announces publication of two volumes reporting the Conference on Education of Psychiatrists

held at Lake of the Ozarks, Missouri, June 9-15, 1975. Organized by the A.P.A. in cooperation with the American Academy of Child Psychiatry, the American Association of Chairmen of Departments of Psychiatry, and the American Association of Directors of Psychiatric Residency Training.

1. PSYCHIATRIC EDUCATION: PROLOGUE TO THE 1980's, Library Bound, 544 pages, $15.00.

This is a substantive, highly readable, report of the Conference, written by Anne H. Rosenfeld with an Editorial Board comprising: Ewald W. Busse, M.D., Chairperson of the Conference, Walter E. Barton, M.D., Bernard Holland, M.D., Donna Norris, M.D., Melvin Sabshin, M.D., Jeanne Spurlock, M.D., and Robert L. Robinson, M.A. (Staff Consultant).

The volume contains ten chapters covering the topical structure of the Conference: Goals and Values; Competence, Evaluation, and Quality Control; The Resident; Faculty; Educational Methods: Means in the Service of Ends; The Program Environment; Research; The Economics of Residency Training; Psychiatry in Its Social Context; and Retrospect and Prospect. It also contains nine appendices describing the organization of the Conference and its participants, together with selected model programs and related informational materials of value.

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Papers commenting on the reports of the commissions, published here for the first time, were presented at the Conference by Melvin Sabshin, M.D., Eugene B. Brody, M.D., Jeanne Spurlock, M.D., Jack R. Ewalt, M.D., Robert J. Stoller, M.D., James R. Comer, M.D., and Bernard Holland, M.D.

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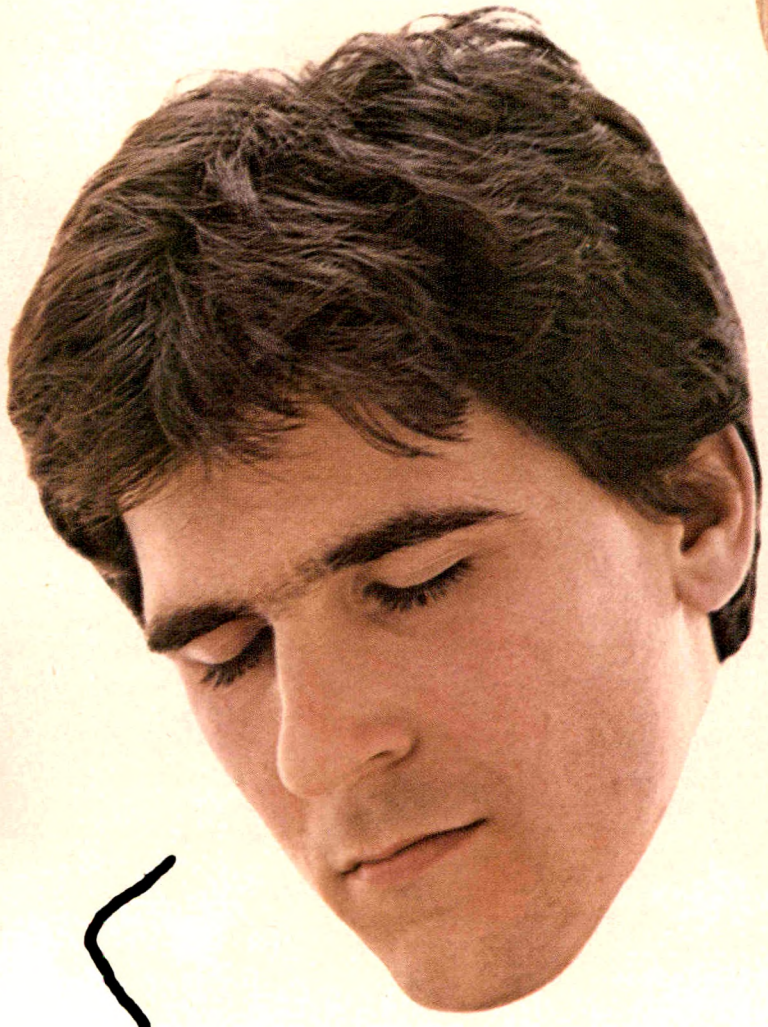
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THE MANY FACES



Help for the apathetic/ withdrawn geriatric*

For the aged person suffering from withdrawn or apathetic senile behavior, Ritalin can often help relieve bothersome symptoms. In one double-blind study[1] of withdrawn, apathetic geriatric patients, Ritalin elicited notable improvement in competence, interest and retardation. While no side effects were reported in this study, blood pressure and pulse variations were noted in both groups but were not considered of exceptional magnitude.



Help for narcoleptic patients

The narcoleptic patient can fall asleep anytime...anyplace... and the consequences can be grave, or at the least, embarrassing. Although there is no known cure for narcolepsy, Ritalin acts promptly to counteract sleepiness and is the medication most frequently used by cited investigators.[2,3] Occasional problems of tolerance may be managed by complete withdrawal of Ritalin during vacation or holidays, then reinstating the drug at lower dosage.[3]

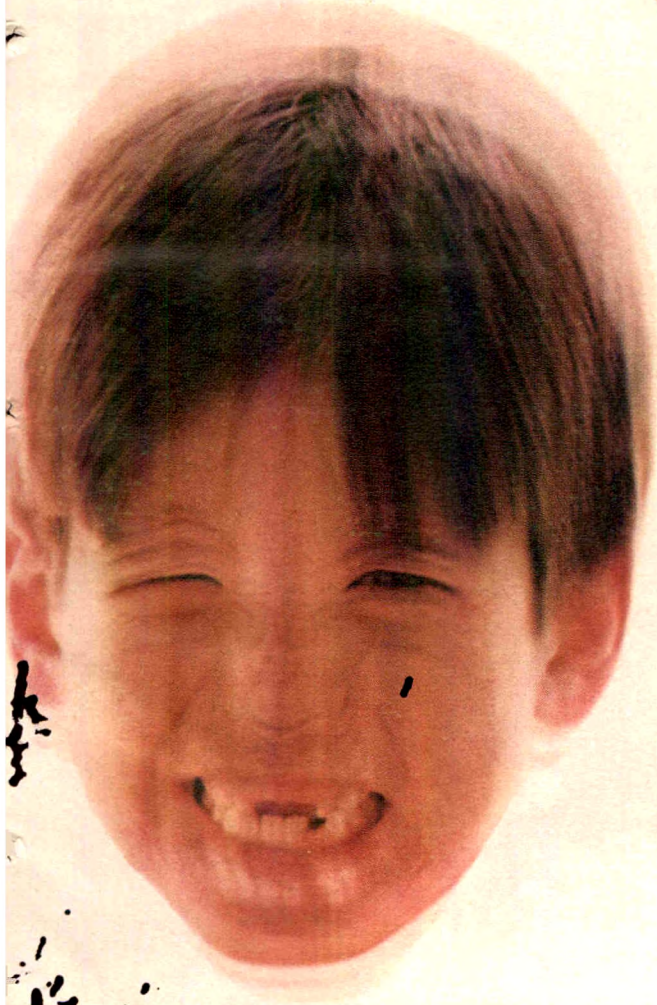
*Ritalin has been evaluated as possibly effective in these indications.

OF RITALIN® CII
(methylphenidate)



Help for patients with mild depression*

As Ritalin acts promptly to relieve symptoms of mild depression, mood and outlook usually improve, and the patient can cope again. Generally, one prescription is sufficient. Ritalin is contraindicated in severe depression.

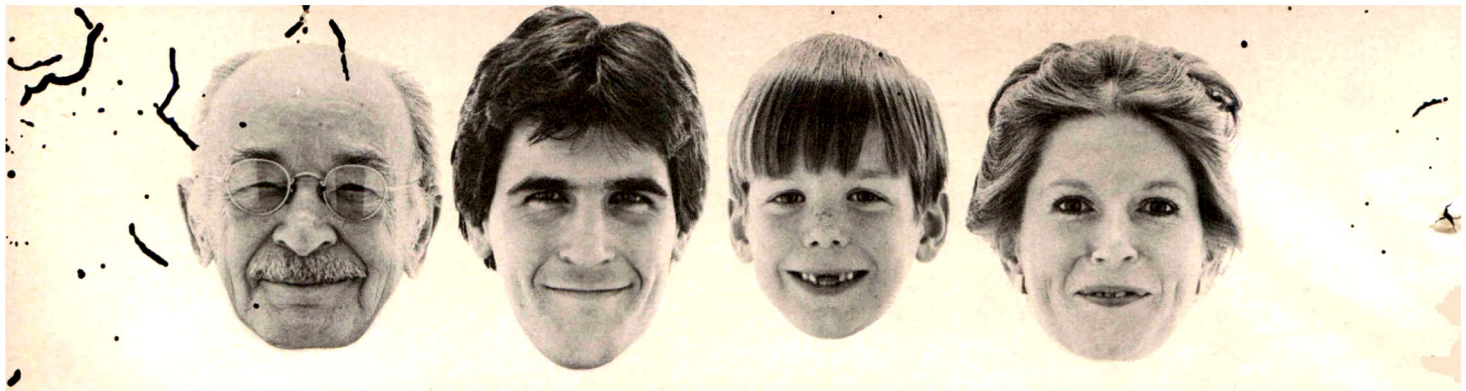


Help for the MBD child

For the child with Minimal Brain Dysfunction (MBD), adjunctive Ritalin is often highly effective when medication is needed to help reduce symptoms such as distractibility,[4] disorganized behavior,[4] and hyperactivity.[4] MBD children responding to Ritalin usually exhibit better classroom performance and improved interpersonal relationships. Moreover, as MBD symptoms are curbed, the affected child often responds more positively to nonpharmacologic modalities.[5] Ritalin should be periodically discontinued to assess the child's condition. Improvement may be sustained when the drug is temporarily or permanently discontinued.

Please turn page for prescribing information.

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RITALIN® Ⓒ
(methylphenidate)
SO MUCH HELP FOR SO MANY PATIENTS

Ritalin® hydrochloride Ⓒ
(methylphenidate hydrochloride USP)

TABLETS

INDICATIONS
Based on a review of this drug by the National Academy of Sciences-National Research Council and/or other information, FDA has classified the indications as follows:
Effective: Minimal Brain Dysfunction in Children—as adjunctive therapy to other remedial measures (psychological, educational, social)
Special Diagnostic Considerations
Specific etiology of Minimal Brain Dysfunction (MBD) is unknown, and there is no single diagnostic test. Adequate diagnosis requires the use not only of medical but of special psychological, educational, and social resources.
Characteristics commonly reported include: chronic history of short attention span, distractibility, emotional lability, impulsivity, and moderate to severe hyperactivity; minor neurological signs and abnormal EEG. Learning may or may not be impaired. The diagnosis of MBD must be based upon a complete history and evaluation of the child and not solely on the presence of one or more of these characteristics.
Drug treatment is not indicated for all children with MBD. Stimulants are not intended for use in the child who exhibits symptoms secondary to environmental factors and/or primary psychiatric disorders, including psychosis.
Appropriate educational placement is essential and psychosocial intervention is generally necessary. When remedial measures alone are insufficient, the decision to prescribe stimulant medication will depend upon the physician's assessment of the chronicity and severity of the child's symptoms.
Effective: Narcolepsy
"Possibly" effective: Mild Depression; Apathetic or Withdrawn Senile Behavior
Final classification of the less-than-effective indications requires further investigation.

CONTRAINDICATIONS
Marked anxiety, tension, and agitation are contraindications to Ritalin, since the drug may aggravate these symptoms. Ritalin is contraindicated also in patients known to be hypersensitive to the drug and in patients with glaucoma.

WARNINGS
Ritalin should not be used in children under six years, since safety and efficacy in this age group have not been established.
Sufficient data on safety and efficacy of long-term use of Ritalin in children with minimal brain dysfunction are not yet available. Although a causal relationship has not been established, suppression of growth (*ie*, weight gain, and/or height) has been reported with the long-term use of stimulants in children. Therefore, patients requiring long-term therapy should be carefully monitored.
Ritalin should not be used for severe depression of either exogenous or endogenous origin.
Ritalin should not be used for the prevention or treatment of normal fatigue states.

There is some clinical evidence that Ritalin may lower the convulsive threshold in patients with prior history of seizures, with prior EEG abnormalities in absence of seizures, and, very rarely, in absence of history of seizures and no prior EEG evidence of seizures. Safe concomitant use of anticonvulsants and Ritalin has not been established. In the presence of seizures, the drug should be discontinued.
Use cautiously in patients with hypertension. Blood pressure should be monitored at appropriate intervals in all patients taking Ritalin, especially those with hypertension.
Symptoms of visual disturbances have been encountered in rare cases. Difficulties with accommodation and blurring of vision have been reported.

Drug Interactions
Ritalin may decrease the hypotensive effect of guanethidine. Use cautiously with pressor agents and MAO inhibitors.
Human pharmacologic studies have shown that Ritalin may inhibit the metabolism of coumarin anticoagulants, anticonvulsants (phenobarbital, diphenylhydantoin, primidone), phenylbutazone, and tricyclic antidepressants (imipramine, desipramine). Downward dosage adjustments of these drugs may be required when given concomitantly with Ritalin.

Usage in Pregnancy
Adequate animal reproduction studies to establish safe use of Ritalin during pregnancy have not been conducted. Therefore, until more information is available, Ritalin should not be prescribed for women of childbearing age unless, in the opinion of the physician, the potential benefits outweigh the possible risks.

Drug Dependence
Ritalin should be given cautiously to emotionally unstable patients, such as those with a history of drug dependence or alcoholism, because such patients may increase dosage on their own initiative.
Chronically abusive use can lead to marked tolerance and psychic dependence with varying degrees of abnormal behavior. Frank psychotic episodes can occur, especially with parenteral abuse. Careful supervision is required during drug withdrawal, since severe depression as well as the effects of chronic overactivity can be unmasked. Long-term follow-up may be required because of the patient's basic personality disturbances.

PRECAUTIONS
Patients with an element of agitation may react adversely; discontinue therapy if necessary.
Periodic CBC, differential, and platelet counts are advised during prolonged therapy.

ADVERSE REACTIONS
Nervousness and insomnia are the most common adverse reactions but are usually controlled by reducing dosage and omitting the drug in the afternoon or evening. Other reactions include hypersensitivity (including skin rash, urticaria, fever, arthralgia, exfoliative dermatitis, erythema multiforme with histopathological findings of necrotizing vasculitis, and thrombocytopenic purpura); anorexia; nausea; dizziness; palpitations; headache; dyskinesia; drowsiness; blood pressure and pulse changes, both up and down; tachycardia; angina; cardiac arrhythmia; abdominal pain; weight loss during prolonged therapy. Toxic psychosis has been reported.
Although a definite causal relationship has not been established, the following have been reported in patients taking this drug: leukopenia and/or anemia, a few instances of scalp hair loss.
In children, loss of appetite, abdominal pain, weight loss during prolonged therapy, insomnia, and tachycardia may occur more frequently; however, any of the other adverse reactions listed above may also occur.

DOSAGE AND ADMINISTRATION
Adults
Administer orally in divided doses 2 or 3 times daily, preferably 30 to 45 minutes before meals. Dosage will depend upon indication and individual response.
Average dosage is 20 to 30 mg daily. Some patients may require 40 to 60 mg daily. In others, 10 to 15 mg daily will be adequate. Patients who are unable to sleep if medication is taken late in the day should take the last dose before 6 p.m.
Children with Minimal Brain Dysfunction
(6 years and over)
Start with small doses (*eg*, 5 mg before breakfast and lunch) with gradual increments of 5 to 10 mg weekly. Daily dosage above 60 mg is not recommended. If improvement is not observed after appropriate dosage adjustment over a one-month period, the drug should be discontinued.
If paradoxical aggravation of symptoms or other adverse effects occur, reduce dosage, or, if necessary, discontinue the drug.
Ritalin should be periodically discontinued to assess the child's condition. Improvement may be sustained when the drug is either temporarily or permanently discontinued.
Drug treatment should not and need not be indefinite and usually may be discontinued after puberty.

HOW SUPPLIED
Tablets, 20 mg (peach, scored); bottles of 100 and 1000.
Tablets, 10 mg (pale green, scored); bottles of 100, 500, 1000 and Accu-Pak® blister units of 100.
Tablets, 5 mg (pale yellow); bottles of 100, 500 and 1000.

665381 C76-16 Rev. 7/76

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2. Daly DD, Yoss RE: Narcolepsy, in *Handbook of Clinical Neurology: Vol 15, The Epilepsies*, Magnus O, Lorentz De Haas AM (Eds), Amsterdam, North-Holland Publishing Company, 1974, pp 836-853.
3. Dement WC, Carskadon MA, Guilleminault C, Zarcone VP: Narcolepsy: Diagnosis and treatment. Primary Care *3*:609-623, 1976.
4. Knobel M: Psychopharmacology for the hyperkinetic child. Arch Gen Psychiatry *6*:193-202, 1952.
5. Seger EY, Hallum G: Methylphenidate in children with minimal brain dysfunction: Effects on attention span, visual-motor skills, and behavior. Curr Ther Res *16*:535-641, 1974.

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The delivery of this community based program is accomplished through interdisciplinary teams drawn from professional groups composed of psychologists, psychiatric social workers, community nurses, therapists, nurses and support staff.

As the Medical Director you will, within the guidelines established by the Branch and the Executive Director, have considerable freedom and influence with responsibility for effective and efficient clinical service. You will direct and control the admission, treatment and discharge procedures and have complete responsibility for assignments and evaluation of a medical staff of nine.

Applicants must have postgraduate psychiatric training and be eligible for full license in Saskatchewan. Graduates of approved medical schools in the United Kingdom, Ireland, South Africa, Australia, New Zealand, Canada and the United States may be eligible for license.

The appointment to this position will be on a contract basis.

BASIC SALARY: up to $61,068 per annum and commensurate with qualifications and experience

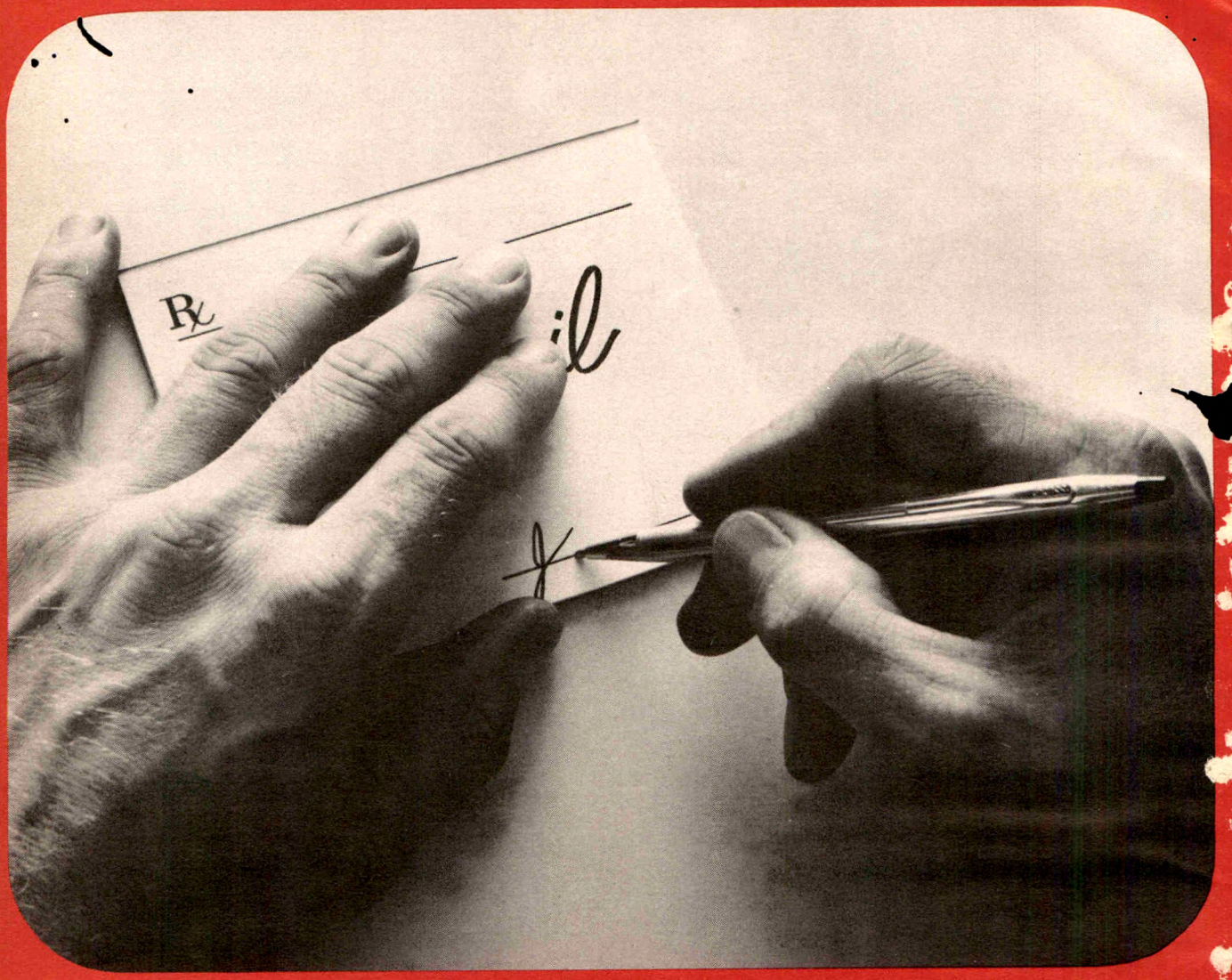
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Executive Director
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There is no substitute



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Contraindications: Known hypersensitivity. Should not be given concomitantly with a monoamine oxidase inhibitor since hyperpyretic crises, severe convulsions, and deaths have occurred. When used to replace a monoamine oxidase inhibitor, allow a minimum of 14 days to elapse before initiating therapy with amitriptyline HCl. Initiate dosage of amitriptyline HCl cautiously with gradual increase in dosage until optimum response is achieved. Not recommended during the acute recovery phase following myocardial infarction.

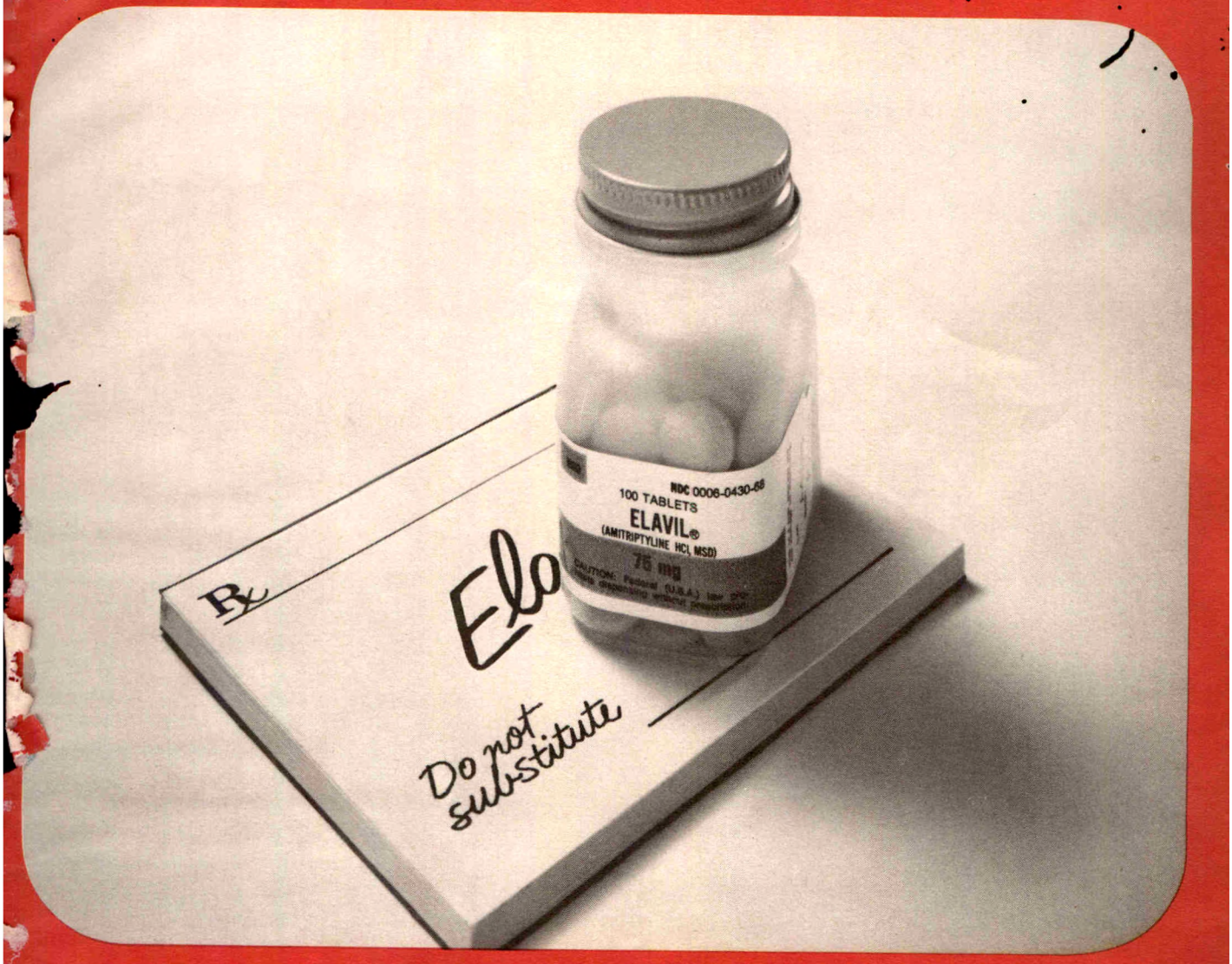
Warnings: May block the antihypertensive action of guanethidine or similarly acting compounds. Should be used with caution in patients with a history of seizures or a history of urinary retention, or with angle-closure glaucoma or increased intraocular pressure; in patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely; arrhythmias, sinus tachycardia and prolongation of the conduction time have been reported, particularly with high doses; myocardial infarction and stroke have been reported with drugs of this class. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. May impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle. In patients who use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage. Safe use during pregnancy and lactation has not been established; in pregnant patients, nursing mothers, or women who may become pregnant, weigh possible benefits against possible hazards to mother and child. Not recommended for patients under 12 years of age.

Precautions: Schizophrenic patients may develop increased symptoms of psychosis; patients with paranoid symptomatology may have an exaggeration of such symptoms; manic depressive patients may experience a shift to the manic phase. In these circumstances, the dose of amitriptyline HCl may be reduced or a major tranquilizer, such as perphenazine, may be administered concurrently.

When given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required; paralytic ileus may occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs. Use cautiously in patients receiving large doses of ethchlorvynol, since transient delirium has been reported on concurrent administration. May enhance the response to alcohol and the effects of barbiturates and other CNS depressants. The possibility of suicide in depressed patients remains until significant remission occurs. Potentially suicidal patients should not have access to large quantities of this drug. Prescriptions should be written for the smallest amount feasible. Concurrent electroshock therapy may increase the hazards associated with such therapy; such treatment should be limited to patients for whom it is essential. When possible, discontinue the drug several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported. Use with caution in patients with impaired liver function.

Adverse Reactions: *Note:* Included in this listing are a few adverse reactions not reported with this specific drug. However, pharmacological similarities among the tricyclic antidepressant drugs require that each reaction be considered when amitriptyline is administered. *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappro

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priate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, increased intraocular pressure, constipation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, leukopenia, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, black tongue, rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache, and malaise; these are not indicative of addiction.

Overdosage: Hospitalize as soon as possible all patients suspected of having taken an overdose. Treatment is symptomatic and supportive. In addition, the intravenous administration of 1 to 3 mg physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage should be repeated as required, particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine.

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For more detailed information, consult your MSD representative or see full prescribing information. Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa. 19486

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   *Other than a primary degree.

2. At least seven years satisfactory experience in the practice of the medical profession, including not less than five years satisfactory experience in psychiatry, of which not less than three years was in child psychiatry.

Applications should be addressed to:

The Reverend Brother Director,
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59 Orwell Road, Rathgar, Dublin 6, Ireland.
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Prescribing Information for

Lidone™ (molindone hydrochloride)

DESCRIPTION — LIDONE (molindone hydrochloride) is a dihydroindolone compound which is not structurally related to the phenothiazines, the butyrophenones or the thioxanthenes.

LIDONE is 3-ethyl-6, 7-dihydro-2-methyl-5-(morpholinomethyl) indol-4(5H)-one hydrochloride. It is a white crystalline powder, freely soluble in water and alcohol and has a molecular weight of 312.67.

ACTIONS — LIDONE (molindone hydrochloride) has a pharmacological profile in laboratory animals which predominantly resembles that of major tranquilizers causing reduction of spontaneous locomotion and aggressiveness, suppression of a conditional response and antagonism of the bizarre stereotyped behavior and hyperactivity induced by amphetamines. In addition, LIDONE antagonizes the depression caused by the tranquilizing agent tetrabenazine.

In human clinical studies tranquilization is achieved in the absence of muscle relaxing or incoordinating effects. Based on EEG studies, LIDONE exerts its effect on the ascending reticular activating system.

Human metabolic studies show LIDONE (molindone hydrochloride) to be rapidly absorbed and metabolized when given orally. Unmetabolized drug reached a peak blood level at 1.5 hours. Pharmacological effect from a single oral dose persists for 24 to 36 hours. There are 36 recognized metabolites with less than 2 to 3% unmetabolized LIDONE being excreted in urine and feces.

INDICATIONS — LIDONE (molindone hydrochloride) is indicated in the management of the manifestations of schizophrenia.

CONTRAINDICATIONS — LIDONE (molindone hydrochloride) is contraindicated in severe central nervous system depression (alcohol, barbiturates, narcotics, etc.) or comatose states, and in patients with known hypersensitivity to the drug.

WARNINGS

Usage in Pregnancy: Studies in pregnant patients have not been carried out. Reproduction studies have been performed in the following animals:

Pregnant Rats Oral Dose —
20 mg/kg/day — 10 days, no adverse effect
40 mg/kg/day — 10 days, no adverse effect

Pregnant Mice Oral Dose —
20 mg/kg/day — 10 days, slight increase resorptions
40 mg/kg/day — 10 days, slight increase resorptions

Pregnant Rabbits Oral Dose —
5 mg/kg/day — 12 days, no adverse effect
10 mg/kg/day — 12 days, no adverse effect
20 mg/kg/day — 12 days, no adverse effect

Animal reproductive studies have not demonstrated a teratogenic potential. The anticipated benefits must be weighed against the unknown risks to the fetus if used in pregnant patients.

Nursing Mothers: Data are not available on the content of LIDONE (molindone hydrochloride) in the milk of nursing mothers.

Usage in Children: Use of LIDONE (molindone hydrochloride) in children below the age of twelve years is not recommended because safe and effective conditions for its usage have not been established.

PRECAUTIONS — Some patients receiving LIDONE (molindone hydrochloride) may note drowsiness initially and they should be advised against activities requiring mental alertness until response to the drug has been established.

...ased activity has been noted in patients ...g LIDONE. Caution should be exercised ...creased activity may be harmful.

...E does not lower the seizure threshold in ...ntal animals to the degree noted with ...ating antipsychotic drugs. However, con... ...eizures have been reported in a few ...

... has an antiemetic effect in animals. A ...ffect may occur in humans and may ...igns of intestinal obstruction or brain ...

ADVERSE REACTIONS

CNS Effects — The most frequently occurring effect is initial drowsiness that generally subsides with continued usage of the drug or lowering of the dose.

Noted less frequently were depression, hyperactivity and euphoria.

Neurological Extrapyramidal Reactions — Extrapyramidal reactions noted below may occur in susceptible individuals and are usually reversible with appropriate management.

Akathisia — Motor restlessness may occur early.

Parkinson Syndrome — Akinesia, characterized by rigidity, immobility and reduction of voluntary movements and tremor, have been observed. Occurrence is less frequent than akathisia.

Dystonic Syndrome — Prolonged abnormal contractions of muscle groups occur infrequently. These symptoms may be managed by the addition of a synthetic antiparkinson agent (other than L-dopa), small doses of sedative drugs, and/or reduction in dosage.

Autonomic Nervous System — Occasionally blurring of vision, tachycardia, nausea, dry mouth and salivation have been reported. Urinary retention and constipation may occur particularly if anticholinergic drugs are used to treat extrapyramidal symptoms.

Hematological — There have been rare reports of leucopenia and leucocytosis. If such reactions occur, treatment with LIDONE may continue if clinical symptoms are absent. Alterations of blood glucose, liver function tests, B.U.N., and red blood cells have not been considered clinically significant.

Metabolic and Endocrine Effects — Alteration of thyroid function has not been significant. Amenorrhea has been reported infrequently. Resumption of menses in previously amenorrheic women has been reported. Initially heavy menses may occur. Lactation associated with LIDONE therapy has not been reported. Increase in libido has been noted in some patients. Impotence has not been reported. Although both weight gain and weight loss have been in the direction of normal or ideal weight, excessive weight gain has not occurred with LIDONE.

Cardiovascular — Rare, transient, non-specific T wave changes have been reported on E.K.G. Association with a clinical syndrome has not been established. Rarely has significant hypotension been reported.

Ophthalmological — Lens opacities and pigmentary retinopathy have not been reported where patients have received LIDONE (molindone hydrochloride). In some patients, phenothiazine induced lenticular opacities have resolved following discontinuation of the phenothiazine while continuing therapy with LIDONE.

Skin — Early, non-specific skin rash, probably of allergic origin, has occasionally been reported. Skin pigmentation has not been seen with LIDONE usage alone.

LIDONE (molindone hydrochloride) has certain pharmacological similarities to other antipsychotic agents. Because adverse reactions are often extensions of the pharmacological activity of a drug, all of the known pharmacological effects associated with other antipsychotic drugs should be kept in mind when LIDONE is used. Upon abrupt withdrawal after prolonged high dosage an abstinence syndrome has not been noted.

Tardive Dyskinesia — Although rarely reported with LIDONE (molindone hydrochloride) symptoms were reversible upon discontinuation of therapy.

Tardive dyskinesia associated with other agents has appeared in some patients on long-term therapy and has also appeared after drug therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). There may be involuntary movements of extremities.

There is no known effective treatment of tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear. Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked. It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time the syndrome may not develop.

DOSAGE AND ADMINISTRATION — Initial and maintenance doses of LIDONE (molindone hydrochloride) should be individualized.

Initial Dosage Schedule

The usual starting dosage is 50 to 75 mg/day.
- — Increase to 100 mg/day in three or four days
- — Based on severity of symptomatology, dosage may be titrated up or down depending on individual patient response.
- — An increase to 225 mg/day may be required in patients with severe symptomatology.
- — Some chronic, treatment-resistant patients may require up to 400 mg/day; (however, the long-term safety of 400 mg/day has not been established).

Elderly or debilitated patients should be started on lower dosage.

Maintenance Dosage Schedule

1. Mild — 5 mg to 15 mg three or four times a day
2. Moderate — 10 mg to 25 mg three or four times a day.
3. Severe — 225 mg/day may be required.

Dosage may be administered once a day.

DRUG INTERACTIONS — Potentiation of drugs administered concurrently with LIDONE (molindone hydrochloride) has not been reported. Additionally, animal studies have not shown increased toxicity when LIDONE is given concurrently with representative members of three classes of drugs (i.e., barbiturates, chloral hydrate and antiparkinson drugs).

MANAGEMENT OF OVERDOSAGE — Symptomatic, supportive therapy should be the rule.

Gastric lavage is indicated for the reduction of absorption of LIDONE (molindone hydrochloride) which is freely soluble in water.

Since the adsorption of LIDONE (molindone hydrochloride) by activated charcoal has not been determined, the use of this antidote must be considered of theoretical value.

Emesis in a comatose patient is contraindicated. Additionally, while the emetic effect of apomorphine is blocked by LIDONE in animals, this blocking effect has not been determined in humans.

A significant increase in the rate of removal of unmetabolized LIDONE from the body by forced diuresis, peritoneal or renal dialysis would not be expected. (Only 2% of a single ingested dose of LIDONE is excreted unmetabolized in the urine. However, poor response of the patient may justify use of these procedures.

While the use of laxatives or enemas might be based on general principles, the amount of unmetabolized LIDONE in feces is less than 1%. Extrapyramidal symptoms have responded to the use of diphenhydramine (Benadryl*) and the synthetic anticholinergic antiparkinson agents (i.e. Artane*, Cogentin*, Akineton*).

HOW SUPPLIED — LIDONE (molindone hydrochloride) capsules are supplied in bottles of 10 in the following dosage strengths and colors:

5 mg (**NDC** 0074-5542-13) blue and cream
10 mg (**NDC** 0074-5543-13) red and cream
25 mg (**NDC** 0074-5544-13) brown and cream

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